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SCIENTIFIC INVESTIGATIONS

Internalizing symptoms and sleep outcomes in urban children with and without asthma

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Study Objectives: This study examines associations between internalizing symptoms and sleep in a sample of urban children with and without asthma, whether asthma status moderates these associations, and whether associations differ by ethnic group.

Methods: Participants were Latino, African American (AA), and non-Latino white (NLW) urban 7- to 9-year-olds with (n = 259) and without (n = 122) persistent asthma. Teacher-reported internalizing symptoms (anxiety, depressive, and somatic) were assessed using the Behavioral Assessment System for Children-2. Sleep duration, variability in sleep duration, and sleep onset latency were assessed with actigraphy.

Results: Depressive symptoms were associated with variability in sleep duration and shorter sleep onset latency; somatic symptoms were associated with variability in sleep duration. In Latino children, depressive symptoms were associated with shorter sleep onset latency. In AA children, anxiety, depressive, and somatic symptoms were associated with variability in sleep duration; somatic symptoms were related to variability in sleep duration in NLW children. The association between internalizing symptoms and sleep outcomes did not differ by asthma status. However, asthma status was a significant moderator when examining these associations by ethnic group: among AA children, depressive symptoms were significantly related to variability in sleep duration only in children with asthma, whereas in NLW children, somatic symptoms were related to variability in sleep duration only in children without asthma.

Conclusions: Targeting specific internalizing symptoms and sleep outcomes may be beneficial in the development of interventions tailored for urban children with and without asthma from specific ethnic groups.

Keywords: asthma, internalizing symptoms, race/ethnicity, sleep, urban children

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BRIEF SUMMARY

Current Knowledge/Study Rationale: A number of studies have demonstrated that urban ethnic minority children with asthma are at increased risk for poor sleep outcomes; however, gaps in understanding what factors may contribute to these associations remain. To date, no studies have investigated how specific internalizing symptoms relevant for urban minority children with asthma, such as anxiety, depressive, and somatic symptoms, may be associated with sleep outcomes.

Study Impact: Study strengths include examination of specific internalizing symptoms and sleep outcomes, use of objective sleep measures (actigraphy), the urban and ethnic diversity of the study sample, and inclusion of children with and without asthma. Results can inform modifiable, co-occurring targets for intervention in need of attention in this high-risk group.

INTRODUCTION

Sleep during childhood is essential to the development of biological, physiologic, cognitive, and psychological processes.[1] Shorter sleep duration can place children at risk for deficits in cognitive skills, memory, and learning, which can affect academic performance,[2] and a variety of adverse mental health and physical outcomes including poor emotion regulation (eg, anxiety and depressive symptoms), externalizing problems (eg, disruptive behavior), increased reports of somatization, and increased risk for obesity.[3] Similarly, the amount of time children take to fall asleep may affect their amount of sleep[4] and may contribute to variability in sleep duration. Both sleep onset latency and changes in the amount of sleep a child obtains have been linked to increased behavioral and emotional problems in childhood.[5]

Sleep outcomes in urban minority children

Children living in urban, economically disadvantaged communities are particularly at risk for poor sleep outcomes due to exposure to urban stressors including lower socioeconomic status (SES),[6] family stress,[7] and poor neighborhood conditions (eg, community violence).[8] Environmental conditions more prevalent in low-SES, urban areas (eg, increased noise during the night, uncomfortable bed, and uncomfortable room temperatures) have been linked to inconsistent and later sleep onset times, wake times, and daytime sleepiness,[9] as well a shorter and more variable sleep duration.[10] Other stressors

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more likely in urban environments, such as witnessing violence, have also been associated with adolescent self-reported sleep problems such as difficulties falling asleep and daytime sleepiness.[8] Studies focusing on sleep in ethnic minority children have found that compared to non-Latino white (NLW) children, ethnic minority children experienced shorter sleep duration[11] and more daytime sleepiness.[10] Given that a higher proportion of ethnic minority children live in urban areas, urban minority children may be at heightened risk for poor sleep outcomes (eg, shorter sleep, more variable sleep, later sleep onset).[12]

Asthma and sleep outcomes in urban minority children

A growing body of evidence has shown that urban minority children with asthma have been found to be particularly vulnerable to poor sleep.[13,14] In the United States, asthma is disproportionally present in African American (AA) and Latino youth, with higher rates in Puerto Rican youth.[15] Children with persistent levels of asthma are more likely to experience nocturnal asthma symptoms,[13,16] which can disrupt their sleep.[17] When asthma is not well controlled, children are likely to experience poorer sleep efficiency,[18] which may lead to increased daytime sleepiness,[19] reduced sleep duration and increased sleep onset latency,[20] poorer school performance, and more frequent school absences.[14]

A number of factors can increase the likelihood of nocturnal asthma, which can make it difficult for urban minority children with asthma to obtain high-quality and a sufficient amount of sleep.[13] For example, they are at greater risk for exposure to environmental triggers,[21] stressful life events including neighborhood violence and school difficulties,[22] cultural stresses (eg, language barriers, acculturative stress),[23] and poor medication adherence.[24] Further, increased risk for nocturnal asthma in combination with stressors of urban poverty place urban minority children with asthma at greater risk for increased levels of psychological stress,[25] which may in turn negatively affect sleep outcomes (eg, sleep duration and sleep onset latency); however, associations between psychological symptoms and sleep outcomes need to be examined in further depth in this group.

Internalizing symptoms and asthma

Numerous studies have assessed the comorbidity of asthma and internalizing symptoms.[26] Increased anxiety,[27] panic symptoms,[28] and symptoms of social anxiety[29] have been documented in youth with asthma compared to those without asthma. Higher levels of depressive symptoms[30] and an increased prevalence of somatic disorders[31,32] have also been reported in children with asthma. Further, youth with asthma and low SES and/or minority group status have been shown to be more likely to experience increased symptoms of anxiety and depression compared to their counterparts without asthma.[25] In addition, higher rates of somatic symptoms have been reported in Latino children with asthma, compared to NLW children with asthma.[32]

Internalizing symptoms and sleep

Links between internalizing symptoms and negative sleep outcomes have been shown through the use of subjective (eg, self-report and teacher-report questionnaires and sleep diaries) and objective (eg, polysomnography and actigraphy) methods of assessing sleep.[33] Urban minority children with asthma may be at greater risk for poorer sleep given the high prevalence of internalizing symptoms. To date, no studies have investigated associations between specific internalizing symptoms relevant for urban minority children with asthma, such as anxiety, depressive, and somatic symptoms, and sleep outcomes. Results from such research can inform modifiable targets for intervention that may be co-occurring and in need of attention in this high-risk group.

The current study

The goal of this study was to examine the associations between internalizing symptoms (ie, anxiety, depressive, and somatic symptoms) and objectively measured sleep outcomes (ie, average sleep duration, variability in sleep duration, and average sleep onset latency) in a sample of Latino, AA, and NLW urban children with and without asthma. We also examined whether asthma status (having persistent asthma or not) moderated these associations. It was hypothesized that higher levels of anxiety, depressive, and somatic symptoms would each be associated with poorer sleep outcomes, and that these associations would be more robust in children with asthma. We then examined whether health status moderated these associations across ethnic groups in our sample. It was hypothesized that the associations between higher levels of anxiety, depressive, and somatic symptoms and negative sleep outcomes would be more robust in children with asthma versus healthy controls, and even more so in ethnic minority children with asthma. This study extends previous research and will help inform the development of tailored asthma and sleep treatments for urban youth.

METHODS

Participants

Data were collected as part of a larger study (NAPS, or Nocturnal Asthma and Performance in School)[10,14,18] assessing the co-occurrence of asthma, sleep quality, and academic functioning in urban children with and without persistent asthma across one academic year. Children and their caregivers were recruited from the four largest urban school districts located in a Northeastern United States city, from hospital-based outpatient pediatric clinics, and from a hospital-based asthma education program. Urban children (mean age = 8.3 years) with (n = 259) and without asthma (n = 122) were enrolled in the study.

All participants were between 7 and 9 years old, attended public school in one of the four targeted urban school districts, and their legal guardian self-identified as Latino (Dominican or Puerto Rican), AA, or NLW. Children without asthma did not have a chronic medical condition. At screening, children with asthma needed to have physician-diagnosed asthma or breathing problems in the previous 12 months as reported by caregiver report and a current prescription for an asthma controller medication, and/or one or more of the following in the past 4 weeks: recurrent daytime or nighttime symptoms, activity limitation, rescue medication use, or 2 or more oral steroid

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bursts during the prior 12 months based on caregiver report. Asthma diagnosis and severity were later evaluated by the study clinicians, and asthma medication use was confirmed. Exclusion criteria for both groups included moderate to severe cognitive impairment as indicated by school placement or use of stimulant medication for ADHD; another pulmonary or chronic health condition; or a diagnosed sleep disorder (eg, restless leg syndrome, chronic insomnia) that could potentially confound the larger study. Overall, 22% of families preferred to complete the protocol in Spanish.

Procedures

Data reported herein were collected during the fall/winter period of each study year (2010–2014). The initial research visit took place in families' homes and consisted of the informed consent/assent process with children and caregivers followed by completion of caregiver surveys concerning the family's demographic characteristics and their child's asthma status and medications. Participants with asthma then completed a second visit at an asthma and allergy clinic with a study clinician who confirmed asthma diagnosis and evaluated disease severity and medication usage. Families of children without asthma completed their second visit at the study research offices. After their second study visit, all children wore an Actiwatch (Philips Respironics, Philadelphia, Pennsylvania, United States) to assess sleep duration and sleep onset. Sleep outcomes were assessed during one 4-week home monitoring period in the fall/winter. Halfway through the visit, research staff met with families to administer sleep questionnaires and to download Actiwatch data. Study assessments were translated into Spanish using standard procedures.[34] Study instruments were presented in English or Spanish by fully bilingual research staff and according to each participant's preference. Approval from the appropriate institutional review board was obtained.

Measures

Demographic information

Caregivers provided demographic information (**Table 1**). To determine whether a family fell at or below the poverty level, the family's self-reported income was divided by the United States federal per capita poverty threshold value based on family size during the year of their study participation.[35] Neighborhood risk was determined using census block membership based on home address. A participant was assigned a score based on the following risk factors: family income, caregiver education, caregiver unemployment, racial/ethnic minority status, non-English speaking, vacant housing, small housing units, and poverty status. Risk scores ranged from 0 to 8 and were based on the number of risk characteristics

Table 1—Sample characteristics of children with and without asthma.

Characteristics	Sample (n = 381)	Asthma (n = 259)	Healthy Control (n = 122)	Group Differences	Effect Size
Mean child age, years	8.31	8.30	8.32	$F_{1,379} = .05$	$\eta_p^2 < .01$
Male, %	53.5	53.3	54.1	$\chi^2 = .02$	$\varphi = .01$
Caregiver race/ethnicity, %				$\chi^2 = 11.68$**	$\varphi = .18$
Latino	47.0	52.1[a]	36.1[b]		
Black	33.1	32.0[a]	35.2[a]		
Non-Latino white	19.9	15.8[a]	28.7[b]		
Weight status, %					
Healthy weight	–	54.0	–		
Overweight/obese	–	46.0	–		
Asthma poorly controlled, %	–	40.0	–		
Use of daily asthma controller, %	–	74.5	–		
At/below poverty threshold, %	67.0	70.0	60.0	$\chi^2 = 3.01$	$\varphi = .09$
Mean neighborhood risk index	4.93	5.01	4.77	$F_{1,379} = 1.01$	$\eta_p^2 = .01$
Mean no. of people in household	4.63	4.62	4.65	$F_{1,379} = 0.02$	$\eta_p^2 < .01$
Mean internalizing symptoms					
Anxiety	47.16	47.54	46.34	$F_{1,379} = 1.67$	$\eta_p^2 < .01$
Depression	48.05	48.23	47.66	$F_{1,379} = 0.52$	$\eta_p^2 < .01$
Somatization	53.53	54.77[a]	50.92[b]	$F_{1,379} = 9.76$**	$\eta_p^2 = .03$
Sleep outcomes, mean in minutes					
Sleep duration	556.66	556.20	557.65	$F_{1,379} = 0.16$	$\eta_p^2 < .01$
Variability in sleep duration	60.08	60.23	59.76	$F_{1,379} = 0.04$	$\eta_p^2 < .01$
Sleep onset latency	17.99	18.41	17.11	$F_{1,379} = 2.15$	$\eta_p^2 = .01$

Means/prevalence scores that have no superscript in common are significantly different from each other. **$P < .01$. η_p^2 = partial eta squared, φ = Cramer phi.

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identified, with higher scores indicating more risk. This index has been associated with asthma outcomes (eg, asthma management and morbidity) in urban children.[36,37] To confirm a diagnosis of asthma and assess for severity of asthma, each asthma participant underwent a physical examination, allergy skin testing, and pulmonary function testing. A study clinician used National Heart, Lung, and Blood Institute Expert Panel Report-3 guidelines to confirm asthma severity level (mild, moderate, or severe).[38] Caregivers reported on current use of asthma controller medication. We assessed asthma control using the Asthma Control Test.[39] The scores can range from 5 to 25 with scores at or below 19 indicating poorly controlled asthma.[40] Sleep-disordered breathing was assessed using the sleep-disordered breathing subscale in the Pediatric Sleep Questionnaire, with scores greater than .33 suggesting high sleep-disordered breathing risk.[41]

Sleep outcomes

Sleep duration and sleep onset latency were assessed using an Actiwatch 2. Children were instructed to wear the Actiwatch on their nondominant wrist at all times except when bathing or swimming. Caregivers provided additional information including their child's morning wake times and evening bedtimes, illnesses other than asthma, and times when the Actiwatch was not worn.[42] Actiware-Sleep version 2.53 software (Philips Respironics) estimated 1-minute epochs as either periods of sleep and wakefulness using activity levels produced in the surrounding 2-minute interval and Actiwatch event markers set by participants at "lights-off" and "lights-on." Analysis of each sleep episode was based on standard scoring rules.[42] Actigraphy data from adherent families were collected across week and weekend nights and were included if at least 5 days[42] of data were available. We based our decision on the range of 5 to 7 nights of data proposed by Acebo and colleagues[42] and we elected 5 nights because we expected adherence in our study to be lower given our sample's increased exposure to urban stressors. Episodes were excluded when (1) the concurrent diary report was not available, (2) the Actiwatch was off for all/part of the sleep period, (3) diary indicated illness other than asthma that could have affected sleep on a given night, or (4) when all or part of the sleep period included external motion (eg, sleeping in a car).[37] Participants had, on average, 17.98 scorable nights (standard deviation = 7.69; range = 2–40). Two sleep outcome variables were calculated based on mean values across the monitoring period including sleep duration, defined as the average total time between evening sleep onset and morning waking and sleep onset latency, defined as the average time between going to bed and actually falling asleep. The third sleep outcome, variability in sleep duration, is the standard deviation of sleep duration across the monitoring period and represents the variability in total time between evening sleep onset and morning waking.[43]

Behavioral Assessment System for Children – 2, Teacher Rating Scales – Child

The Behavioral Assessment System for Children – Teacher Rating Scales form is a teacher-report measure used to assess behavioral and emotional functioning in children and adolescents.[44] The full measure comprises composite scales, each consisting of small subdomains of functioning. Three distinct subscales were used to assess symptoms of anxiety, depression, and somatization. Teachers were asked to rate the frequency of certain symptoms and/or behaviors on a four-point Likert scale (never, sometimes, often, and almost always). Items assessing anxiety symptoms involved reporting on a child's frequency of worries including worries about things that cannot be changed, schoolwork, making mistakes, and negative evaluation from peers. The depression symptoms subscale items assess emotion regulation, mood, self-worth, and loneliness. Somatization items assess somatic experiences including general health complaints, stomach problems, headaches, visits to the school nurse, and pain. Cronbach alphas for each scale indicate similar reliability scores across general and clinical samples, and range from .78 to .94.[44]

Data screening

Data screening was conducted to examine normality of distributions, potential outliers, and missing data. First, we examined patterns of missing data using the Little missing completely at random (MCAR) test,[45] which indicated that data were MCAR (P = .239). Thus, we imputed missing data using Expectation Maximization using the Missing Values Analysis package in SPSS. Most of the variables of interest were positively skewed with the exception of average sleep duration. Winsorization and Mahalanobis Distance procedures intended to normalize the data and adjust outliers were used.[46] Maximum likelihood estimation method was used as this type of estimation method provides unbiased and efficient estimates of the variance and covariance parameters in both normally distributed and skewed data.[47] For all regression models, only significant findings were reported. Analyses were conducted using SPSS version 23.0 (IBM Corp, Armonk, New York, United States). Chi-square effect sizes (ES) are expressed as Cramer phi (φ), which is similar to a point-biserial correlation with .1 considered a small effect, .3 a medium effect, and .5 a large effect.[48] Effect sizes for analyses of variance are expressed as partial eta-squared (η_p^2) with .01 considered a small effect, .06 considered a medium effect, and .14 a large effect.[48] Effect sizes for multiple regression analyses are expressed as Cohen f^2 with .02 considered a small effect, .15 a medium effect, and .35 a large effect.[48]

Data analysis plan

Associations among demographic variables, anxiety, depression, and somatization symptoms, and sleep outcomes (ie, average sleep duration, variability in sleep duration, and average sleep onset latency) were examined via Pearson correlations when both variables were continuous, or analyses of variance when examining continuous variables across discrete groups. Preliminary analyses were used to identify demographic variables related both to each component of internalizing symptoms and sleep outcome variable, so that they would be accounted for in subsequent analyses.

Following preliminary analyses, we examined the association between each internalizing symptom variable (the independent variable) and each sleep outcome variable (the dependent variable) in separate linear regression analyses using generalized linear models (**Figure 1**). Models were then stratified by ethnic group. The next set of analyses assessed the extent to which health

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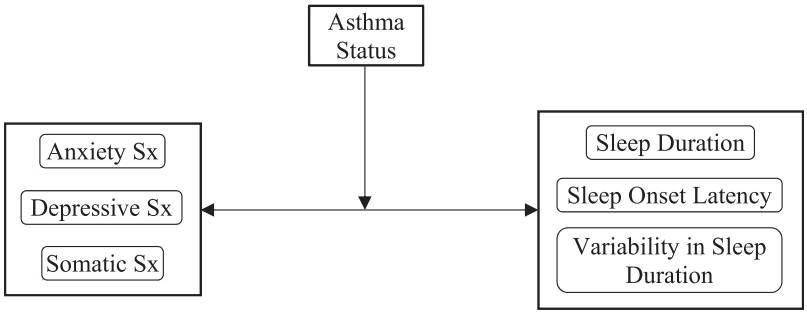
Figure 1—Conceptual model of analyses.



This figure represents the main effects between each internalizing symptoms (Sx) variable (the predictor variable) and each sleep outcome variable (the dependent variable), and the moderating effect of asthma status on each of these pathways via separate linear regression analyses using generalized linear models. Models were run using the entire sample and then stratified by ethnic group (ie, Latino, African American, and non-Latino white). Significant main effects and moderation effects by the entire sample and by ethnic group are outlined in Table 3.

status moderated the relations between anxiety, depressive, or somatic symptoms and sleep outcomes (Figure 1). Covariates were entered in the first step when indicated. Post hoc probing was conducted to clarify interaction terms that were significant or represented statistical trends. All analyses previously described were conducted first across the entire sample, and then stratified by ethnic group. It is important to note that analyses were designed based on a priori hypotheses about potential predictors and outcomes. An alpha level of 0.05 was used for all statistical tests. Only significant findings were reported.

RESULTS

Preliminary analyses

Table 1 describes the characteristics of the total sample (n = 381; 53.5% male; mean age = 8.3 years), children with persistent asthma (n = 259), and healthy control patients (n = 122). Forty-seven percent identified as Latino (n = 179), 33% as AA (n = 126), and 20% as NLW (n = 76). Most of the patients (67%; n = 239) fell at or below the poverty threshold. By ethnic group, 80.7% of Latino children, 64.7% of AA children, and 35.3% of NLW children fell at or below the poverty threshold. Five percent of children had anxiety symptoms in the clinically significant range, whereas 2.6% fell in the at-risk range. Similarly, 5% of children had depressive symptoms in the clinically significant range, whereas 2.4% fell in the at-risk range. For somatization symptoms, 13.7% fell in the clinically significant range, whereas 9.2% fell in the at-risk range. Regarding asthma, 40% (n = 89) of the sample of children with asthma was classified as having poorly controlled asthma (based on the Asthma Control Test). Finally, among children with asthma, 74.5% of caregivers (n = 173) reported their children were taking an asthma controller medication.

Age was negatively related to average sleep duration ($r = -.29$, $P < .001$) and somatic symptoms ($r = -.12$, $P < .05$). Neighborhood risk was negatively associated with average sleep duration ($r = -.15$, $P < .01$) and positively associated with variability in sleep duration ($r = .12$, $P < .05$). There were differences by sex in somatic symptoms, with females reporting higher levels of somatic symptoms (mean for females = 55.42; $F_{1,379} = 9.34$, $P = .002$; small ES, $\eta_p^2 = .02$). Children at or below the poverty level reported shorter average sleep duration ($F_{1,356} = 8.60$, $P = .004$; small ES, $\eta_p^2 = .02$), higher variability in sleep duration ($F_{1,356} = 8.91$, $P = .003$; small ES, $\eta_p^2 = .02$), and slightly lower levels of anxiety symptoms ($F_{1,356} = 4.42$, $P = .036$; small ES, $\eta_p^2 = .01$). Children with asthma experienced higher levels of somatic symptoms ($F_{1,379} = 9.76$, $P = .002$; small ES, $\eta_p^2 = .03$). Given our plan involved examining main effect associations and moderation analyses by ethnic group, we did not include ethnicity as a covariate; however, we note ethnic differences in sample characteristics in Table 2. The number of scored nights a participant had completed over the monitoring period was not significantly associated with any of the sleep outcomes in this study. In addition, sleep-disordered breathing was not associated with any of the sleep outcomes. Demographic characteristics significantly correlated with both the predictor and outcome variables were included as covariates in subsequent analyses.

Table 2 outlines differences in internalizing symptoms and sleep outcomes in children with and without asthma by ethnic group. Examination of differences by health status and ethnic group revealed NLW children with asthma had longer average sleep duration than Latino and AA children with asthma ($F_{2,256} = 12.31$, $P < .001$; medium ES, $\eta_p^2 = .09$), and AA children with asthma experienced more variability in their sleep duration than NLW children with asthma ($F_{2,256} = 3.54$, $P = .030$; small ES, $\eta_p^2 = .03$). Among children without asthma, AA children experienced longer average sleep duration ($F_{2,119} = 3.52$, $P = .033$; medium ES, $\eta_p^2 = .06$) and higher levels of depressive symptoms than NLW children ($F_{2,119} = 4.63$, $P = .012$; medium ES, $\eta_p^2 = .07$).

Associations between internalizing symptoms and sleep outcomes

Separate regressions were performed using generalized linear models to examine the associations between anxiety, depressive, and somatic symptoms and each sleep outcome including average sleep duration, variability in sleep duration, and average sleep onset latency in the entire sample of children (Table 3).

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Table 2—Ethnic group differences in sample characteristics, internalizing symptoms, and sleep outcomes in children with and without asthma.

Children With Asthma	Latino (n = 135)	AA (n = 83)	NLW (n = 41)	Group Differences	Effect Size
Sample characteristics					
Asthma poorly controlled, %	32.7[a]	50.6[b]	35.1[a,b]	χ^2 = 6.51*	φ = .17
Use of daily asthma controller, %	72.6	80.5	70.3	χ^2 = 2.03	φ = .09
At/below poverty threshold, %	82.6[a]	62.5[b]	39.5[c]	χ^2 = 28.71***	φ = .34
Mean neighborhood risk index	5.50	5.20[a]	3.02[b]	$F_{2,256}$ = 26.36***	η_p^2 = .17
Mean no. of people in household	4.64	4.63	4.41	$F_{2,256}$ = .291	η_p^2 < .01
Mean internalizing symptoms					
Anxiety	47.16	46.73	50.47	$F_{2,256}$ = 2.58	η_p^2 = .02
Depression	47.77	49.19	47.79	$F_{2,256}$ = 1.00	η_p^2 = .01
Somatization	53.85	55.71	55.87	$F_{2,256}$ = 0.77	η_p^2 = .01
Sleep outcomes, mean in minutes					
Sleep duration	548.96[a]	557.88[a]	576.61[b]	$F_{2,256}$ = 12.31**	η_p^2 = .09
Variability in sleep duration	60.08[a,b]	63.63[a]	53.81[b]	$F_{2,256}$ = 3.54*	η_p^2 = .03
Sleep onset latency	17.36[a]	18.64[a,b]	21.35[b]	$F_{2,256}$ = 3.64*	η_p^2 = .03
Children Without Asthma	**Latino (n = 44)**	**AA (n = 43)**	**NLW (n = 35)**	**Group Differences**	**Effect Size**
Sample characteristics					
At/below poverty threshold, %	74.4[a]	69.2[a]	30.0[b]	χ^2 = 16.00***	φ = .39
Mean neighborhood risk index	5.31[a]	5.49[a]	3.20[b]	$F_{2,119}$ = 12.01***	η_p^2 = .17
Mean no. of people in household	4.64	4.65	4.53	$F_{2,119}$ = 0.10	η_p^2 < 01
Mean internalizing symptoms					
Anxiety	46.32	47.04	45.50	$F_{2,119}$ = 0.47	η_p^2 = .01
Depression	46.75[a,b]	49.96[a]	45.99[b]	$F_{2,119}$ = 4.63*	η_p^2 = .07
Somatization	51.22	51.70	49.58	$F_{2,119}$ = 0.70	η_p^2 = .01
Sleep outcomes, mean in minutes					
Sleep duration	554.92[a,b]	550.77[a]	569.54[b]	$F_{2,119}$ = 3.52*	η_p^2 = .06
Variability in sleep duration	62.36	60.91	55.06	$F_{2,119}$ = 1.07	η_p^2 = .02
Sleep onset latency	17.02	17.61	16.62	$F_{2,119}$ = 0.19	η_p^2 < .01

Means/prevalence scores that have no superscript in common are significantly different from each other. *P < .05, **P < .01, ***P < .001. AA = African American, NLW = non-Latino white, η_p^2 = partial eta squared, φ = Cramer phi.

Demographic variables related to both the internalizing symptoms indicator and sleep outcome variable in each analysis were adjusted for when indicated in initial analyses. Additional analyses adjusting for age, sex, and neighborhood risk independent of whether they were associated with both predictor and outcome variables were completed and compared to initial analyses.

Higher levels of depressive symptoms were associated with more variability in sleep duration (B = .39, SE = .15, P = .007; very small ES, f^2 = .004) and shorter average sleep onset latency (B = −.17, SE = .06, P = .003; very small ES, f^2 = .005). Additionally, higher levels of somatic symptoms were associated with more variability in sleep duration (B = .30, SE = .09, P = .001; very small ES, f^2 = .007). After adjusting for age, sex, and neighborhood risk, all significant findings remained in the entire sample.

All analyses were then stratified across different ethnic minority groups (ie, Latino, AA, and NLW; **Table 3**). The most robust results were found among AA children in which higher levels of anxiety symptoms (B = .47, SE = .20, P = .017, very small ES, f^2 = .01), depressive symptoms (B = .60, SE = .22, P = .006; small ES, f^2 = .015), and somatic symptoms (B = .38, SE = .15, P = .010; very small ES, f^2 = .013) were associated with more variability in sleep duration. Among Latino children, higher levels of depressive symptoms were associated with shorter average sleep onset latency (B = −.29, SE = .09, P = .001; small ES, f^2 = .015). Among NLW children, higher levels of somatic symptoms were related to more variability in sleep duration (B = .48, SE = .19, P = .014; small ES, f^2 = .021). After adjusting for age, sex, and neighborhood risk across all analyses, significant findings remained expect in two cases: (1) A main effect between somatic symptoms and sleep onset latency was no longer found for Latino children, and (2) no significant main effects were found in NLW children.

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Table 3—Significant associations between internalizing symptoms and sleep outcomes stratified by ethnic group.

Whole Sample	**B**	**SE**	**95% CI**	**Effect Size (f^2)**
Outcome: variability in sleep duration				
Model 1				
Asthma (0 = no asthma)	−0.25	2.25	−4.65 to 4.15	
Depression symptoms	0.39**	0.15	0.11 to 0.68	.004
Model 1				
Asthma (0 = no asthma)	0.68	2.26	−3.76 to 5.11	
Somatic symptoms	0.30**	0.09	0.12 to 0.48	.007
Outcome: sleep onset latency				
Model 1				
Asthma (0 = no asthma)	−1.39	0.87	−3.10 to 0.32	
Depression symptoms	−0.17**	0.06	−0.28 to −0.06	.005
Latino	**B**	**SE**	**95% CI**	**Effect Size (f^2)**
Outcome: sleep onset latency				
Model 1				
Asthma (0 = no asthma)	−0.64	1.34	−3.26 to 1.99	
Depression symptoms	−0.29**	0.09	−0.46 to −0.12	.015
African American	**B**	**SE**	**95% CI**	**Effect Size (f^2)**
Outcome: variability in sleep duration				
Model 1				
Asthma (0 = no asthma)	−2.87	3.55	−9.81 to 4.08	
Anxiety symptoms	0.47*	0.20	0.08 to 0.86	.011
Model 1				
Asthma (0 = no asthma)	−3.18	3.52	−10.09 to 3.72	
Depression symptoms	0.60**	0.22	0.18 to 1.02	.015
Model 2				
Asthma (0 = no asthma)	−3.01	3.47	−9.81 to 3.79	
Depression symptoms	0.07	0.34	−0.59 to 0.74	
Depression × asthma	0.87*	0.43	0.02 to 1.72	.008
Model 1				
Asthma (0 = no asthma)	−1.21	3.58	−8.23 to 5.81	
Somatic symptoms	0.38*	0.15	0.09 to 0.66	.013
Non-Latino White	**B**	**SE**	**95% CI**	**Effect Size (f^2)**
Outcome: variability in sleep duration				
Model 1				
Asthma (0 = no asthma)	4.24	4.38	−4.34 to 12.82	
Somatic symptoms	0.48*	0.19	0.10 to 0.86	.021
Model 2				
Asthma (0 = no asthma)	6.64	4.42	−2.03 to 15.31	
Somatic symptoms	1.33	0.46	0.42 to 2.24	
Somatic × asthma	−1.02*	0.51	−2.02 to −0.03	.013

*P < .05. **P < .01. B = unstandardized beta, CI = confidence interval, f^2 = Cohen f-squared, model 1 = main effect, model 2 = moderation effect, SE = standard error.

Moderation effects of health status on association between internalizing symptoms and sleep outcomes

Analyses were conducted to test the moderating role of asthma status (ie, patients with asthma versus healthy control patients) in the association between specific internalizing symptoms and sleep outcomes (Table 3). In the entire sample, health status did not moderate these associations. Analyses by ethnic group revealed that asthma status moderated the relationship between levels of depressive symptoms and variability in sleep duration (B = .87, SE = .43, P = .046; very small ES, f^2 = .008) such that

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higher levels of depressive symptoms were associated with more variability in sleep duration only in AA children with asthma (B = .94, SE = .28, P = .000; small ES, f^2 = .022). In NLW children, health status moderated the association between levels of somatic symptoms and variability in sleep duration (B = −1.02, SE = .51, P = .043; very small ES, f^2 = .013), in which higher levels of somatic symptoms were associated with more variability in sleep duration only in NLW children without asthma (B = 1.33, SE = .46, P = .010; small ES, f^2 = .028).

Similarly, after adjusting for age, sex, and neighborhood risk across all analyses, significant findings remained expect in two cases: (1) although the main effect was no longer significant for Latino children, a significant interaction by asthma status was found in which higher levels of somatic symptoms were associated with shorter average sleep onset latency only for Latino children with asthma (B = −.44, SE = .17, P = .009; very small ES, f^2 = .010), and (2) no significant interaction effects were found in NLW children. Age, sex, and neighborhood risk were only associated with some of the predictor variables and sleep outcome variables across analyses. The inclusion of these variables as covariates when they were not associated with the sleep outcomes may be removing meaningful variance from the predictor variables; thus, results from these additional analyses should be interpreted with caution.

DISCUSSION

A number of studies have demonstrated that urban minority children with asthma are at increased risk for poorer sleep outcomes[16,17]; however, factors that may contribute to these associations need to be identified in research with this population. Our findings begin to address this gap by examining whether internalizing symptoms more prevalent in urban children with asthma, such as anxiety, depressive, and somatic symptoms, may be related to specific sleep outcomes (ie, average sleep duration, variability in sleep duration, and average sleep onset latency) in a sample of Latino, AA, and NLW urban children with and without asthma. Results indicated that urban minority children who are at greater risk for experiencing internalizing symptoms may be particularly vulnerable to poor sleep hygiene and related sleep outcomes. Furthermore, asthma status may moderate the associations between certain internalizing symptoms and sleep outcomes in urban children from specific ethnic backgrounds.

We first examined the associations between internalizing symptoms (ie, anxiety, depressive, and somatic symptoms) and objectively measured sleep outcomes (ie, average sleep duration, variability in sleep duration, and average sleep onset latency) in our entire sample. Results indicated children with higher levels of depressive and somatic symptoms tended to have more inconsistent sleep duration across monitored nights. Consistent with previous research, urban risk factors including neighborhood risk and poverty level were associated with more variability in sleep duration,[10] which may be related to inconsistent or poor sleep hygiene behaviors (eg, inconsistent bedtimes and waketimes). In addition to urban risk factors, internalizing symptoms such as depressive and somatic symptoms may also challenge implementing healthy sleep behaviors consistently (eg, a consistent bedtime routine and consistent sleep schedule) and should be further explored in the context of specific sleep hygiene behaviors.

Results examining associations between specific internalizing symptoms and sleep outcomes across ethnic groups indicated that AA children experiencing higher levels of anxiety, depressive, and somatic symptoms were more vulnerable to inconsistencies in their sleep duration. Effect sizes ranged from very small to small; however, even small differences in variability in sleep may be clinically important and warrant further examination. Based on characteristics of our sample, Latino and AA children had significantly more variation in sleep duration than NLW children. Perhaps AA children were more vulnerable to inconsistent sleep across nights as this group was also noted to have high levels of poverty and neighborhood risk. It may be that other environmental, cultural, and health factors make it more difficult for urban minority children to implement consistent sleep hygiene behaviors resulting in more variation in sleep duration across nights. Furthermore, internalizing symptoms in this context may further hinder the implementation of healthy sleep routines.

Non-Latino white children with higher levels of somatic symptoms also experienced more variability in their sleep duration. Although we expected urban minority children to have higher levels of somatic symptoms, these levels did not differ across ethnic groups, which may partly explain similar associations between somatic symptoms and variability in sleep across the different ethnic groups in our sample. Similar to the entire sample, Latino children with higher levels of depressive symptoms experienced shorter sleep onset latency. The direction of this association was unexpected. Perhaps depressive symptoms in the context of children's increased exposure to urban stressors may be expressed as fatigue in urban children, which may then be associated with decreased arousal at nighttime and shorter sleep onset.[8,49] Additional research examining objective measurements of sleep in diverse populations is needed to gain further insight into how depressive symptoms relate to sleep onset latency, specifically in urban children.

We also examined whether having persistent asthma or not contributed to the association between internalizing symptoms and sleep outcomes in our sample. Health status did not appear to moderate these associations in the entire sample of children. However, analyses by ethnic group did reveal an effect of health status on associations between certain internalizing symptoms and sleep outcomes. Specifically, in AA children, the association between higher levels of depressive symptoms and more variability in sleep duration was significant only in children with asthma. In our sample, AA children with asthma had a higher proportion of children with poorly controlled asthma. Differences in sample characteristics such as asthma control, along with high levels of poverty, neighborhood risk, and variability in sleep duration, between AA children with asthma and other ethnic background groups in our sample may have contributed to our findings. For example, AA children experiencing poor asthma control in the context of urban stressors may have experienced more difficulty implementing

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consistent sleep hygiene practices, thus strengthening the association between their internalizing symptoms and poor sleep outcomes.

In NLW children, health status moderated the association between levels of somatic symptoms and variability in sleep duration in an unexpected way. Specifically, the association between higher levels of somatic symptoms and more variability in sleep duration was only significant for children without asthma compared to those with asthma. Consistent with previous literature, children with asthma in our sample across all ethnic groups reported higher somatization symptoms.[31] However, NLW children without asthma reported more variation in sleep duration than NLW children with asthma (**Table 2**). The relationship between higher levels of somatic symptoms and variability in sleep duration in NLW children without asthma may have been stronger given that this group reported more variability in sleep duration compared to NLW children with asthma. Given the small sample size of this subgroup, replication of these findings is needed to better understand this relationship.

Contrary to our hypotheses, higher levels of anxiety symptoms were associated with more variability in sleep duration for AA children only and did not seem to contribute to children's other sleep outcomes. The lack of significant main effect and moderation findings in most analyses conducted with anxiety symptoms as the predictor variable may partially be explained by the limited variance in anxiety symptoms across children with and without asthma and different ethnic backgrounds in our sample. Lack of differences in anxiety symptoms in children with versus without asthma may be due to the younger age range of our sample, as studies using similar aged samples (mean age = 8.4 years) have found combined internalizing symptoms and not anxiety symptoms alone to be elevated in children with asthma.[50] It also should be noted that there are inconsistencies in how anxiety symptoms and sleep outcomes are operationally defined and assessed in prior pediatric research. For example, a recent meta-analysis documented multiple studies using objective reports of sleep outcomes (eg, actigraphy) that failed to reproduce previously documented associations between anxiety and self-reports of sleep outcomes in children.[51] As proposed by McMakin and Alfano,[51] methodology that considers the multidimensional nature of anxiety and sleep (ie, self-reported and objective) in the context of longitudinal studies is needed to further clarify these inconsistencies, particularly in urban minority children.

Several limitations of this study warrant attention. Though we recognize the risks of analyzing multiple comparisons, we erred on the side of detecting findings that might be clinically significant and not statistically significant. We also collected sleep outcome data over multiple nights via actigraphy and average sleep outcome variables were used, thus limiting the ability to test longitudinal causal effects. In addition, a single informant (ie, teacher report) assessment of children's internalizing symptoms was used that does not capture children's expression of symptoms across different settings throughout the day. Future research should assess sleep outcomes year-round versus only the fall/winter due to the seasonal variation in asthma symptoms and potential impact on sleep outcomes. Although we included children with varying weight status given the high comorbidity between asthma and obesity, we recognize that weight status may influence sleep outcomes, and this is an important topic to explore in future research. The larger study included only 7- to 9-year-old children as this is a critical time when children begin to develop independent asthma self-management behaviors; therefore, current results may not generalize to children in other age groups. Although this study included a narrow age range (7–9), we did not assess pubertal development of participants. Early pubertal development may have affected daytime sleepiness and shorter sleep duration particularly in children from specific ethnic groups[52]; this should be considered in future research with this population. Finally, we assessed medication use on any type of controller at baseline; however, we did not assess whether children were prescribed oral corticosteroids specifically during the study. This is a limitation as oral corticosteroids can be linked to insomnia and shorter sleep duration.[53]

Longitudinal studies using larger samples of youth with wider age ranges and multiple informants are needed to help identify sensitive developmental periods when aspects of mental health may be more influential to children's sleep. Additional examination of biopsychosocial factors and potential pathways through which internalizing symptoms and sleep outcomes are related will also help clarify previous findings. Furthermore, future studies should assess more culturally relevant forms of anxiety (eg, acculturative stress), urban stressors (eg, urban poverty), stressful life events, and specific factors related to asthma adjustment and control, as these factors may be more relevant to internalizing symptoms and sleep in urban minority children with and without asthma.[14,37] Additional sleep indicators, such as average wake times, may be important to consider in future research as wake times may affect circadian preference, sleep duration, variability in sleep duration, and sleep onset latency. Future studies may also benefit from exploring how weekend catch-up sleep may influence sleep outcomes in urban samples.

Internalizing symptoms may have unique effects on sleep outcomes in urban children with and without asthma. The current findings can help inform the development of tailored asthma and sleep interventions in urban children who are at risk for poor asthma control as each internalizing symptom group and sleep outcome represent potential treatment targets (eg, do specific types of internalizing symptoms affect how children fall asleep vs. maintain sleep, etc.). Sleep interventions tailored for urban minority children with and without asthma may want to specifically focus on behaviors related to consistent bed and wake times as these behaviors will promote more consistent and adequate sleep duration across each night. Health care providers serving urban pediatric populations should routinely ask about anxiety, depressive, and somatic symptoms as these symptoms may contribute to children's sleep health and may affect the implementation of consistent sleep hygiene behaviors. It is important that providers also acknowledge urban stressors such as poverty and neighborhood risk when addressing internalizing symptoms and making behavioral sleep recommendations in this context.

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ABBREVIATIONS

AA, African American
ES, effect size
MCAR, missing completely at random
NAPS, Nocturnal Asthma and Performance in School
NLW, non-Latino white
SES, socioeconomic status

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