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Children's Sleep and Cognitive Functioning: Race and Socioeconomic Status as Moderators of Effects

Joseph A. Buckhalt and Mona El-Sheikh
Auburn University

Peggy Keller
Notre Dame University

Race and socioeconomic status (SES) moderated the link between children's sleep and cognitive functioning. One hundred and sixty-six 8- to 9-year-old African and European American children varying in SES participated. Sleep measures were actigraphy, sleep diaries, and self-report; cognitive measures were from the Woodcock–Johnson III and reaction time tasks. Children had similar performance when sleep was more optimal, but after controlling for SES, African American children had lower performance with sleep disruptions. Children from lower and higher SES had similar performance with better sleep quality and less variability in sleep schedules, but when sleep was more disrupted, higher SES children had better performance. Examination of environmental variables associated with race and SES that may underlie these effects may lead to directions for interventions to improve cognitive performance.

Sleep deprivation in American children is becoming a matter of broad national concern. A nationwide poll conducted by the National Sleep Foundation (NSF, 2004) indicated that many children are getting insufficient amounts of sleep, and that the quality of their sleep is too often compromised. Estimates of the numbers of children presenting with sleep problems vary depending on definitional criteria and type of measure, but a range of estimates have been reported, from around 20% (Mindell, Owens, & Carskadon, 1999) to as high as 37% (Owens, Spirito, McGuinn, & Nobile, 2000), and 41% (Archbold, Pituch, Panahi, & Chervin, 2002). While the functions of sleep are still largely unknown, contemporary research is confirming the common belief that developing brains need to spend considerable portions of each day in sleep, and an adequate amount of good-quality sleep is essential for optimal child functioning (Dahl, 1996). Building upon research with adults and with clinical samples of children, pediatric sleep researchers have begun to explore the relations between sleep and cognitive functioning in typically developing healthy children (e.g., Sadeh, Gruber, & Raviv, 2002). This study extends that literature by examining relations between multiple facets of sleep in children, including schedule, quantity, and quality, in relation to several important dimensions of cognitive functioning, as well as by assessing the role of child race and socioeconomic status (SES) in moderating the connection between sleep and cognitive functioning.

In adults, sleep impairment has been linked to a wide variety of cognitive measures in both experimental and correlational studies with clinical and nonclinical samples. A stringent meta-analysis by Pilcher and Huffcut (1996) indicated that with sleep deprivation, cognitive processing in adults is affected significantly, with sleep-deprived subjects performing at levels often 2 standard deviations (*SD*s) lower than controls. Most of the research linking sleep with cognition in childhood has been carried out with children who have a diagnosed sleep disorder. For example, children with sleep-disordered breathing show low performance on a number of cognitive measures, including attention, memory, and academic achievement (Blunden, Lushington, Lorenzen, Martin, & Kennedy, 2005; Gozal, 1998; Kaemingk et al., 2003; also see Ebert & Drake, 2004 for a review). Much sleep research has also been carried out with clinical samples of children for whom difficulties with learning and attention are the primary referral problem. The sleep of children with attention deficit hyperactivity disorder (ADHD) has been studied for over 20 years. Reports of sleep

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Correspondence concerning this article should be addressed to Joseph A. Buckhalt, Department of Counselor Education, Counseling Psychology & School Psychology, 2084 Haley Center, Auburn University, Auburn, AL 36849. Electronic mail may be sent to buckhja@auburn.edu.

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disturbance based on subjective reports are quite common (e.g., Chervin, Dillon, Basseti, Ganoczy, & Pituch, 1997; Cohen-Zion & Ancoli-Israel, 2004), but evidence based on objective measures of polysomnography and actigraphy is less conclusive (e.g., Gruber, Sadeh, & Raviv, 2000; Gruber & Sadeh, 2004).

While the mechanisms by which cognitive processing is compromised by sleep loss have only recently begun to be clarified, basic research in sleep is converging on the conclusion that good quality and sufficient quantity of sleep are necessary for memory consolidation, and that disruptions in sleep such as those caused by poor flow of air through breathing passages may be responsible for dysfunctions in neurotransmitter activity required for cognitive processing during waking hours (Hobson & Pace-Schott, 2002; Pace-Schott & Hobson, 2002; Siegel, 2001; Stickgold, 2005). But sleep deprivation research has been limited somewhat by lack of consilience between sleep and cognitive researchers, and ontogenetic sleep research has often lacked a coherent guiding theory of cognitive development and little consensus in construct definition and measurement of cognition.

Horne (1988, 1993) first suggested that of all brain regions, functioning of the prefrontal cortex (PFC) may be most affected by sleep loss, and demonstrated that performance on tasks thought to depend on the PFC, such as novel language and creativity tests, show significant decrements with sleep loss. Drawing from the work of Horne and others, Dahl (1996) described a developmental model for the development of sleep and arousal regulation in which PFC functioning has a central role. In Dahl's model, sleep loss is thought to affect those tasks that require smooth executive functioning, efficient working memory, and the synchronization of attention and arousal in performing goal-directed tasks, particularly when the goals are abstract or complex.

Most sleep deprivation experiments comparable to those that have been carried out with adults are not ethically appropriate for children, but a few studies have experimentally restricted sleep and measured subsequent cognitive performance. Initial studies restricted or extended sleep for one night and showed that restricted sleep was associated with poorer performance for some, but not all, expected tasks (Fallone, Acebo, Arnedt, Seifer, & Carskadon, 2001; Randazzo, Muelbach, Schweitzer, & Walsh, 1998). Two subsequent studies have involved experimental manipulation of sleep over longer periods. Sadeh, Gruber, and Raviv (2003) had children restrict or extend their sleep for 1 hr over three nights and measured cognitive functioning with the Neurobehavioral Evaluation System. Children who extended their sleep had improved post intervention performance on two of nine measures derived from six tasks: digit memory forward (but not backward) and reaction time (RT) on the Continuous Performance Test. Additionally, for simple RT, the performance of the sleep-restricted group declined, while that of the sleep-extended group remained stable. Fallone, Acebo, Seifer, and Carskadon (2005) used a within-subjects design with 1 week of baseline, 1 week of sleep restriction, and 1 week of sleep optimization to show that children whose sleep was restricted were rated by teachers to have more academic problems compared with baseline and optimized conditions, and more attention problems compared with the baseline condition.

In addition to these experimental studies, a few correlational studies using objective assessments of sleep have also been reported. Children's sleep quantity and quality measured with actigraphy has been related to visual and auditory working memory (Steenari et al., 2003) and to executive control (Sadeh et al., 2002). In the latter study, it was concluded that more complex tasks, which involve executive control, and also tasks that require some inhibition of response, were affected by fragmented sleep, while simpler tasks of motor speed, memory, and RT were not affected. And in a study of matched groups from the general population, O'Brien et al. (2004) found that children meeting polysomnography criteria for sleep-disordered breathing had lower scores for General Conceptual Ability (*g*) and Nonverbal Ability on the Differential Ability Scales.

Findings of impairment for more complex tasks (O'Brien et al., 2004; Sadeh et al., 2002) versus simpler tasks (Sadeh et al., 2003) warrant further exploration of the types of tasks affected by sleep differences in children. Efforts to test Dahl's (1996) model are complicated to some degree by the variety of cognitive measures used to measure executive functioning and working memory, and better construct specificity and measurement are needed. Few studies have used test batteries that are widely used in schools and clinical practice and standardized on large, representative samples of children. In addition, more research is needed to determine the exact sleep parameters (e.g., schedule, quantity, or quality) that are most closely related to cognitive performance.

Racial and SES Differences in Sleep

Only recently have sleep researchers begun to report significant differences in sleep parameters for African Americans (AAs) in comparison with Euro-

pean Americans (EAs). Durrence and Lichstein (2006) report that half of the literature on sleep of AAs has appeared in the last 5 years. They reviewed 30 studies, all with adult subjects, and derived some general conclusions: (1) AAs have rates of sleep-disordered breathing twice that of EAs (Ancoli-Israel et al., 1995; Redline et al., 1997); (2) AAs take longer to fall sleep and nap longer than EAs (Lichstein, Durrence, Riedel, Taylor, & Bush, 2004); and (3) AAs have fundamental differences in sleep architecture, particularly in depth of sleep and rapid eye movement (REM) sleep even when confounding factors are controlled (Profant, Ancoli-Israel, & Dimsdale, 2002). Fewer studies exist with AA children, but they indicate that the rates of asthma and sleep-disordered breathing are greater for AAs than EAs (Redline et al., 1999; Rosen et al., 2003; Stepanski, Zayyad, Nigro, Lopata, & Basner, 1999). In a large ($N = 1{,}043$) longitudinal study of a community sample from 2 to 8 years old, in which sleep was examined through parental report, AA children (26% of sample) were found to have shorter average nighttime sleep durations, more napping during the day, and more weekend ''oversleep,'' a differential between school- and weekend-night sleep commonly considered indirect evidence of sleep deprivation (Crosby, LeBourgeois, & Harsh, 2005). Chervin et al. (2003) have previously proffered the hypothesis that sleep-disordered breathing may explain a substantial part of the academic achievement deficits of AA children. But their study, which used parent-reported sleep problems and teachers' ratings of school performance along with group-administered tests of reading and mathematics, showed that SES, and not race, was a significant explanatory factor.

SES has been a frequently studied variable in health research, and while a number of factors have been proposed as mechanisms in the link between poor health and low SES, the exact mechanisms are still poorly understood (Adler et al., 1994; Williams, Yu, Jackson, & Anderson, 1997). Few studies have addressed how differences in sleep quality or duration may help determine, or reflect, health disparities based on SES, but some evidence suggests that sleep quality may mediate the effect of SES on mental and physical health (Moore, Adler, Williams, & Jackson, 2002). Poorer sleep in low-SES individuals may be related to a variety of factors, including work schedules, overcrowded households, chronic stressors associated with scarcity of resources, diet and alcohol consumption, and even poorer temperature control in the sleep environment (Williams, 1999). While cognitive and achievement differences in children by SES have long been documented, the role of sleep in the link between SES and those outcomes is largely unknown. Bates, Viken, Alexander, Meyers, and Stockton (2002) showed that for a sample of low income predominantly EA preschoolers, higher variability in sleep schedule was related to poorer school adjustment, but cognitive performance was not investigated.

Although children's race and SES have been examined in relation to sleep, to our knowledge, no study has examined these factors as moderators of the sleep-cognitive functioning link. Support for moderation effects would not speak to issues of causality, but rather would explicate for whom and under which conditions the relations between sleep and cognitive outcomes are evident. Examination of moderators rests on the assumption that although sleep disruptions may be predictive of child cognitive problems, the impact of sleep problems on children may not be uniform and may differ by race or SES. The health disparity view (e.g., Carter-Pokras & Baquet, 2002) is that specific populations, including ethnic and racial minorities as well as those of lower SES, may have an increased burden of adverse conditions (Healthy People 2010, 2006; National Institutes of Health, 2006).

Our hypothesis is that effects of disruptions in a primary domain of biological regulation, namely sleep, may have a differential impact on individuals based on their race or SES. This hypothesis rests on the assumption that AA and lower SES children are likely to be exposed to more stressors in their environment than their EA and higher SES counterparts, and that additional stressors (e.g., poor sleep in this study) may be related to lower levels of cognitive functioning. Through our examination of these moderation effects, we test the novel proposition that sleep differences may be related to the long reported cognitive differences between AAs and EAs, and lower and higher SES children. In an attempt to unconfound the frequently reported association between race and SES, we recruited AA and EA children across a wide SES range.

To strengthen construct measurement for sleep, we examined multiple facets of sleep including objective assessments of sleep schedule, quantity, and quality. We also examined subjective reports of sleepiness and sleep problems. For measurement of cognitive functioning, we chose the Woodcock–Johnson III Tests of Cognitive Ability (WJ III; Woodcock, McGrew, & Mather, 2001), which has a complex hierarchical factor structure based on the Cattell–Horn–Carroll theory of intelligence (Carroll, 1993), and uses a large number of subtests to derive multiple factors, including general intellectual ability (*g*),

executive functioning, and working memory. We also selected RT tasks capable of distinguishing fine gradations of performing simple tasks requiring extended vigilance and attention. The inclusion of both RT tasks and several WJ III tests was intended to facilitate exploration of sleep relations with a range of tasks varying in the type and degree of cognitive demand. We expected that less sleep and poorer quality sleep would be associated with complex, as opposed to simple cognitive tasks, and that those tasks engaging executive functioning and working memory would be most related.

Method

Sample Recruitment

Children were recruited from the third grade of a public school system in the Southeastern U.S. Schools provided names and home telephone numbers of parents of students, and we contacted parents by telephone. During the call, we explained the study briefly and discussed a few exclusion criteria. Exclusion criteria included chronic or acute physical illness (including asthma), ADHD, learning disabilities, mental retardation, and a history of diagnosed sleep problems. Owing to parameters related to another set of research questions unrelated to this paper, a further exclusion criterion was that children had to be living with two parents who were either married or cohabiting for at least the past 2 years. Biological mothers of all children participated. Of those families who met our inclusion criteria, 66% participated, 28% declined to participate, and 6% indicated that they were too busy and asked to be called at a later date. We were unable to collect any information about families who declined to participate, so the effects of self-selection are unknown. Mothers provided informed consent for their own and their child's participation, and children provided informed assent. Participating families received monetary compensation for their time and effort.

Participants

Seventy-four boys and 92 girls with a mean age of 8.72 years ($SD = 0.29$; range 7 years 2 months to 11 years) participated. Mothers completed the Puberty Development Scale (PDS) (1 = *prepubertal*, 2 = *early pubertal*, 3 = *midpubertal*, 4 = *late pubertal*, 5 = *postpubertal*; Petersen, Crockett, Richards, & Boxer, 1988) during their visit to the lab. The mean scores were 1.44 ($SD = 0.34$; range = 1.0–2.4) for girls and 1.24 ($SD = 0.23$; range 1.0–2.2) for boys. Children were mostly prepubertal, with 94% of the children in the prepubertal stage and 6% in the early pubertal stage.

SES of the sample was diverse (raw score mean = 35.92; $SD = 9.56$; Hollingshead, 1975) with the following percentages of families in each of the five SES levels: 27% in either level 1 or 2 (e.g., unskilled and semiskilled workers), 41% in level 3 (e.g., skilled workers), and 32% in level 4 or 5 (e.g., minor professional or professional).

Racial identity of the child was identified by the parent according to 2000 U.S. census categories. All parents selected a sole category of ''White'' or ''Black or AA'' with the following result: 69% EA; 31% AA. The sample constitution is very similar to the racial and SES composition of the school district from which it was drawn, which includes 75% White, 22% Black or AA, <1% Native American or Alaska Native, <1% Asian, and 2% with ethnic identification of Hispanic or Latino. We oversampled to include a wide range of SES from both AA and EA families; yet race and SES were still somewhat related (rs = .17 for SES raw score and .24 for SES level, ps < .05). Note that we chose in this paper to use the term race rather than ethnicity. Much attention has been given to the proper use and measurement of race and ethnicity in scientific research (e.g., Entwisle & Astone, 1994) and we elected to use race.

Procedure

An actigraph was delivered to the child's home, and parents were instructed to attach it to the child's non-dominant wrist at bedtime for seven consecutive nights. Parents kept a diary of their child's daily bedtime and wake time, and were called once each day to obtain this information. Typically, on the day following the last day of actigraphy, children came to our university laboratory for one visit where they were administered the WJ III, the RT tasks, and the sleep questionnaire via interview. To reduce potential confounds, children's sleep was examined during the regular academic year, with the exclusion of school holidays.

The children were medication-free during the week of actigraphy, with the exceptions that nine subjects took some allergy medicine (these nights were excluded from actigraphy analyses).

Whenever possible, visits to the lab were scheduled in the mid to late afternoon during weekdays. Roughly 90% of the visits were during weekdays and 10% were conducted on Saturdays.

Measures

School Sleep Habits Survey (SHS). Children completed the SHS (Wolfson & Carskadon, 1998), which

has previously been used with adolescents as young as 13 (Acebo & Carskadon, 2002), and has been demonstrated to have good reliability and validity (Carskadon, Seifer, & Acebo, 1991; Wolfson et al., 2003) for adolescents as well as for children as young as six (El-Sheikh & Buckhalt, 2005). Only the Sleep/Wake Problems Scale (10 items) and the Sleepiness Scale (9 items) were used for this analysis. On the Sleep/Wake Problems Scale, children endorsed the frequency of several sleep and wake problems including oversleeping, falling asleep in classes and various events, and staying up late at night. Each item was rated for frequency from *never* (1) to *everyday/night* (5), with a range of possible total scores from 10 to 50. Coefficient α for the Sleep/Wake Problems Scale was .67. The nine sleepiness items were scored on a 4-point scale in relation to the child's statements of struggling to stay awake or falling asleep during nine daily activities (e.g., attending a performance, watching TV, in class, traveling, etc.); an additional item regarding feeling sleepy while driving a car was not administered to children given their young age. Scores could range between 9 and 36. Coefficient α for the Sleepiness Scale was .70.

Actigraphy. Activity between bedtime and wake time was monitored, and daily sleep logs were completed by the parents to cross-validate sleep start and end times. The actigraph was an Octagonal Basic Motionlogger (Ambulatory Monitoring Inc., Ardsley, NY), a small and lightweight (45 g) sleep device the size of a wristwatch. Motion during sleep was continuously monitored by the actigraph in 1-min epochs using zero crossing mode. Actigraphic raw data were downloaded and transformed into the pertinent sleep variables via the Octagonal Motionlogger Interface with ACTme software and the analysis software package (ActionW2, 2002). These sleep/wake measures have been demonstrated to have validity in calculations of sleep parameters, including those for children, when compared with polysomnography (Sadeh, Sharkey, & Carskadon, 1994; Sadeh, Acebo, Seifer, Aytur, & Carskadon, 1995). Procedures for setting times for sleep onset followed a lab protocol developed at the E. P. Bradley Hospital Sleep Laboratory at Brown University (Acebo & Carskadon, 2001). Movement at a threshold level for a period of time based on the Sadeh scoring algorithm is scored as ''awake'' or ''asleep'' (Sadeh et al., 1994, 1995). Very good reliability and validity for actigraphic measures have been reported (Acebo et al., 1999; Sadeh et al., 2002).

Sleep schedule measures were (a) sleep onset time—the first minute of three consecutive minutes of sleep as indicated by the Sadeh algorithm; (b) sleep end or wake time—the last minute of five consecutive minutes scored as sleep before a large activity period consistent with reported waking time. In addition, variability in either (c) sleep onset or (d) wake time was examined through the mean centered coefficient of variation statistic (Snedecor & Cochran, 1967). Sleep quantity was indicated by (e) sleep duration—sometimes referred to as sleep period—reflecting time from sleep onset time to sleep end. Measures of sleep quality were: (f) sleep efficiency (% of motionless sleep); and (g) sleep activity index—% of epochs with activity. Although we examined the relations between race, SES, and all of the derived actigraphy variables listed here, the moderation analyses focused on sleep variables more consistently tied to child functioning in the literature (duration, efficiency, activity, and variability in sleep onset or wake time).

Actigraphy variables were based on seven nights of averaged data, with some exceptions noted later. Night-to-night stability of these variables, was examined through intraclass correlations. α statistics were as follows: sleep onset time = .83; wake time = .76; sleep duration = .59; sleep efficiency = .87; and sleep activity = .94. Consistent with Acebo et al.'s (1999) suggestion of aiming for a stability level of .70, all but one of these variables exceeded this level.

Seventy-four percent of the children had actigraphy data for the whole week. However, because of either actigraph malfunction, forgetting to wear the device, wearing the device in a nonsuitable environment (e.g., car), or use of allergy medicine, 24% had data for fewer than seven nights ($M = 5.33$ days; $SD = 0.97$); 2% of children ($n = 4$) had no actigraphy data. This proportion of valid actigraphy data for children is considered very good (Acebo et al., 1999).

Cognitive assessment—WJ III. Children were administered individually six tests of the WJ III. The WJ III is a well-normed, highly reliable, and valid comprehensive system for measuring general intellectual ability *(g)* and specific cognitive abilities in nine factorial dimensions of intelligence. For this study, six tests were chosen: verbal comprehension (VC); concept formation (CF); visual matching (VM); numbers reversed (NR); auditory working memory (AWM); and decision speed (DS). VC includes four subtests: picture vocabulary, synonyms, antonyms, and verbal analogies, and is considered a good measure of crystallized intelligence. CF is a novel controlled-learning task that requires categorical reasoning based on principles of inductive logic. It is a test of fluid reasoning and also measures an aspect of executive functioning: flexibility in shifting mental

set. VM is a test of perceptual processing speed. NR is a test of short-term memory span and also engages working memory, as the numbers must be held in working memory while performing a mental operation (reversing the sequence). AWM also measures short-term auditory memory and working memory. In addition, divided attention is required. A series of numbers mixed with words is presented, and one must recall the series reporting the words in correct order first, then the numbers. DS measures visual processing speed by requiring that two conceptually similar items are selected from multiple trials presenting rows of alternatives.

The particular six tests were chosen to derive scores that reflected general intellectual ability, fluid and crystallized intellectual ability, simple processing speed, working memory, and executive functioning. Five cluster scores composed of combinations of tests, based on principal components factor analyses (Woodcock et al., 2001), were derived. For increased validity and reduction of the number of variables, analyses focused on the following cluster scores and not on constituent test scores: (1) Brief intellectual ability, based on three tests (VC; CF; and VM) with a median reliability of .95 across all ages in the standardization, and correlated .91 for children ages 6–8 years with the general intellectual ability index that is based on 10 tests (Woodcock et al., 2001); (2) processing speed (DS; VM); (3) working memory (NR; AWM); (4) cognitive efficiency (VM; NR; Cognitive efficiency is described as automatic information processing capacity related to executive and automatic processing); and (5) verbal ability, a cluster score based on the four separate verbal subtests of VC. Scaled scores with $M = 100$; $SD = 15$ were derived from age-based norms.

RT tasks. These tasks measure speed and variability of response speed across multiple trials and were computer-administered with Psych/Lab (Abrams, 2004) Donders program that obtains RTs under several different conditions. The child was seated before a computer and instructed to focus on a "+" in the screen's center. The right index was placed on the "/" key, and the left index finger was placed on the "Z" key. In the simple reaction time condition (Donders Type A), an "X" appears intermittently in a box to the right of the "+" and the child was instructed to press the "/" key as quickly as possible. If the response was too slow, incorrect, or if no key was pressed when one was required, a short tone is presented. In the second condition, choice reaction time 1 (Donders Type C), a box is present to the left and to the right of the "+" and an "X" may appear in either box. The instruction is to press the "/" key if the "X" appears in the right box. The task is more difficult due to the necessity of monitoring the left box and having to inhibit a response with the right finger, and is thought to engage executive functioning. In the third condition, choice reaction time 2 (Donders Type B), a box is present to the left and to the right of the "+" and an "X" may appear in either box. The instruction is to press the "/" key with the right finger if the "X" appears in the right box or press the "Z" key with the left finger if the "X" appears in the left box. Conditions were presented in a fixed order with the following parameters: 10 practice trials/condition; 20 test trials/condition; a 500 ms intertrial interval; 100 ms minimum allowed RT; and 1,500 ms maximum allowed RT. When responses were faster or slower than the criteria, the trial was repeated. Based on all test trials within each condition, averaged means and standard deviations were derived for analyses. Examination of children's responses during the three conditions (types A, B, and C) indicated that either the means (rs = .66–.76, ps < .001), or *SD*s (rs = .37–.42, ps < .001) were significantly correlated. Thus, to reduce the number of analyses, one averaged mean and one *SD* were derived and used in all subsequent analyses.

Results

Preliminary Analyses

We examined the role of the child's age, gender, and puberty status in relation to the sleep and cognitive functioning variables. Older age was associated with increased variability in sleep onset ($r = -.26$, $p < .001$) and wake time ($r = -.16$, $p < .05$). Older age was also related to better cognitive performance on all WJ III tests (rs = .16–.21, ps < .05) and possibly RT ($r = -.15$, $p = .06$) and RT variability (*SD*; $r = -.16$, $p = .05$). Puberty status was not related to any of the sleep or cognitive functioning measures. In relation to gender-related effects, t tests indicated that, in comparison with boys, girls had lower levels of sleep activity, $t(159) = 3.81$, $p < .001$, better sleep efficiency, $t(159) = -2.39$, $p < .01$, and longer sleep duration, $t(159) = 3.38$, $p < .001$. Means and *SDs* for girls' and boys' sleep variables, respectively, were 41.35 (12.63) and 48.91 (12.29) for sleep activity, 88.14 (7.47) and 85.16 (8.24) for sleep efficiency, and 8 hr 41 min (34 min) and 8 hr 22 min (38 min) for sleep duration. Furthermore, girls ($M = 110.04$; $SD = 13.75$) had higher processing speed scores than boys ($M = 105.06$; $SD = 14.41$), $t(164) = -2.27$, $p < .05$. Finally, boys ($M = 439.41$ ms; $SD = 60.43$) had faster RTs than girls ($M = 486.39$;

$SD = 80.55$), $t(145) = -3.96$, $p < .001$. Because of these significant relations, age and gender were statistically controlled in the primary analyses.

Associations Among Variables

Table 1 presents correlations among the primary study variables. Only modest associations between sleep quality (activity and efficiency) and either sleep quantity (duration) or variability in sleep schedule variables were evident. Similarly, few relations between actigraphy-based sleep and subjective measures of sleep were detected. Correlations among the measures of cognitive performance are also presented in Table 1. The subscales of the WJ III were moderately to highly related. Furthermore, all of the WJ III scales were significantly correlated with a faster RT and lower levels of variability on the Donders task, indicative of a better performance.

Sleep and Cognitive Functioning

Correlations between the sleep and cognitive variables are presented in Table 1. Processing speed was associated with sleep quality and duration in the expected directions. Sleepiness was correlated with lower levels of cognitive performance on several WJ III scales. Similarly, higher levels of sleep/wake problems were associated with worse cognitive performance on several WJ III scales as well as with RT and variability.

Comparisons Between EA and AA Children Effects on Sleep and Cognitive Functioning Variables

Because race and SES were associated, albeit at a low level, analyses of race-related effects controlled for SES. This allows for the examination of race related associated with sleep independent of the constraints or allowances that may be afforded by economic considerations (e.g., cosleeping because of family preferences vs. the number of bedrooms). Table 2 presents the results of multivariate analysis of covariances (MANCOVAs) comparing the mean values of children's sleep schedule, quantity, and quality as well as cognitive functioning across AA and EA children, with SES as a covariate. In addition to the primary sleep variables examined in this study, Table 2 presents data on additional sleep schedule variables for a better delineation of sleep variables that varied between AA and EA children. The significant multivariate F indicated differences between AA and EA children's objective sleep measures. Specifically, in comparison with AA children, EA children experienced more sleep activity, longer sleep duration, later wake time during the

Table 1
Correlations Among Measures of Sleep and Cognitive Performance

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Sleep activity—actigraphy													
2. Sleep efficiency—actigraphy	−.74***												
3. Sleep duration—actigraphy	−.14†	.18*											
4. Variability in sleep onset—actigraphy	.16*	−.14†	−.18*										
5. Variability in wake time—actigraphy	.04	−.01	−.03	.34***									
6. Sleepiness—child report	−.01	−.03	−.09	−.01	.04								
7. Sleep/wake problems—child report	.05	−.06	−.08	.05	−.03	.46***							
8. Intellectual ability—WJ III	−.12	.06	.11	−.13	−.01	−.16*	−.13						
9. Verbal ability—WJ III	.03	.03	.13	−.21**	−.05	−.22**	−.22**	.73***					
10. Processing speed—WJ III	−.31***	.18*	.14†	−.01	.03	−.07	−.03	.66***	.24**				
11. Working memory—WJ III	−.03	.03	−.04	−.02	−.09	−.22**	−.16*	.57***	.47***	.37***			
12. Cognitive efficiency—WJ III	−.19*	.09	−.01	−.00	−.01	−.17*	−.14†	.71***	.40***	.69***	.81***		
13. Donders reaction time (mean)	−.06	.03	.11	−.04	−.01	−.02	.14†	−.31***	−.16*	−.31***	−.27**	−.33***	
14. Donders reaction time (*SD*)	.04	−.03	.03	.12	.03	.04	.16*	−.33***	−.21**	−.26**	−.28**	−.33***	.72***

Note. WJ III = Woodcock–Johnson III.
†$p < .08$, *$p < .05$, **$p < .01$, ***$p < .001$.

Table 2
African and European American Children's Sleep and Cognitive Performance

	African American		European American		
	M	*SD*	*M*	*SD*	*F*
Actigraphy-based sleep measures					4.19(10, 124)***
Sleep activity	40.48	12.43	46.44	12.26	8.14(1, 133)**
Sleep efficiency	87.34	7.93	86.82	7.03	0.19(1, 133)
Sleep duration (hrs:min)	8:19	35 min	8:41	35 min	9.43(1, 133)**
Sleep onset time, weekdays (p.m.)	9:27	49 min	9:13	42 min	1.60(1, 133)
Wake time, weekdays (a.m.)	5:33	37 min	5:50	35 min	7.27(1, 133)**
Sleep onset time, weekends (p.m.)	10:05	70 min	10:06	61 min	0.05(1, 133)
Wake time, weekends (a.m.)	6:55	64 min	6:53	51 min	0.01(1, 133)
Variability in sleep onset	0.036	0.031	0.027	0.015	4.67(1, 133)*
Variability in wake time	0.078	0.063	0.068	0.068	0.62(1, 133)
Subjective reports of sleep					5.89(2, 146)**
Sleepiness	15.28	4.60	13.50	4.47	4.26(1, 147)*
Sleep/wake problems	19.11	5.70	15.97	4.21	11.27(1, 147)***
Woodcock–Johnson III					6.48(5, 142)***
Intellectual ability	98.43	11.83	105.05	11.10	6.31(1, 146)*
Verbal ability	95.17	12.82	106.25	9.48	28.64(1, 146)***
Processing speed	107.72	12.31	108.31	14.79	0.26(1, 146)
Working memory	105.67	14.25	110.79	15.84	1.40(1, 146)
Cognitive efficiency	103.07	13.99	106.44	14.08	0.47(1, 146)
Donders reaction time task					0.40(2, 131)
Mean	467.58	69.36	463.37	78.03	0.01(1, 132)
SD	144.32	33.67	137.49	38.22	0.33(1, 132)

Note. *SD* = standard deviation; SES = socioeconomic status. SES is included as a covariate.
*$p < .05$, **$p < .01$, ***$p < .001$.

week, and less variability in sleep onset time. EA children's sleep duration was about 23 min longer than that of AAs, and on weekdays, AA children woke up around 17 min earlier than EAs. Furthermore, the multivariate *F* for subjective reports of sleep was significant and indicated that EA children reported less sleepiness and fewer sleep/wake problems in comparison with AAs.

Table 2 also presents the results of the MANCOVA comparing mean levels of cognitive performance across AA and EA children, controlling for SES. The multivariate *F* indicated that race was a significant predictor of children's cognitive performance. Follow-up ANOVAs indicated that AA children had lower scores on the WJ III intellectual ability and verbal ability scales. It is important to note that AAs and EAs did not significantly differ in relation to either the age or the gender composition of the subsamples. However, in comparison with EAs ($M = 1.29$; $SD = 0.27$), AAs ($M = 1.49$; $SD = 0.38$) were more biologically mature as indexed by the puberty scale, $t(158) = 3.55$, $p < .001$. AA girls ($M = 1.60$; $SD = 0.39$) were more biologically mature than EA girls ($M = 1.37$; $SD = 0.29$), $t(87) = 3.02$, $p < .005$. For AA girls, 78% had scores <2 (prepubertal); 22% had scores between 2 and 2.4 (early pubertal). Of EA girls, 95% had scores <2 and 5% had scores of 2. Similarly, AA boys ($M = 1.33$; $SD = 0.31$) were more biologically mature than EA boys ($M = 1.21$; $SD = 0.19$), $t(69) = 1.86$, $p = .06$. For AA boys, 95% had scores <2; 5% between 2 and 2.2. All EA boys had scores <2. Even though the majority of children were prepubertal, puberty status was controlled in primary analyses.

Sleep and Cognitive Functioning: SES-Related effects

Correlations between SES and cognitive performance indicated that SES was significantly associated with all scales on the WJ III (rs = .22–.29, all ps $< .01$), with higher SES related to better performance. Furthermore, SES was associated with fewer sleep/wake problems ($r = -.17$, $p < .05$). These significant relations between SES and either sleep or cognitive functioning were still evident when race was controlled.

Moderators of Relations Between Sleep Duration and Cognitive Performance

Hierarchical multiple regressions were run to determine whether race and SES interacted with

sleep in predicting cognitive performance (Aiken & West, 1991). In the first step of each regression, to conduct stringent examinations of relations between sleep and cognitive functioning, we controlled for children's age (Sadeh, Raviv, & Gruber, 2000), puberty status (Carskadon, Vieira, & Acebo, 1993), and gender (Sadeh et al., 2000), and entered the two potential moderators (race, SES); for parsimony, and given the focus of this study, we present betas associated with the potential moderators, but not for the other control variables in each regression analysis. In the second step, a sleep measure was added. Finally, the two-way interactions between the sleep measure and the three moderators were entered in the third step. In each equation, all predictors and moderators were centered. Significant interactions were graphed by computing predicted values of cognitive performance at high (+1 *SD*) and low (−1 *SD*) values for the moderator and sleep measure. Slopes in the graphs were examined to determine whether they were significantly different from zero. For succinctness, only representative graphs are included. It should be noted that, a significant *interaction term* indicates that the two plotted lines in any of the graphs are significantly different from each other. On the other hand, tests of slope significance merely indicate whether each slope is significantly different from zero.

Relations between sleep duration and cognitive performance are presented in Table 3. Race interacted with sleep duration in predicting intellectual ability (graph not shown) and processing speed (Figure 1a). Thus, the association between sleep duration and these cognitive functioning domains was different between AA and EA children. The pattern of moderation effects was very similar for intellectual ability and processing speed, and illustrated that the slopes representing the association between sleep duration and either intellectual ability or processing speed were significantly different from zero for AA, but not EA children, and only for AAs was sleep duration related to higher intellectual ability or to processing speed (Figure 1a). The reader is reminded that the slopes do not need to be significantly different from zero to establish that the two slopes differ from one another; the latter is determined by a significant interaction term in the regression equation.

SES also interacted with sleep duration in predicting children's intellectual ability and processing speed (see Table 3). As shown in Figure 1b, longer sleep duration was related to greater intellectual ability for children of higher, but not lower, SES. Similarly, longer sleep duration was related to greater processing speed for children of higher, but not lower SES (graph not shown). Whereas children of lower and higher SES were very similar in their intellectual ability and processing speed when they had shorter sleep duration, the two groups differed markedly when they had longer sleep duration, with better cognitive performance observed for higher SES children.

Table 3
Relations Between Children's Sleep Duration and Cognitive Performance

Step and variables	β	R^2	ΔR^2	ΔF
Intellectual ability				
Step 1		.141	.141	4.43(5, 135)***
Race	.17*			
SES	.23**			
Step 2		.142	.001	0.14(1, 134)
Sleep duration	.03			
Step 3		.192	.051	4.15(2, 132)*
Race × Sleep Duration	−.21*			
SES × Sleep Duration	.21*			
Processing speed				
Step 1		.180	.180	5.97(5, 136)***
Race	−.09			
SES	.25**			
Step 2		.197	.017	2.82(1, 135)†
Sleep duration	.14†			
Step 3		.231	.034	2.95(2, 133)*
Race × Sleep Duration	−.16†			
SES × Sleep Duration	.17*			

Note. SES = socioeconomic status. Analyses controlled for child age, gender, and pubertal status.
†$p < .10$, *$p \leq .05$, **$p < .01$, ***$p < .001$.

Moderators of Relations Between Sleep Quality and Cognitive Performance

Relations between sleep activity and cognitive performance are presented in Table 4. After controlling for child characteristics (age, gender, and puberty status) and potential moderators (race, SES), sleep activity predicted unique variance in children's cognitive efficiency and processing speed, with increased activity related to worse cognitive outcomes. In addition, SES interacted with sleep activity in predicting children's working memory. The interaction (not shown) illustrated that the predicted means for working memory were very similar for children from higher and lower SES when their sleep activity was low. However, when sleep activity was high, higher SES children performed much better on the working memory tests than children from lower SES families.

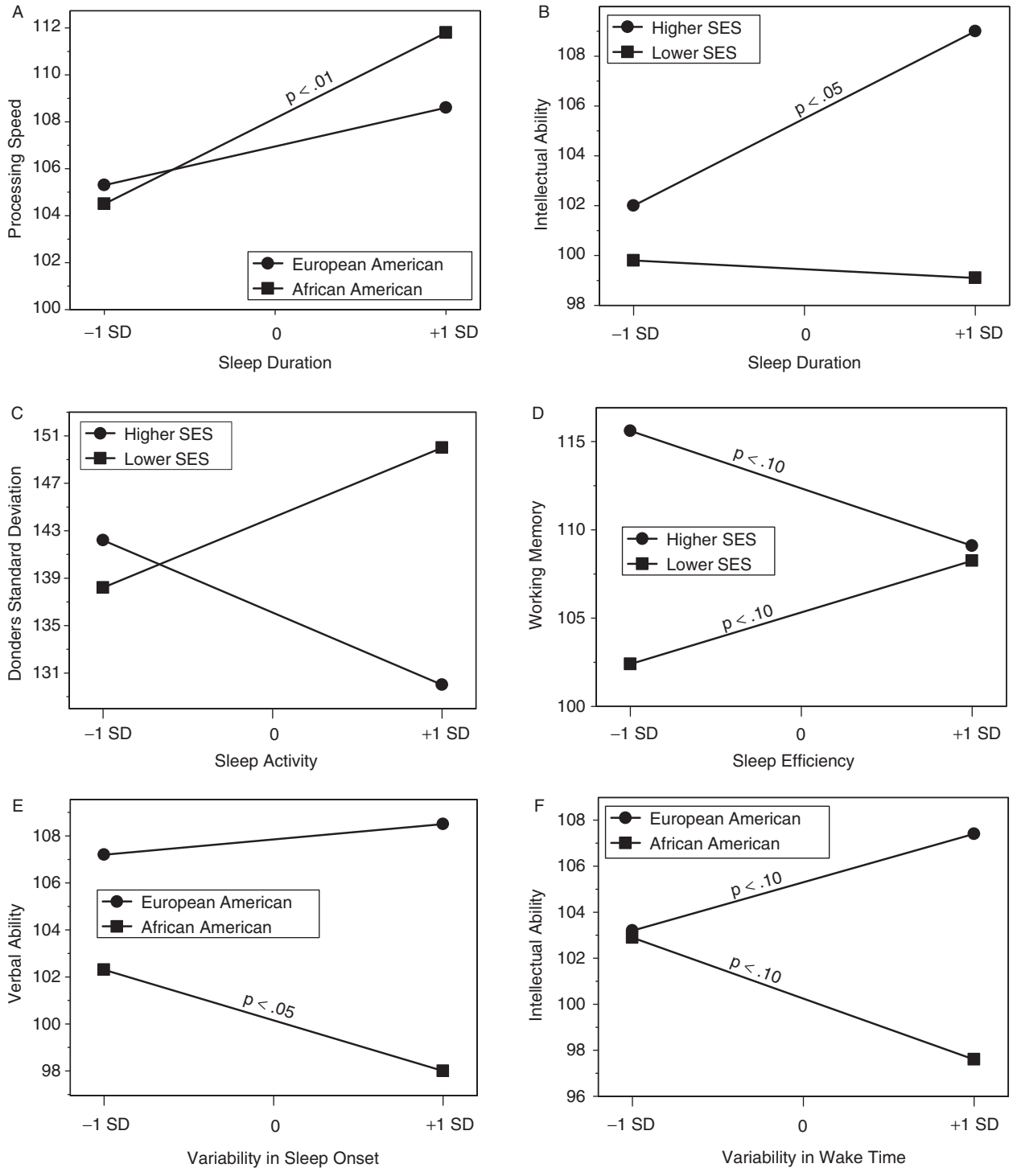

Figure 1. Race and socioeconomic status as moderators of relations between children's sleep and cognitive performance. For slopes that were significantly different from zero, or approached conventional levels of significance, the *p* value is presented next to the slope. Sleep variables are depicted on the x-axis and present either increased sleep duration, activity during sleep, better sleep efficiency, or increased variability in either sleep onset or wake time throughout the week of actigraphic assessment.

Furthermore, SES interacted with sleep activity in predicting performance on the RT task (Table 4). This interaction is presented in Figure 1c; neither slope was significantly different from zero. The graph illustrates that children from higher and lower SES families performed very similarly on the RT task

Table 4
Relations Between Children's Sleep Activity and Cognitive Performance

Step and variables	β	R^2	ΔR^2	ΔF
Cognitive efficiency				
Step 1		.092	.092	2.76(5, 137)*
Race	−.01			
SES	.18*			
Step 2		.117	.026	3.94(1, 136)*
Sleep activity	−.17*			
Step 3		.126	.008	0.64(2, 134)
Race × Sleep Activity	−.09			
SES × Sleep Activity	.09			
Processing speed				
Step 1		.180	.180	5.97(5, 136)***
Race	−.09			
SES	.25**			
Step 2		.244	.064	11.42(1, 135)**
Sleep activity	−.27**			
Step 3		.252	.008	0.69(2, 133)
Race × Sleep Activity	−.06			
SES × Sleep Activity	−.05			
Working memory				
Step 1		.099	.099	3.00(5, 136)*
Race	.02			
SES	.18*			
Step 2		.100	.000	0.12(1, 135)
Sleep activity	.01			
Step 3		.144	.044	3.45(2, 133)*
Race × Sleep Activity	−.13			
SES × Sleep Activity	.23**			
Donders standard deviation				
Step 1		.054	.054	1.38(5, 121)
Race	−.07			
SES	−.08			
Step 2		.056	.002	0.26(1, 120)
Sleep activity	.05			
Step 3		.097	.041	2.67(2, 118)†
Race × Sleep Activity	.04			
SES × Sleep Activity	−.22*			

Note. SES = socioeconomic status. Analyses controlled for child age, gender, and pubertal status.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

Table 5
Relations Between Children's Sleep Efficiency and Cognitive Performance

Step and variables	β	R^2	ΔR^2	ΔF
Working memory				
Step 1		.098	.098	2.58(5, 119)*
Race	.07			
SES	.15			
Step 2		.098	.000	0.00(1, 118)
Sleep efficiency	.00			
Step 3		.185	.087	6.17(2, 116)**
Race × Sleep Efficiency	.13			
SES × Sleep Efficiency	−.29**			
Donders standard deviation				
Step 1		.069	.069	1.77(5, 119)
Race	−.11			
SES	−.06			
Step 2		.071	.002	0.21(1, 118)
Sleep efficiency	−.04			
Step 3		.125	.055	3.62(2, 116)*
Race × Sleep Efficiency	.03			
SES × Sleep Efficiency	.23*			

Note. SES = socioeconomic status. Analyses controlled for child age, gender, and pubertal status.
*$p < .05$, **$p < .01$.

when sleep activity was low. However, when sleep activity was high, children from lower SES homes evidenced more variability in their performance on the RT task than those from higher SES homes.

Table 5 presents relations between sleep efficiency and cognitive performance. SES interacted with sleep efficiency in predicting children's working memory. This interaction is depicted in Figure 1d; the slopes approached conventional levels of statistical significance. The graph illustrates that children from higher and lower SES families had very similar working memory scores when they had higher levels of sleep efficiency. However, at lower levels of sleep efficiency, children from higher and lower SES backgrounds had very different predicted means for working memory; higher SES children performed around 1 *SD* higher on the working memory tests than those from lower SES families.

SES also interacted with sleep efficiency in predicting children's performance on the RT task (Table 5), and the interaction was very similar to that depicted in Figure 1d. Specifically, children from higher and lower SES backgrounds were very similar in their performance on the RT task when they had good sleep efficiency. Conversely, with lower levels of sleep efficiency, children from varying SES backgrounds differed in their performance, with lower SES children exhibiting increased variability.

Moderators of Relations Between Variability in Sleep and Cognitive Performance

Table 6 presents the results of hierarchical regressions testing for relations between variability in children's sleep schedule and cognitive performance. Race interacted with variability in sleep onset time in predicting either intellectual ability (graph not depicted) or verbal ability (Figure 1e); the two graphs were very similar. As shown in Figure 1e, greater variability in sleep onset time was related

Table 6
Relations Between Variability in Children's Sleep Schedule and Cognitive Performance

Step and variables	β	R^2	ΔR^2	ΔF
Intellectual ability				
Step 1		.142	.142	4.46(5, 135)***
Race	.14			
SES	.24**			
Step 2		.143	.001	0.19(1, 134)
Variability in sleep onset	−.04			
Step 3		.178	.035	2.81(2, 132)†
Race × Variability in Sleep Onset	.15†			
SES × Variability in Sleep Onset	.10			
Verbal ability				
Step 1		.175	.175	5.72(5, 135)***
Race	.35***			
SES	.13			
Step 2		.186	.012	1.90(1, 134)
Variability in sleep onset	−.11			
Step 3		.211	.024	2.04(2, 132)
Race × Variability in Sleep Onset	.17*			
SES × Variability in Sleep Onset	−.07			
Intellectual ability				
Step 1		.143	.143	4.45(5, 133)***
Race	.18*			
SES	.23**			
Step 2		.144	.000	0.06(1, 132)**
Variability in wake time	−.02			
Step 3		.196	.052	4.21(3, 130)*
Race × Variability in Wake Time	.22*			
SES × Variability in Wake Time	.10			

Note. SES = socioeconomic status. Analyses controlled for child age, gender, and pubertal status.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

significantly to decreased verbal ability for AA, but not EA children.

Furthermore, race interacted with variability in wake time in predicting intellectual ability (Table 6). This interaction is depicted graphically in Figure 1f; both slopes approached conventional levels of statistical significance. The graph illustrates almost identical levels of Intellectual Ability for AA and EA children when variability in wake time was low. However, when variability in wake time was high, AA children had lower intellectual ability scores.

Because very little research on this topic has been conducted, the study is necessarily exploratory to some extent. Accordingly, many moderator analyses were conducted, and a question can be raised as to whether accumulation of Type I error is problematic for interpretation of the results. Of the interaction analyses conducted, 5% would be expected to be significant by chance. A good many more than that were found to be significant, and the interactions show a clear pattern of effects, leading us not to display them all.

Post Hoc Analyses

To gain a better understanding of variables that may be associated with either race or SES that could have impacted the observed relations between these variables and sleep, some additional analyses were conducted. Data for these analyses were obtained from the daily diary that the child and parent kept for the week of actigraphy, and which were obtained during this week through nightly callings to the families. These analyses first focused on relations between race or SES and children's napping behavior, caffeine consumption, and home size and occupancy factors thought to be related to sleep. Next, we examined sleep in relation to the napping, caffeine consumption, and home factors that differed by race or SES.

Napping. Chi-square analysis indicated that in comparison with EAs (9% of children), AAs were more likely to nap during the weekend (28%), $\chi^2(1) = 5.36$, $p < .05$. For children who napped, the average daily duration of these naps was 25.00 min

Table 7
Partial Correlations Between Home Environment Variables and the Childs' Race, SES, and Sleep Performance

	Child has own bedroom	No. of persons sharing child's bedroom	Persons to bedrooms ratio
1. Race	.20*	−.23**	−.13
2. SES	.16†	−.19*	−.08
3. Sleep activity	−.06	.06	−.02
4. Sleep efficiency	.19*	−.19*	−.04
5. Sleep duration	.16*	−.21**	−.01
6. Variability in sleep onset	−.18*	.29***	.20*
7. Variability in wake time	−.12	.22**	.21**

Note. SES = socioeconomic status. Correlations are partial and controlled for the child's age, gender, and puberty status. Race was coded 0 for African Americans and 1 for European Americans.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

($SD = 35.70$) for EA, and 19.70 min ($SD = 20.9$) for AAs, which was not significantly different between the two groups. For weekdays, there were no differences between EAs and AAs in either frequency of napping (24% and 21%) or average daily duration of naps ($M = 14.76$ and $SD = 15.97$ for EAs; $M = 14.90$ and $SD = 15.79$ for AAs). SES was not related to napping. Partial correlations that controlled for the child's age, gender, and puberty status indicated that the duration of naps was not associated with sleep with the exception that the duration of napping on (a) weekends was related to lower sleep duration ($r = -.17$, $p < .05$), and (b) weekdays was associated with variability in morning wake time ($r = .22$, $p < .01$). Similar analyses for napping frequency yielded a significant relation to sleep duration ($r = -.22$, $p < .01$). Furthermore, the occurrence of any napping on weekends (0 vs. 1 scores) was related to decreased sleep duration ($r = -.23$, $p < .005$), and variability of both sleep onset and wake time (rs = .18 and .17, respectively, ps $< .05$). Investigations of relations between napping and cognitive performance did not yield any significant findings, with the exception that the occurrence of any naps on weekdays was related to better verbal ability ($r = .16$, $p < .05$) whereas weekend napping was associated with worse verbal ability scores ($r = -.17$, $p < .05$).

Bedroom sharing. AA children (39%) were more likely to share a bedroom than EAs (24%), $\chi^2(1) = 4.04$, $p < .05$; this association was still evident when SES was controlled in an analysis of covariance (ANCOVA), $F(1, 147) = 4.72$, $p < .05$. As shown in Table 7, race was related to the number of individuals who shared the child's bedroom, although not with the ratio of the number of people who lived in the house in relation to the number of bedrooms (density). On average, children shared their rooms with .34 individuals, mostly siblings ($SD = 0.61$; range = 0–4). The average ratio of number of individuals living in the residence by the number of bedrooms was 1.43 ($SD = 0.37$; range 0.75–3.00). Furthermore, SES was related to the likelihood that the child shared his or her bedroom as well as the total number of individuals sharing the child's room; no other SES variable was related to the home environment factors examined.

As shown in Table 7, children's sleep quality, duration, and variability in sleep schedule were related to the child having his or her own bedroom as well as the number of persons who shared the child's bedroom. Furthermore, the variability in sleep onset and wake time was related to the persons to bedrooms ratio in the child's residence. The cognitive functioning measures were not associated with any of the room sharing or density variables examined.

Caffeine consumption. In relation to caffeine consumption, an ANCOVA that controlled for SES indicated that in comparison with AAs, EAs consumed more caffeinated drinks during weekend mornings, $F(1, 127) = 5.12$, $p < .05$, weekend afternoons, $F(1, 126) = 2.77$, $p < .05$, and weekday afternoons, $F(1, 126) = 6.10$, $p < .05$. Means and SDs of number of caffeinated drinks for AAs and EAs, respectively, were .03 (.12) and .17 (.36) for weekend mornings, .25 (.36) and .55 (.59) for weekend afternoons, and .17 (.21) and .31 (.39) for weekday afternoons. SES was not associated with caffeine consumption. Only caffeine consumption during schoolday evenings (after 6 p.m.) was associated with sleep disruptions as evidenced in increased sleep activity ($r = .21$, $p < .05$) and decreased efficiency ($r = -.26$, $p < .005$). Caffeine intake was not related to any of the cognitive functioning measures.

Discussion

We examined relations between several dimensions of sleep and cognitive functioning in a large sample of healthy elementary school-age children. Building on a small but growing literature, the results demonstrate that several sleep dimensions were associated with different types and levels of cognitive functioning. Findings also extend knowledge by documenting differences between AA and EA children's sleep patterns and disruptions. More importantly, not all children who slept less, had more sleep disruptions, or showed less consistent sleep schedules were at an equal risk for lower cognitive

performance. Rather, after controlling for SES, race-related moderation effects indicated that whereas AA and EA children had very similar performance when sleep was more optimal, AA children were at increased risk for lower cognitive performance associated with sleep disruptions. Furthermore, the most robust SES-moderation findings illustrated that whereas children from lower and higher SES had similar cognitive functioning when they had good sleep quality and less variability in their sleep schedules, the two groups showed divergent scores when sleep was more disrupted, with higher SES children evidencing better cognitive performance.

Significant associations between sleep and cognitive measures were not very robust. Nevertheless, somewhat consistent patterns emerged. WJ III processing speed was related to both measures of sleep quality (activity and efficiency) as well as to sleep duration. In addition, cognitive efficiency was related to sleep activity, and verbal ability was related to variability in sleep onset, with better performance for children with a more consistent sleep schedule. Interestingly, and of importance, increased sleepiness was related to lower levels of cognitive performance on several WJ III scales including intellectual ability, verbal ability, working memory, and cognitive efficiency. Sleep/wake problems were also associated with lower levels of performance on several WJ III scales including verbal ability and working memory as well as slower and more variable RTs. These results offer some support for Dahl's (1996) hypothesis regarding working memory and executive functioning, but simpler tasks were also related to poorer sleep.

A question can be raised as to whether differences in cognitive functioning associated with sleep parameters are transient or more enduring. Intraindividual cognitive deficits associated with sleep deprivation in adults have been shown to ameliorate with restorative sleep (e.g., Dinges, Orne, Whitehouse, & Orne, 1987), but studies of the degree or speed of recovery of cognitive performance have not been carried out with children. It is easy to imagine how performance on tasks requiring attention, good working memory, and efficient executive processing may suffer with periodic sleep deprivation and then return to baseline levels after sleep. But our results show that cognitive measures thought to be less subject to day-to-day disruption, namely verbal and general intellectual ability, are related to sleep differences. The associations found in this study between simple speeded tasks and more global intelligence measures have been previously noted by cognitive researchers (e.g., Buckhalt, 1991; Buckhalt, Whang, & Fischman, 1998; Jensen, 1998). Impairment of the development of critical elementary processing skills that are sensitive to long-term sleep disruptions or sleep loss could have a cumulative deleterious effect on intellectual ability and school achievement (see Dahl, 2005). Children in our sample had cognitive processing scores in the average range. Further exploration of the questions we raise here should be performed with children whose performance falls in below- and above-average ranges.

The present sample was composed of a large percentage of AAs (33%) and a wide range of SES; SES and race were only modestly related. After controlling for SES, race-related differences were observed in children's sleep and indicated that, in comparison with EAs, AA children had less sleep activity, shorter sleep duration, more delayed sleep onset times on weekdays, more variability in sleep onset time, and higher levels of self-reported sleepiness and sleep/wake problems. Thus, almost all of these findings, with the sole exception of sleep activity, suggest better sleep in EA children. To examine variables that may account for sleep differences in children from the two racial groups, we conducted post hoc analyses. We found that AAs were more likely than EAs to nap during the weekend; no group differences in napping during the week were found. Furthermore, AA children were more likely to share a bedroom than EAs even when SES was controlled. Because SES was not related to napping behavior, and race was associated neither with the number of people who lived in the house nor with the number of bedrooms, it is likely that napping and room sharing may reflect cultural practices related to sleep in these AA and EA families. Note, however, that the total number of individuals sharing the child's room was related to SES, suggesting that economic factors may influence decisions regarding bedroom sharing. The scant available evidence regarding race-related effects in the sleep of normally developing children indicates that in comparison with EAs, AAs have shorter nighttime sleep durations, more napping, and more weekend ''oversleep,'' a differential between school-night and weekend-night sleep commonly considered indirect evidence of sleep deprivation (Crosby et al., 2005). As sleep measures in that study were derived from parent report, our findings corroborate and extend that finding based on objective assessments via actigraphy. Differences between AA and EA children's sleep are likely affected by the cultural, social, and economic milieu of these children. Furthermore, as our sample was drawn from a rural and suburban area of the Southeast U.S., there may be aspects of our results that are not generalizable to

other regions. Future research should be directed toward a thorough investigation of environmental factors that vary with culture and social class and may be associated with sleep differences.

In addition to the group differences between EA and AA children in relation to sleep, race moderated the association between sleep and cognitive functioning. These moderation effects were found even after controlling for several child characteristics related to sleep in the pediatric sleep medicine literature (age, gender, and puberty status) and SES. Thus, our assessment of moderation effects was very stringent. The most robust pattern of interaction effects illustrated that in comparison with EAs, AAs' cognitive performance was more negatively related to shorter sleep duration and more variability in sleep onset and wake time. The connection between sleep schedule variability and child functioning is consistent with Bates et al. (2002) who found that sleep duration variability and bedtime variability (parent reported) were related to preschool adjustment in a lower SES (predominantly White) sample. Furthermore, whereas cognitive performance was similar for children from both racial groups when they had more consistency in sleep schedule in relation to both sleep onset time and wake time, EA children had better scores with increasing variability in sleep schedule. These findings raise the possibility that sleep parameters may relate to differential performance on high-stakes academic tests. The plausibility of this interpretation is strengthened by the fact that after controlling for SES, race-related differences in children's cognitive performance were found for the intellectual ability and verbal ability scales of the WJ III. Similarly, race-related moderation effects of sleep were only evident in relation to intellectual ability and verbal ability.

SES was modestly related to all of the WJ III scales. However, after controlling for race, age, sex, and puberty status, moderation findings were evident for either (a) sleep quality (activity and efficiency) in relation to working memory and variability in RT (indicative of poor concentration and distractibility); and (b) sleep duration in relation to intellectual ability and processing speed. The pattern of moderation effects for (a) illustrates that whereas children from lower and higher SES had similar cognitive functioning scores when they had good sleep quality, the two groups showed divergent scores when sleep was more disrupted, with better performance for higher SES children. Children from higher and lower SES families had much more similar intellectual ability and processing speed scores with decreased sleep duration. However, with increased sleep duration, the two groups were different, with better cognitive functioning for higher SES children. This pattern of interaction effects was different from that observed for race-related effects and suggests that sleep duration may be related to better cognitive performance only for children from higher SES homes. For children from lower SES families, average levels of cognitive functioning were evident regardless of sleep duration. It could be that an increased number of stressors, environmental hazards, or less optimal medical care and nutrition associated with low SES and cognitive functioning, *independent* of those associated with sleep could be operating. Obviously, this possibility is speculative; yet it highlights the importance of future examinations of contextual factors associated with the sleep disruptions-cognitive functioning link.

Conclusions drawn from the present data cannot adequately address *why* AA and low SES children may be more vulnerable to the effects of sleep disruptions. However, the race related and the majority of the SES related moderation findings are generally consistent with the notion of health disparity (National Institute of Health, 2006), and suggest that not all children are at equal risk for cognitive difficulties when a fundamental aspect of biological regulation is disrupted.

It is also noteworthy that we found race- and SES-related effects even after controlling for specific factors that have been demonstrated to affect sleep problems and various outcomes in children, namely asthma and other related breathing problems. The rates of these conditions have been found to be elevated in AA compared with EA children (Fagan et al., 2001; Persky et al., 1998; Rosen et al., 2003), and further exploration of the questions investigated here should be performed with samples that include children affected by the conditions. We excluded children with chronic and acute illness of any type, but frequency of illness and quality of medical care may differ by SES. When night-time breathing is compromised, as it often is with upper respiratory infections, sleep is impaired, and daytime cognitive functioning decrements often follow. Linking a variety of health disparities associated with low SES to sleep and cognitive functioning in children warrants attention in future studies.

This study required the presence of two parents in the household. While little research has been performed that addresses family factors and child sleep, it is reasonable that variables such as consistency of children's sleep schedule could be related to the number of parents present. There are some disparities by race and SES regarding single parenthood

(Dunifon & Kowalski-Jones, 2002), and further differences in sleep patterns should be examined in families where only one parent or caregiver is present much of the time. Further research should investigate many aspects of family and home functioning that could feasibly be related to better and poorer sleep. It is possible that the presence of multiple stressors and health disparities may make some children more vulnerable to the negative sequelae of poor sleep than others. Resiliency and risk have multiple determinants, and it has long been suggested that multiple stressors may have effects that are independent, additive, and/or cumulative (e.g., Evans & English, 2002).

Children with a history of ADHD or LD were excluded from this study, and whether the present results extend to children with those conditions remains to be seen. ADHD has been linked conclusively to parent- and self-reported sleep problems, but less conclusively to objectively measured sleep (Cohen-Zion & Ancoli-Israel, 2004). More research is needed to supplant the limited number of studies, some with small and heterogeneous samples. It is also important to conduct more studies of the relationship of sleep and cognitive functioning across a wide span of cognitive functioning in developing children, including lower ranges excluded from this study.

We restricted our sample to prepubertal children because the onset of puberty is associated with changes in sleep patterns. Adolescents manifest a sleep phase delay that is thought to be driven by biological mechanisms and reinforced by social factors (Carskadon, Acebo, & Jenni, 2004). As postpubertal adolescents begin to tolerate later sleep onsets, but continue to wake early, they may be getting insufficient total sleep time and/or insufficient sleep in particular stages of sleep critical for daytime functioning. The link between sleep and cognition should be studied across the transition into and through puberty. The fact that AA children reach puberty earlier than EAs (Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001) may be important to consider in understanding mechanisms of sleep/cognition relationships. AA children in this study were considered prepubertal, but had slightly higher PDS scores. Those differences could be related to sleep duration differences, as there is a decline in sleep amount with age that may be related to biological, rather than chronological, age. Further research in this area would benefit from attention to measures of pubertal status and timing. We used the PDS (Petersen et al., 1988) in this study, the most widely used measure of self- or parent-report that does not have pictures or drawings (Dorn, Dahl, Woodward, & Biro, 2006). In a comprehensive guide to pubertal status and pubertal timing assessment, Dorn et al. (2006) conclude that even the ''gold standard'' of physician examination and Tanner (1962) stage rating has serious limitations, and development of more precise measures is called for.

While we have addressed several limitations thus far, one significant limitation requires further comment. The study was cross-sectional and correlational. Very few longitudinal studies of sleep parameters with typically developing children have been conducted (Jenni, Fuhrer, Iglowstein, Molinari, & Largo, 2005), and those have been descriptive survey-based studies of subjective reports of sleep parameters that have not included cognitive assessment. Conclusions regarding directionality of effects and changes in associations over time cannot be ascertained in the present study. In addition to experimental studies, development and relationships between systems associated with cognitive functioning and with sleep regulation should be studied longitudinally, with more sophisticated statistical techniques that allow greater inferential power, such as latent growth curve modeling (Little, Schnabel, & Baumert, 2000), and dynamical systems analysis (Boker & Graham, 1998; Granic & Patterson, 2006). The changes and relationships in these systems are likely very complex, and our analyses must be equal to the questions we ask.

In spite of these limitations, our results offer the first demonstration that the relationship between cognitive performance and sleep may differ among children of different racial/ethnic groups and SES levels. Pending replication and further investigation of environmental variables that may underlie these differences, directions for interventions to enhance cognitive performance of low-performing children may be forthcoming.

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