

Childhood Sleep Problems, Response Inhibition, and Alcohol and Drug Outcomes in Adolescence and Young Adulthood

Maria M. Wong, Kirk J. Brower, Joel T. Nigg, and Robert A. Zucker

Background: To our knowledge, no prospective studies examine the relationships among childhood sleep problems, adolescent executive functioning, and substance outcomes (i.e., substance use and substance-related problems). In this study, we examined whether childhood sleep problems predicted adolescent sleep problems and response inhibition. We also tested whether adolescent sleep problems and poor response inhibition mediated the relationship between childhood sleep problems and substance (alcohol and drug) outcomes in young adulthood.

Methods: Study participants were 292 boys and 94 girls ($M = 4.85$, $SD = 1.47$) from a community sample of high-risk families and controls.

Results: When compared to their counterparts, those with trouble sleeping in childhood were twice as likely to have the same problem in adolescence. Childhood overtiredness predicted poor response inhibition in adolescence. Persistent trouble sleeping from childhood to adolescence and response inhibition in adolescence mediated the relationship between childhood sleep problems and drug outcomes in young adulthood, whereas overtiredness in childhood directly predicted alcohol use outcomes and alcohol-related problems in young adulthood.

Conclusions: This is the first study showing a long-term relationship between childhood sleep measures and subsequent alcohol and drug outcomes. The developmental and clinical implications of these findings were discussed. Prevention and intervention programs may want to consider the role of sleep problems and response inhibition on substance use and abuse.

Key Words: Childhood Sleep Problems, Response Inhibition, Alcohol-Related Problems, Drug-Related Problems, Illicit Drug Use.

SLEEP PROBLEMS ARE a major public health issue in the U.S. An estimated 30 million Americans have frequent or chronic insomnia (National Institute of Health, 2006). In a recent national study, 51% of 6th to 12th graders reported feeling tired or sleepy and 31% reported having difficulty staying asleep at night at least once in week in the last 2 weeks (National Sleep Foundation, 2006). Another major public health issue in the U.S. is alcohol and drug use among adolescents and young adults. By the end of high school, 72% have consumed alcohol (more than a few sips), 55% reported having been drunk at least once, and 47% have used

at least one illicit drug (Johnston et al., 2008a). Among college students, 83% have tried alcohol, 41% have engaged in heavy drinking (five or more drinks in a row) at least once in the past 2 weeks, and 35% have used at least one illicit drug (Johnston et al., 2008b). This study examined the relationship between childhood sleep problems and alcohol and drug-related problems in young adulthood. We also tested whether sleep problems and response inhibition in adolescence were significant mediators of this relationship.

SLEEP PROBLEMS AND SUBSTANCE USE/ABUSE

The relationship between sleep problems and substance use/abuse has been demonstrated in both adults and adolescents. Insomnia has been shown to prospectively predict alcohol problems among some adults (Brower, 2001). In the Epidemiological Catchment Area study ($N = 7,954$), individuals with persistent insomnia at baseline were more likely than individuals without insomnia to experience the first onset of alcohol abuse or dependence 1 year later (Ford and Kame-row, 1989). This relationship holds even when the analyses were performed on adults without a psychiatric disorder at Time 1 ($N = 5,340$; Weissman et al., 1997). A study of health maintenance organization participants in southeast Michigan ($N = 979$) found a nonsignificant but positive trend for the effect of insomnia at baseline on onset of alcohol abuse or

From the Department of Psychology (MMW), Idaho State University, Pocatello, Idaho; Addiction Research Center (KJB, RAZ), Department of Psychiatry, University of Michigan, Ann Arbor, Michigan; Oregon Health and Science University (JTN), Portland, Oregon.

Received for publication October 9, 2009; accepted January 19, 2010.

Reprint requests: Maria M. Wong, Department of Psychology, Idaho State University, Pocatello, ID 83209-8112; Fax: 208-282-4832; E-mail: wongmari@isu.edu or

Robert A. Zucker, Addiction Research Center, Department of Psychiatry, University of Michigan, Rachel Upjohn Building, 4250 Plymouth Road, Ann Arbor, MI 48109-5740; Fax: 734-998-7992; E-mail: zuckerra@umich.edu.

Copyright © 2010 by the Research Society on Alcoholism.

DOI: 10.1111/j.1530-0277.2010.01178.x



dependence 3 years later (Breslau et al., 1996). Moreover, individuals with insomnia in the same study were significantly more likely to develop onset of nicotine dependence and drug abuse/dependence within the following 3 years.

Research on the relationship between childhood sleep problems and substance use in adolescents and young adults is limited. This knowledge gap has been recognized by the National Institute of Health (National Institute of Health, 2006). Three cross-sectional studies indicate that sleep patterns and problems are associated with substance use. In a World Health Organization survey of school children's health behavior, irregular sleep schedules, and perceived daytime tiredness were associated with increased use of cigarettes and alcohol in 1,057 15-year-old Finnish adolescents (Tynjala et al., 1997). Among a group of French secondary school students ($N = 763$), poor sleepers (self-reports of insomnia and the use of sleeping pills) were more likely than others to use alcohol, cigarettes, and illicit drugs (Vignau et al., 1997). Data from the U.S. National Household Survey on Drug Abuse ($N = 13,381$ adolescents) also indicated a similar relationship between sleep problems and substance use. Adolescents aged 12 to 17 who reported having trouble sleeping were more likely than others to use alcohol, cigarettes, and other illicit drugs (Johnson and Breslau, 2001).

To the best of our knowledge, our previous work (Wong et al., 2004, 2009) has been the only longitudinal study examining the relationship between childhood sleep problems and adolescent substance use. In one study, we found that controlling for parental alcoholism, maternal ratings of having trouble sleeping and overtiredness at 3 to 5 years of age predicted onset of any use of alcohol, marijuana, and illicit drugs, as well as onset of occasional or regular use of cigarettes by age 14 in 257 boys (Wong et al., 2004). A subsequent report based on 292 boys and 94 girls from the same sample found that sleep trouble and overtiredness at 3 to 8 years of age predicted onset of alcohol, cigarette, and marijuana use among boys and onset of alcohol use among girls in adolescence (Wong et al., 2009).

Two important questions remain unanswered. First, our previous studies indicated that trouble sleeping and overtiredness in childhood were related to early onset of substance use. Given this finding, do the same childhood measures predict problems related to alcohol and other drugs, e.g., binge drinking, driving under the influence of alcohol, missing school or work because of substance use? This is an important question to consider, given the serious impact that these problems have on the individuals who suffer from them. As our longitudinal study participants reached young adulthood (aged 18 to 20), onset of substance use became more common and incidents of substance-related problems increased. Thus, we were able to systematically examine the relationship between childhood sleep problems and substance-related problems at this later developmental period.

Second, what might mediate the relationship between childhood sleep problems and substance-related problems? In previous studies, we examined a number of potential mediators, including internalizing and externalizing problems, depres-

sion, attention problems, and aggression (Wong et al., 2004, 2009). Although trouble sleeping and overtiredness were significantly associated with internalizing and externalizing problems, depression and attention problems, none of these variables mediated the relationship between sleep variables and substance use. In this study, we tested whether adolescent sleep problems and response inhibition mediated the relationship between trouble sleeping/overtiredness in early childhood and substance-related problems in young adulthood. In the following section, we discuss the background research related to these two potential mediators.

CHILDHOOD SLEEP PROBLEMS AND ADOLESCENT SLEEP PROBLEMS

There are only limited data on the relationship between childhood sleep problems and adolescent sleep problems.

Existing research shows that sleep problems are persistent and there are stable individual differences in sleep problems over time. Such differences have been reported in studies with infants, young children, and adolescents. One study of 308 infants showed that 41% of infants with a sleep problem at 8 months still had a problem at 3 years old. The corresponding figure for those without a sleep problem was 26% (Zuckerman et al., 1987). Another study of 490 children reported a moderate but significant correlation ($r = 0.29$) between parental report of sleep problems at age 4 and age 15 (Gregory and O'Connor, 2002). A study of 704 adolescents found that among those who had a sleep problem at 13 years of age (e.g., having difficulty falling asleep, needing more sleep), 49% continued to have a problem at 15 years of age (Morrison et al., 1992).

Most of the existing studies on the persistence of sleep problems used information from only one source (e.g., parental report or self-report). Except for one study (Gregory and O'Connor, 2002), most studies assessed sleep in a period of approximately 3 years. In this study, we examined the relationship between maternal report of childhood sleep problems (aged 3 to 8) and adolescent self-report of sleep problems (aged 11 to 17). Unlike most previous studies, information about sleep problems was gathered from more than one source, and the assessment period covered a relatively long time period. If there was indeed a relationship between maternal report of childhood sleep problems and adolescent self-report of sleep problems, we wanted to ascertain whether adolescent sleep problems mediated the relationship between childhood sleep problems and alcohol and drug-related problems in young adulthood.

CHILDHOOD SLEEP PROBLEMS AND ADOLESCENT RESPONSE INHIBITION

Response inhibition is the ability to suppress a prepared response to perform a less-prepared response with respect to new information (Logan, 1994; Logan and Cowan, 1984). It is a cognitive process related to executive function (EF), a

higher-order cognitive construct involved in the planning, initiation, and regulation of goal-directed behavior (Miyake et al., 2000; Pennington and Ozonoff, 1996). Response inhibition is closely related to intentional behavior, planning, and self-regulation (Barkley, 1997).

Existing research showed that sleep deprivation, sleep fragmentation, and sleep problems affect cognitive processes, including executive functioning in adults (Durmer and Dinges, 2005; Pilcher and Huffcutt, 1996). There is evidence that sleep deprivation adversely affects inhibition or processes related to inhibition among adults, including the ability to suppress a prepotent response (Chuah et al., 2006), to detect error and to engage in error remedial action (Tsai et al., 2005), and to make good decisions involving distracting and unexpected information (Harrison and Horne, 2000).

Research on the long-term effects of sleep loss and sleep problems on EFs in children and adolescents are scant. Studies showed that sleep restriction and fragmented sleep adversely affect EFs. A study of 12 healthy adolescents found that after one night of sleep loss, performance on an addition task and a memory task was significantly lower than baseline (Carskadon et al., 1981). Another study compared 16 children who were randomly assigned to two groups—one group was restricted to have 5 hours of sleep; another group was allowed to have 11 hours of sleep. Children who slept for 5 hours had poorer performance on measures of verbal creativity and set shifting (Randazzo et al., 1998). One study reported that sleep extension and restriction significantly affect neurobehavioral functioning (Sadeh et al., 2002). Seventy-seven healthy children were asked to either restrict or extend their sleep by an hour for 3 consecutive nights. When compared to baseline measures, children in the sleep-extended group performed significantly better than baseline measures on a digit forward memory task and a continuous performance task that measured sustained visual attention, response inhibition, and motor speed. The performance of the sleep-restricted group and the control group remained stable. In a naturalistic study of 135 healthy children, fragmented sleep was associated with lower neurobehavioral functioning, particularly complex tasks that required sustained attention and behavioral inhibition (Sadeh et al., 2002).

The studies reviewed earlier suggest that sleep restriction and fragmentation have an adverse short-term effect on executive functioning, including tasks that require response inhibition. Children who experience frequent sleep restriction and fragmentation may not have the motivation or mental resources needed for tasks that required alertness and sustained attention. In the long run, these children may not develop habits or the ability to engage in behaviors that are necessary for executive functioning tasks, including response inhibition, planning, and problem solving. It is unclear whether sleep problems have a long-term effect on executive functioning processes such as response inhibition. One goal of the current study is to examine this long-term relationship.

Poor EF in general and poor response inhibition in particular have been linked to substance use and problems. Sons of

male alcoholics show greater deficits in EF than controls ($N = 44$; Peterson et al., 1992). Lower EF scores predicted increases in alcohol consumption 3 years later in 104 sons of alcoholics (Deckel and Hesselbrock, 1996). In a high-risk sample of 198 participants, children of alcoholics exhibited lower response inhibition than controls (Nigg et al., 2004). In 498 children of alcoholics and controls, lower response inhibition predicted number of alcohol and drug problems as well as number of illicit drugs use in adolescence (Nigg et al., 2006). In the same study, other EF tasks did not predict substance use outcomes independent of response inhibition.

Our review suggests that sleep deprivation and sleep problems are associated with impaired EFs and inhibitory processes. One question that remains unexamined is whether sleep problems also predict response inhibition prospectively over a long period of time. For instance, do children with sleep problems show poor response inhibition as adolescents? Most existing prospective studies on sleep and EFs are short term (lasting for a few days only). Long-term data are currently lacking. In this study, we examined whether childhood sleep problems (age 3 to 8) were associated with adolescent response inhibition (age 15 to 17). We also tested whether adolescent response inhibition mediated the effect of sleep problems on substance-related problems in young adulthood.

OVERVIEW

This study has two goals. First, we examined whether overtiredness and trouble sleeping in childhood predicted sleep problems and response inhibition in adolescence. Second, we examined whether adolescent sleep problems and response inhibition mediated the relationship between childhood overtiredness/sleep trouble and substance outcomes in young adulthood. These outcomes include binge drinking, blackouts after drinking, driving under the influence of alcohol, number of alcohol-related problems, number of illicit drugs used, and any illicit drug-related problems.

METHOD

Participants

The present study is part of an ongoing longitudinal family study of the development of risk for alcohol and other substance use disorders (Zucker and Fitzgerald, 1991; Zucker et al., 2000). The larger study recruited a population-based sample of 311 alcoholic men, their partners (whose substance use disorder was free to vary), and their initially 3- to 5-year-old sons. The 3- to 11-year-old daughters in the families were also invited to participate after the study began. The majority of these girls joined the project between 6 and 11 years of age.

Alcoholic men were identified by population sampling methods involving (i) a canvass of all courts in a four-county-wide area for drunk drivers with high blood alcohol levels ($BAL > 0.15\%$); (ii) a neighborhood canvass in the areas where the court-selected alcoholics lived to recruit additional alcoholics. The neighborhood canvass also recruited a control group of children and their families who resided in the same neighborhood as the alcoholic families, but whose parents had no lifetime history of substance abuse/dependence. Offspring of control families were age matched to the male child in the

alcoholic family residing in the same neighborhood. Both biological parents were required to be living together in the same household (either as married couples or domestic partners) and to have a 3- to 5-year-old son living with them at the time of recruitment. Presence of fetal alcohol syndrome was an exclusionary criterion. Analyses of benchmark indicators reported by Zucker and colleagues (2000) showed that the level of psychiatric comorbidity among alcoholic men in this sample is higher than that in the general U.S. population of alcoholics, but somewhat lower than that found in clinical populations.

The current sample consists of 292 boys and 94 girls and both of their biological parents. They were selected from the original study because data on both sleep problems and substance use were present. This represents 94% of the total sample. Seventy-five percent of participants (223 boys; 67 girls) had at least one parent who met a lifetime alcohol use disorder diagnosis when they were 3 to 8 years old, and 25% of participants were controls.

All families were Caucasian-American. Less than 4% of the population in the study sampling area that met inclusion criteria were non-Caucasian. Given the study's sample size, if non-Caucasian ethnic/racial groups were included, the number available would not permit any effective analysis to be performed. As there is an extensive literature showing a relationship between substance abuse and ethnic/racial status (Hasin et al., 2007; Kessler et al., 2005), including such variation in the study without being able to statistically model its effects would only contribute to error. Therefore, investigators originally opted to exclude this variation. The study thereafter recruited an additional sample of both African-American and Hispanic families using parallel recruitment criteria; however, offspring from these families are largely preadolescent and thus do not yet provide the endpoint data necessary for this study.

Procedure

Trained interviewers who were blind to family diagnostic status collected the data. The contact time for each family varied, depending on the data collection wave. Typically, each parent was involved for 9 to 10 hours and each child for seven hours spread over seven sessions. A variety of age-appropriate tasks (e.g., questionnaires, semi-structured interviews, and interactive tasks) were administered, and most of the contacts occurred in the families' homes. Special arrangement was made to collect data from families who had relocated. No families were lost because of relocation.

Participants and their parents were interviewed at 3-year intervals. Participants were 3 to 5 years old at Time 1, 6 to 8 years old at Time 2, 9 to 11 years old at Time 3, 12 to 14 years old at Time 4 and 15 to 17 years old at Time 5. Additionally, participants were interviewed annually between 11 and 17 years of age on questions regarding alcohol and drug use. The data presented in this article include six regular waves and seven annual waves of the study ending at age 17.

Measures

Sleep Problems. Sleep problems in childhood (3 to 8 years of age) were measured by maternal ratings on the Child Behavior Checklist (CBCL; Achenbach, 1991a). The CBCL is a widely used instrument that measures common behavioral problems. Its reliability and validity have been demonstrated. Two items were used to indicate sleep problems: having trouble sleeping, and overtiredness. Responses to each item were scored on a three-point rating scale (0 = not true; 1 = somewhat or sometimes true; 2 = very true or often true). A relatively small percentage of the sample had a score of 2 on either item (e.g., trouble sleeping at 3 to 8 years of age: 7.8% had a score of 1 and 3.6% had a score of 2; overtiredness at 3 to 8 years of age: 25.4% had a score of 1 and 1.6% had a score of 2). Therefore, each item was recoded as a dichotomous variable (0 = not true, 1 = sometimes or often true). The two items had a

significant relationship with one another, i.e., children who had trouble sleeping were more likely to be overtired than children who did not have trouble sleeping [$\chi^2(1) = 13.413, p < 0.001$].

Sleep problems in adolescence (11 to 17 years of age) were measured by the Youth Self Report (YSR; Achenbach, 1991b). Like the CBCL, the YSR is a widely used self report measuring behavioral problems. The same two items, having trouble sleeping and overtiredness, were used to indicate sleep problems. Responses were given on a three-point scale (0 = not true; 1 = somewhat or sometimes true; 2 = very true or often true) and were later recoded as dichotomous variables (0 = not true, 1 = sometimes or often true). In the latent growth modeling analyses, annual data of both dichotomous variables from 11 to 17 years of age were analyzed. In the mixed model analyses, data from 11 to 17 years of age were used to form two dichotomous variables, having trouble sleeping and overtiredness (0 = absence of problem between 11 and 17 years of age; 1 = presence of problem between 11 and 17 years of age). The two variables were significantly associated with one another [$\chi^2(1) = 38.399, p < 0.001$].

Response Inhibition. Response inhibition refers to the suppression of a prepotent motor response upon receiving goal-relevant information (Logan, 1994). We used the tracking version of the Stopping Task to measure response inhibition once between 15 and 17 years of age. Participants were presented with an "X" or "O" on a computer screen and were asked to press one of the computer keys as quickly as possible upon seeing the stimuli. They were asked to stop themselves from striking the keys upon hearing a tone following the stimulus on the screen. Stop signal reaction time (SSRT) was used as an index of response inhibition. SSRT was calculated by subtracting average stop signal delay from average go reaction time (Logan, 1994; Logan and Cowan, 1984).

Alcohol and Other Drug Use. Substance use outcomes and substance-related problems were assessed by the Drinking and other Drug Use History Questionnaire—Youth Version (DDHQ-Y; Zucker and Fitzgerald, 2002) and the Diagnostic Interview Schedule—Child Version (DISC; Shaffer et al., 2000) annually from 11 to 17 years of age and once between 18 and 20 years of age. All of the items in DDHQ-Y have been extensively used in a variety of survey and clinical settings. The questionnaire measures age of onset of alcohol and illicit drug use, the frequency and quantity of alcohol use and problems, and the frequency of other drug use and problems.

For the purpose of this article, six variables were examined. These include (i) alcohol-related variables—binge drinking, blackouts, driving under the influence of alcohol, and number of alcohol-related problems and (ii) drug-related variables—number of illicit drugs used and presence of drug-related problems.

We asked participants to indicate whether they had problems related to their drinking and drug use (e.g., having to miss school, got into trouble with my parents). A total of 31 problems were listed separately for drinking and drug use. For alcohol, we counted the number of problems reported by participants of 18 to 20 years of age. In addition, three items from the DDHQ-Y were selected for separate analyses because of their significance on the participants' health, safety, or daily functioning. They are binge drinking in the last 6 months, experiencing a blackout after drinking ("Had blackout, i.e., could not remember later what you had done while drinking"), and driving under the influence of alcohol ("Had driven a car when I had a good bit to drink"). All three are dichotomous variables; for each, a response of "0" implies the absence and "1" implies the presence of the behavior or experience by participants of 18 to 20 years of age.

With regards to drug-related problems, we examined the presence or absence of any problem (0 = no, 1 = yes) by participants of 18 to 20 years of age. Moreover, we asked participants to read through a list of 20 illicit drugs (e.g., marijuana, cocaine, methamphetamine)

and indicate whether they had used them. We then counted the total number of illicit drugs used by participants of 18 to 20 years of age. Past research has shown that early onset of drinking and other drug use are robust predictors of alcohol use disorders in adulthood (DeWit et al., 2000; Grant and Dawson, 1997).

Parental Alcoholism. Parental lifetime alcohol use disorder diagnosis (alcohol abuse or dependence) when the child was 3 to 8 years old was assessed by three instruments: the Short Michigan Alcohol Screening Test (Selzer et al., 1975), the Diagnostic Interview Schedule Version III (Robins et al., 1980), and the Drinking and Drug History Questionnaire (Zucker and Fitzgerald, 2002). Based on information collected by these instruments, a trained clinician made diagnoses of parental alcoholism using DSM-IV criteria. The availability of three sources of information collected over three different sessions, separated sometimes by as much as several months, served as an across method validity check on respondent replies. In cases of discrepant information, the data represented by the majority of information sources was used in establishing the diagnosis. Inter-rater reliability for the diagnosis was excellent ($\kappa = 0.81$). Children were coded as having an alcoholic parent if either parent met lifetime criteria for alcohol abuse or dependence at 3 to 8 years of age (0 = neither parent had a diagnosis; 1 = either parent had a diagnosis).

Figure 1 shows all measures used in the study.

Plan of Analysis

We used two-level mixed models for most analyses (Raudenbush and Bryk, 2002). Two-level mixed models take into account the clustering (nonindependent) nature of the sample. Some participants came from the same family. On average, each family had 1.77 children participated in the study. One set of analyses examined the relations between childhood sleep problems and adolescent sleep problems. A second set of analyses examined the relations between childhood sleep problems and adolescent response inhibition. In these analyses, Level-1 variables are maternal ratings of having trouble sleeping and overtiredness, gender (0 = female, 1 = male), and age of participants. Level-2 variables are lifetime parental alcoholism (0 = absence; 1 = presence). A third set of analyses examined how childhood sleep problems, adolescent sleep problems, and adolescent response inhibition may be related to substance outcomes in young adulthood. In these analyses, Level-1 variables are maternal ratings of having trouble sleeping and overtiredness, adolescent self-report of having trouble sleeping and overtiredness, gender (0 = female, 1 = male), and age of participants. Level-2 variables are lifetime parental alcoholism (0 = absence; 1 = presence).

We used two methods of the product of coefficient approach to test whether adolescent sleep problems and response inhibition mediated the relations between childhood sleep problems and substance outcomes (MacKinnon, 2008; MacKinnon et al., 2002). The first method involves testing whether the joint product of Z_α (α is the effect of sleep problems on response inhibition) and Z_β (β is the effect

of response inhibition on substance outcomes) is equal to zero ($H_0: Z_\alpha Z_\beta = 0$) (MacKinnon, 2008; Meeker et al., 1981). The mediated effect is significant if it exceeds the 95% critical value. The second method tested whether the 95% asymmetric confidence interval (CI) of $\alpha\beta$ includes 0 ($H_0: \alpha\beta = 0$). There is a significant mediated effect when the 95% CI does not include zero (MacKinnon, 2008; MacKinnon et al., 2002).

In addition to two-level mixed model analysis, we also used latent growth modeling analysis (McArdle and Epstein, 1987; Meredith and Tisak, 1990) to examine the relationship between childhood sleep problems and the development of adolescent sleep problems (age 11 to 17). As sleep trouble and overtiredness in adolescence were dichotomous variables (absence or presence of a problem), latent growth modeling was performed on their logits (log odds or logarithmic transformation of the odds of an event; $\text{odds} = \frac{\text{probability}}{1-\text{probability}}$). The outcome variables are logits of the probability of an adolescent sleep problem from 11 to 17 years of age. We used childhood sleep problems to predict the latent intercept (i.e., probability of having a particular sleep problem at age 11) and slope of adolescent sleep problems (i.e., rate of change of probability of a particular sleep problem from 11 to 17 years of age). In these analyses, we did not model the family effect. Previous research shows that the effect of clusters on latent growth models depends on cluster size and intraclass correlations (Muthen, 2000; Muthen and Satorra, 1995). Based on the rule of thumb suggested in those studies, we determine that the size of clusters (or families) in our sample is small and the potential lack of independence in the data for some families did not significantly bias the latent growth modeling analysis.

RESULTS

Descriptive Statistics

At 3 to 8 years of age, about one-tenth of children (11%) were rated by their mothers as having trouble sleeping and more than one quarter (27%) were rated as overtired. One-third (33%) of the children had either sleep problem. Children who were rated as having problems sleeping were also more likely to be rated as overtired and vice versa [$\chi^2(1) = 13.41, p < 0.001$]. There were no gender differences in sleep trouble [$\chi^2(1) = 1.922, p = 0.166$] or overtiredness [$\chi^2(1) = 0.200, p = 0.655$]. Having an alcoholic parent at 3 to 8 years of age did not significantly increase the risk of either problem (sleep trouble: $\chi^2(1) = 0.577, p = 0.448$; overtiredness: $\chi^2(1) = 0.001, p = 0.972$).

At 11 to 17 years of age, about one quarter of adolescents (26%) reported having trouble sleeping and approximately

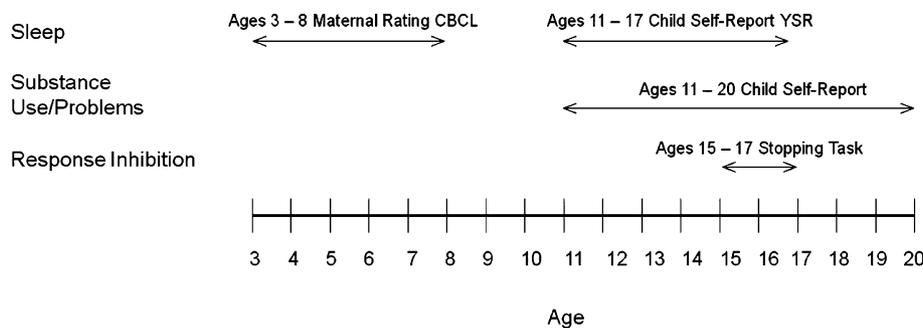


Fig. 1. Longitudinal measures of the study.

Table 1. Multilevel Model Predicting Trouble Sleeping, Overtiredness, and Response Inhibition in Adolescence

Variable/effect	Trouble sleeping	Overtired	Response inhibition
Fixed effects (parameter coefficients)			
Fixed effect intercept	-0.887 (0.407)	-0.504 (0.372)	218.758 (15.601)***
Level 1 variables			
Trouble sleeping at age 3 to 8	0.881 (0.378)* OR = (2.414)	0.156 (0.362)	9.536 (17.048)
Overtired at age 3 to 8	-0.018 (0.296)	0.019 (0.262)	25.437 (11.243)*
Age centered	-0.003 (0.142)	-0.129 (0.127)	-11.032 (5.250)*
Gender	-0.181 (0.299)	-0.336 (0.275)	23.948 (10.703)*
Level 2 variables			
Parental alcoholism	-0.124 (0.295)	0.298 (0.287)	15.055 (11.841)
Random effects (variance components)			
Level 1 effect, r_{ij} residual	0.042 (0.716)	0.006 (0.028)	5,289.895 (867.477)***
Level 2 intercept, u_{0j} intercept	-	-	1,835.408 (864.400)*

Numbers listed are parameter coefficients and their standard errors (in parentheses).

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

one-third of adolescents (32%) reported feeling overtired. The two sleep measures were associated with one another. Those who had trouble sleeping were more likely to feel overtired and vice versa [$\chi^2(1) = 38.40, p < 0.001$]. The two sleep measures were not associated with gender [sleep trouble: $\chi^2(1) = 0.17, p = 0.68$; overtiredness: $\chi^2(1) = 1.17, p = 0.28$] or parental alcoholism [sleep trouble: $\chi^2(1) = 0.09, p = 0.77$; overtiredness: $\chi^2(1) = 2.18, p = 0.14$].

By 18 to 20 years of age, 47% reported at least one binge drinking episode in the last 6 months, 26% had experienced blackouts, 12% had driven after consuming a significant amount of alcohol ("had driven a car when I had a good bit to drink"), 60% reported having had at least one alcohol-related problem; 62% reported having used illicit drugs; 24% reported having had at least one drug-related problem.

Childhood and Adolescent Sleep Trouble and Overtiredness

Results of mixed models using childhood sleep measures to predict adolescent sleep measures were presented in Table 1. Two-way interaction effects (e.g., having trouble sleeping X parental alcoholism, having trouble sleeping X gender, gender X parental alcoholism) were tested but none was significant and were therefore dropped from the analyses. Controlling for parental alcoholism, gender, age, and childhood overtiredness, maternal ratings of having trouble sleeping at 3 to 8 years of age were significantly related to self-report of having trouble sleeping at 11 to 17 years of age. Those who had trouble sleeping in childhood were 2.41 times more likely to have trouble sleeping in adolescence. In contrast, maternal ratings of overtiredness at 3 to 8 years of age were not associated with self-report of having trouble sleeping when other variables in the model were controlled for. Controlling for parental alcoholism, gender, and age, neither maternal ratings of having trouble sleeping nor overtiredness had a significant relationship with adolescent report of overtiredness.

In addition, we also used latent growth modeling analysis to examine whether maternal ratings of sleep trouble and overtir-

edness were related to the development of sleep trouble and overtiredness in adolescence. As sleep trouble and overtiredness were dichotomous variables, latent growth modeling was performed on their logits (log odds). The unconditional latent growth model of sleep trouble showed a good fit to the data, $\chi^2(120) = 67.718, p = 1.000$ (note: a nonsignificant chi-square value indicates a good fit). The mean intercept was significantly different from zero [$\hat{u}_\alpha = -1.722 (0.264), p < 0.001$]. In other words, the logit of sleep trouble at age 11 was estimated to be -1.722 (probability = $\frac{1}{1+e^{-\log \pi}}$), i.e., the estimated probability that an 11-year-old participant would report having sleep trouble is 0.151. However, the mean slope was not significantly different from zero, suggesting that across all participants the probability of reporting sleep trouble over time was quite stable [$\hat{u}_\beta = -0.121 (0.072), p = 0.09$]. The variance of both the intercept and slope estimates were significantly different from zero [$\hat{\sigma}_\alpha^2 = 5.196 (1.744), p < 0.01$; $\hat{\sigma}_\beta^2 = 0.273 (0.094), p < 0.01$], indicating there were significant individual differences on both the initial status and subsequent change. The conditional latent growth model tested whether parental alcoholism, gender, and sleep trouble in childhood predicted the latent growth trajectories. Neither parental alcoholism nor gender significantly predicted latent intercept [parental alcoholism: $\beta = 0.536 (0.479), p = 0.263$; gender: $\beta = 0.608 (0.469), p = 0.195$] or slope [parental alcoholism: $\beta = -0.106 (0.125), p = 0.398$; gender: $\beta = -0.239 (0.128), p = 0.061$] of sleep trouble. Sleep trouble in childhood significantly predicted both the initial status (i.e., age 11) [$\beta = 1.807 (0.635), p < 0.01$] and slope [$\beta = -0.368, (0.173), p < 0.05$] of sleep trouble. Model implied trajectories of sleep trouble from 11 to 17 years of age were plotted in Fig. 2. When compared to their counterparts, those who had trouble sleeping in childhood were 5.84 times more likely to report having such trouble at age 11. Such relationship appears to be weaker over time, as indicated by the decreasing slope. At 16 to 17 years of age, the two groups did not seem to be different on the probability of sleep trouble.

The unconditional latent growth model of overtiredness showed a good fit, $\chi^2(122) = 117.741, p = 0.592$. The mean

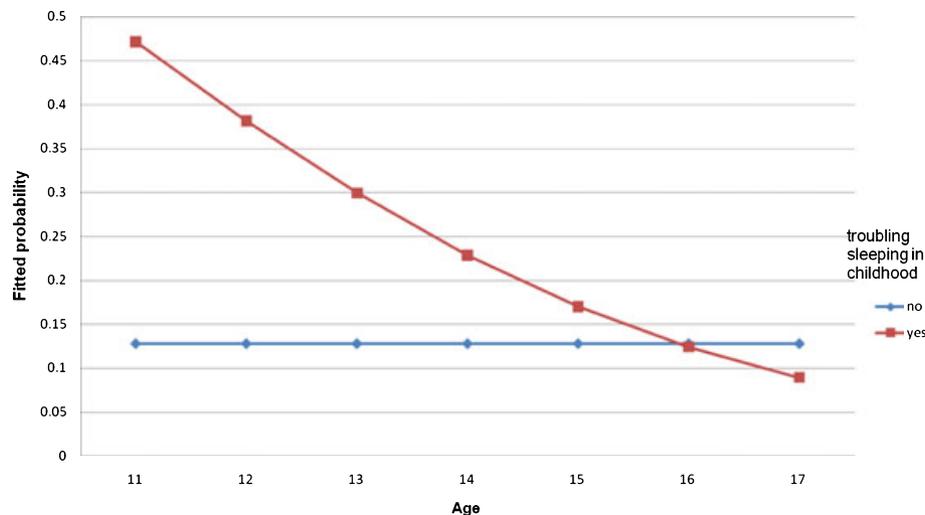


Fig. 2. Model-implied trajectories of having trouble sleeping in adolescence.

intercept of overtiredness was significantly different from zero [$\hat{u}_\alpha = -0.897$ (0.184), $p < 0.001$], i.e., the estimated probability that an 11-year-old participant would report being overtired is .290. However, the mean slope was not significantly different from zero [$\hat{u}_\beta = -0.012$ (0.045), $p = 0.795$], suggesting that across all participants the probability of reporting overtiredness over time was quite stable. The variance of both the intercept and slope estimates was significantly different from zero [$\hat{\sigma}_\alpha^2 = 3.528$ (1.228), $p < 0.01$; $\hat{\sigma}_\beta^2 = 0.1$ (0.05), $p < 0.01$], indicating that there were significant individual differences on both the initial status and subsequent change. The conditional latent growth model showed that parental alcoholism, gender, or overtiredness in childhood did not predict either the latent intercept [parental alcoholism: $\beta = 0.269$ (0.569), $p = 0.637$; gender: $\beta = -0.119$ (0.568), $p = 0.834$; childhood overtiredness: $\beta = -0.108$ (0.569), $p = 0.637$] or slope of overtiredness in adolescence [parental alcoholism: $\beta = -0.087$ (0.136), $p = 0.525$; gender: $\beta = 0.002$ (0.145), $p = 0.988$; childhood overtiredness: $\beta = 0.187$ (0.132), $p = 0.156$].

Childhood Sleep Problems and Response Inhibition in Adolescence

Results of mixed models using childhood sleep problems to predict response inhibition (Stop Signal Reaction Time or SSRT) are presented in Table 1. Again, two-way interaction effects were tested but none was significant. The intercept was 218.76 units, which was the estimated SSRT for a 15-year-old female control (i.e., Non-COA) who did not have trouble sleeping and was not overtired at 3 to 8 years of age. Age had a negative relationship with response inhibition, indicating that older adolescents had shorter SSRT. Gender had a positive relationship with response inhibition, suggesting that adolescent boys had a longer SSRT than adolescent girls. Controlling for parental alcoholism, age and gender, maternal ratings of overtiredness at 3 to 8 years of age significantly

predicted response inhibition at 15 to 17 years of age. Children who were rated as overtired had poorer response inhibition in adolescence (on the average, SSRT was 25.44 units longer) than children who were not rated as overtired. Controlling for overtiredness, having trouble sleeping in childhood was not significantly related to adolescent response inhibition.

Sleep Problems, Response Inhibition, and Substance Outcomes

Multilevel models results using childhood and adolescent sleep measures, adolescent response inhibition to predict substance outcomes in young adulthood are presented in Tables 2 and 3. We first used sleep problems, response inhibition, parental alcoholism, age of participant and gender to predict each substance-use outcome and substance-related problem variable. The parameter coefficients obtained from the mixed models analysis were then used to test whether adolescent response inhibition or sleep measures significantly mediated the effects of childhood sleep measures on each outcome in young adulthood.

Binge Drinking and Alcohol-Related Problems. Controlling for age, parental alcoholism, adolescent sleep measures and response inhibition, overtiredness at 3 to 8 years of age significantly predicted all four alcohol outcomes in emerging adulthood, including presence of binge drinking, blackouts, “having driven a car after having a good bit to drink,” and number of alcohol-related problems. When compared to their counterparts, those who were overtired at 3 to 8 years of age were 2.8 times more likely to have engaged in binge drinking, 2.0 times more likely to have experienced blackouts from drinking, and 2.3 times more likely to have driven a car under the influence of alcohol by 18 to 20 years of age. On the average, they reported more alcohol problems in emerging adulthood than participants who were not overtired at 3 to 8 years of age. Sleep measures and response inhibition

Table 2. Multilevel Model Predicting Alcohol-Related Problems in Young Adulthood

Variable/effect	Binge drinking	Blackouts	Driving under the influence of alcohol	Number of alcohol problems
Fixed effects (parameter coefficients)				
Fixed effect intercept	-1.328 (0.873)	-1.486 (0.531)**	-5.256 (1.209)***	2.117 (1.555)
Level 1 variables				
Trouble sleeping at age 3 to 8	-0.186 (0.532)	-0.811 (0.516)	-0.904 (0.794)	-1.831 (1.263)
Overtired at age 3 to 8	1.019 (0.441) (OR = 2.77)*	0.692 (0.297) (OR = 1.998)*	0.842 (0.394) (OR = 2.321)*	1.676 (0.842)*
Trouble sleeping at age 12 to 17	0.406 (0.467)	0.003 (0.312)	0.484 (0.431)	0.915 (0.858)
Overtired at age 12 to 17	0.185 (0.407)	-0.001 (0.001)	-0.331 (0.439)	1.135 (0.797)
Response inhibition	-0.002 (0.002)	-0.001 (0.002)		0.0001 (0.004)
Age centered	0.574 (0.237) (OR = 1.776)*	0.270 (0.143)	0.253 (0.175)	0.891 (0.350)*
Gender	1.65 (0.485) (OR = 3.205)*	-0.342 (0.305)	0.532 (0.485)	-0.968 (0.866)*
Level 2 variables				
Parental alcoholism	0.305 (0.417)	0.543 (0.335)	2.749 (1.080)*	2.023 (0.835)*
Random effects (variance components)				
Level 1 effect, r_{ij} residual	0.598 (1.374)	0.003 (0.013)	0.001 (0.001)	27.533 (4.468)***
Level 2 intercept, u_{0j} intercept	-	-	-	5.601 (4.060)

Numbers listed are parameter coefficients and their standard errors (in parentheses).

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 3. Multilevel Model Predicting Drug-Related Problems in Young Adulthood

Variable/effect	Number of illicit drugs	Any drug-related problems
Fixed effects (parameter coefficients)		
Fixed effect intercept	-0.627 (0.466)	3.545 (0.632)***
Level 1 variables		
Trouble sleeping at age 3 to 8	-0.484 (0.400)	-0.024 (0.469)
Overtired at age 3 to 8	0.120 (0.264)	-0.058 (0.333)
Trouble sleeping at age 12 to 17	0.373 (0.269)	0.893 (0.299) (OR = 2.442)**
Overtired at age 12 to 17	0.001 (0.251)	0.267 (0.291)
Response inhibition	0.004 (0.001)**	0.002 (0.001)
Age centered	0.402 (0.115)***	0.283 (0.128) (OR = 1.328)*
Gender	0.187 (0.268)	0.330 (0.324)
Level 2 variables		
Parental alcoholism	0.542 (0.264)*	1.184 (0.395)**
Random effects (variance components)		
Level 1 effect, r_{ij} residual	3.541 (0.531)***	0.000 (0.003)
Level 2 intercept, u_{0j} intercept	0.211 (0.457)	-

Numbers listed are parameter coefficients and their standard errors (in parentheses).

* $p \leq 0.05$. ** $p \leq 0.01$. *** $p \leq 0.001$.

in adolescence did not significantly predict any alcohol outcomes.

Illicit Drug Use and Drug-Related Problems. Controlling for age, parental alcoholism, and sleep measures, adolescent response inhibition significantly predicted the number of illicit drugs used by 18 to 20 years of age. Adolescents with longer SSRT were more likely to report using more illicit drugs. For every standard deviation increase in SSRT (i.e., 88 seconds), number of illicit drugs was estimated to increase by 0.44 (88*0.005). This effect may seem small in absolute terms. However, given that the mean number of illicit drugs used by 18 to 20 years of age is only 1.5, the effect of response inhibition cannot be ignored. Additionally, having trouble sleeping in adolescence significantly predicted the presence of any drug-related problems by emerging

adulthood. Controlling for other factors in the model, those who had trouble sleeping in adolescence were 2.4 times more likely to have had a drug-related problem by emerging adulthood.

Did response inhibition or having trouble sleeping in adolescence mediate the effect of childhood sleep measures on drug outcomes? Two sets of mediation analyses were performed to address this question. The first set of analyses used response inhibition as a potential mediator. The second set of analyses used having trouble sleeping in adolescence as a potential mediator.

Findings from the first set of mediation analyses show that there was a statistically significant effect of childhood overtiredness on adolescent response inhibition ($\hat{\alpha} = 25.437$, $S_{\hat{\alpha}} = 11.243$, $p < 0.05$). There was also a significant effect of response inhibition on number of illicit drugs used

($\hat{\beta} = 0.004$, $S_{\hat{\beta}} = 0.001$, $p < 0.01$). Response inhibition in adolescence significantly mediated the effect of overtiredness in childhood on number of illicit drug used in young adulthood ($Z_{\alpha} = 2.263$, $Z_{\beta} = 4$, $Z_{\alpha}Z_{\beta} = 8.908$, $p < 0.05$; 95% asymmetric CI = 0.014 to 0.216).

Findings from the second set of mediation analyses show that there was a significant effect of having trouble sleeping in childhood on having trouble sleeping in adolescence ($\hat{\alpha} = 0.881$, $S_{\hat{\alpha}} = 0.378$, OR = 2.414, $p < 0.05$). There was also a significant effect of having trouble sleeping in adolescence on the presence of drug-related problems ($\hat{\beta} = 0.893$, $S_{\hat{\beta}} = 0.299$, OR = 2.442, $p < 0.01$). Having trouble sleeping in adolescence significantly mediated the effects of having trouble sleeping in childhood on drug-related problems ($Z_{\alpha} = 2.331$, $Z_{\beta} = 2.987$, $Z_{\alpha}Z_{\beta} = 6.963$, $p < 0.05$; 95% asymmetric CI = 0.104 to 1.759).

DISCUSSION

Childhood sleep trouble and overtiredness appear to have both direct and indirect relationships with substance use and substance-related problems in young adulthood. To the best of our knowledge, this is the first study showing a long-term relationship between childhood sleep measures and subsequent alcohol and drug outcomes. "Having trouble sleeping" in childhood predicted a higher probability of "having trouble sleeping" in adolescence, which in turn predicted the presence of drug-related problems in young adulthood. Overtiredness in childhood predicted lower response inhibition in adolescence, which in turn predicted the number of illicit drugs used. Overtiredness in childhood also directly predicted the presence of binge drinking, blackouts, driving after drinking alcohol, and number of lifetime alcohol problems in young adulthood.

Our previous work has shown that childhood sleep problems were associated with age of onset of substance use in adolescence (Wong et al., 2004, 2009). In those studies, overtiredness and having trouble sleeping predicted onset of alcohol, cigarette, and illicit drugs use among boys, and onset of alcohol use only among girls. As our participants became young adults, an association between childhood overtiredness or having trouble sleeping and substance-related problems emerged. The effect was the same for both male and female participants (there was no gender X sleep measure interaction). Although we cannot be certain that childhood overtiredness or trouble sleeping are causally linked to alcohol and illicit drug use and related problems, the results of this study underscore the developmental significance of childhood sleep problems. Children with sleep problems appear to be more vulnerable to the risks of substance use as well as problem use.

Maternal ratings of sleep trouble in childhood predicted self-report of sleep trouble in adolescence over an extended period of time (over 10 years). The relationship holds whether we examine "absence or presence of having trouble sleeping in adolescence" (as in the two-level mixed modeling analysis) or the developmental trajectories of having trouble sleeping

over time (as in the latent growth curve analysis). These findings provide preliminary evidence that maternal ratings of sleep trouble have some predictive validity. Our results also indicate that sleep problems persist over time, a finding that is consistent with past studies (Gregory and O'Connor, 2002; Kataria et al., 1987; Zuckerman et al., 1987). Thus, children who had trouble sleeping were more likely to experience the same problem in adolescence. "Having trouble sleeping" in adolescence in turn predicted the presence of any drug-related problems. Having trouble sleeping in adolescence significantly mediated the effect of having trouble sleeping in childhood on substance outcomes.

We did not provide data regarding changes in maternal ratings of sleep problems over time. Future studies might compare the developmental trajectories of sleep problems in maternal ratings and self-reports and examine whether the two types of trajectories are related to one another in meaningful ways. Research could also examine the similarities and differences of subjective report of sleep problems (e.g., parental ratings, self-reports) with more objective measures (e.g., data from actigraphs and polysomnography).

Maternal ratings of overtiredness had no relationship with adolescent report of overtiredness. According to a study conducted by the National Sleep Foundation (2006), more than one half (56%) of adolescents aged 11 to 17 reported that they got less sleep than they needed to feel their best and 51% said they felt too tired or sleepy during the day. Feeling overtired appears to be very common among U.S. adolescents. Changes in sleep architecture (e.g., decreases in rapid eye movement and slow wave sleep) (Carskadon et al., 1981; Wolfson and Carskadon, 1998) and sleep habits in adolescence (e.g., developing irregular sleep habits, not getting enough sleep) (National Sleep Foundation, 2006) may have resulted in overtiredness among many respondents, not only those with early childhood sleep problems. Such effects would serve to attenuate our results; the fact that they did is another reason to have confidence in these findings.

Maternal ratings of overtiredness in childhood significantly predicted lower response inhibition. "Tiredness" may imply any or all of the three following states—(i) central nervous system fatigue, i.e., mental exhaustion with impairment in concentration and thinking; (ii) physical fatigue, i.e., a sense of physical exhaustion; and (iii) sleepiness, i.e., an urge to sleep (Moldofsky and MacFarlane, 2005). There are many possible causes of childhood overtiredness, some of which (e.g., inadequate nutrition) may have nothing to do with sleep. However, it is useful to note that childhood overtiredness had a highly significant relationship with "having trouble sleeping" in both childhood (maternal rating) and adolescence (self-report). Seen in this way, the relationship between overtiredness and response inhibition are consistent with previous studies showing that sleep problems (e.g., sleep deprivation, sleep fragmentation) have a negative impact on inhibitory processes (Randazzo et al., 1998; Sadeh et al., 2002, 2003). This study is the first one to show a relationship between overtiredness and response inhibition over a span of

more than 10 years and during an interval when great developmental changes are taking place.

Response inhibition is a component of EF. It involves the ability to interrupt a prepared (prepotent) response while taking into account information relevant to the actor's goal (Logan, 1994). Thus, it is an essential element of self-regulation (Barkley, 1997; Logan, 1994). Self-regulatory processes including inhibitory processes require mental effort (Muraven et al., 1998). If overtiredness implies physical and mental fatigue and self-regulation requires sustained mental effort, the relation between overtiredness and response inhibition seems logical. This study highlights the possibility that such relations persist over time. To the extent that response inhibition is related to impulse control (Logan et al., 1997), delay of gratification (Mischel et al., 1989), sustained attention (Barkley, 1997; Nigg, 2000), and goal-oriented behavior (Nigg, 2000), childhood sleep problems may have long-term implications for the development of such abilities.

Lower levels of response inhibition in adolescence predicted the number of illicit drugs used. The mediating effect of response inhibition is significant, indicating that overtiredness had an indirect effect on the outcomes via poor response inhibition. Overtired children, regardless of their gender and their parents' alcoholism status, were more likely than their counterparts to develop impaired response inhibition, which was associated with using more illicit drugs. Studies have repeatedly linked poor EF and response inhibition to substance use and related problems (Deckel and Hesselbrock, 1996; Nigg et al., 2006; Peterson et al., 1992). Our findings are consistent with those studies and underscore the importance of understanding the sleep problems-response inhibition pathway to substance use and abuse.

Overtiredness in childhood had a direct relationship with alcohol outcomes, including binge drinking, blackouts, driving under the influence of alcohol, and number of alcohol-related problems. It is unclear what factors may explain this direct relationship. There are at least two possible explanations. First, individuals who are overtired may be more likely to use alcohol to relieve its physical discomfort or distress. The desire to relieve discomfort may lead some participants to excessive drinking and poor decision making, resulting in problems such as binge drinking, blackouts, and driving under the influence of alcohol. Second, parents may perceive hyperactivity and restlessness in young children as overtiredness. Childhood hyperactivity and restlessness is an important risk factor for alcohol use and alcohol-related problems (Caspi et al., 1996; Molina and Pelham, 2003).

It is not clear why alcohol and drug outcomes were predicted by different sleep items. The results, however, were not completely contradictory. For example, trouble sleeping at 3 to 8 years of age predicted neither alcohol nor drug outcomes. Likewise, overtiredness at 12 to 17 years of age predicted neither alcohol nor drug outcomes. It is true, however, that overtiredness at 3 to 8 years of age predicted all of the alcohol outcome variables and none of the drug outcome variables. Likewise, trouble sleeping at 12 to 17 years of age predicted

drug-related problems but none of the alcohol outcomes. Our data cannot explain these discrepancies. What they do show is that the two early childhood sleep items have different relationships with alcohol and drug outcomes. Overtiredness in childhood has a direct relationship with alcohol outcomes. Additionally, it predicted lower response inhibition in adolescence, which had a positive relationship with the number of illicit drugs used. In contrast, having trouble sleeping in childhood had an indirect relationship with drug-related problems, mediated by having trouble sleeping in adolescence. In other words, only persistent trouble sleeping from childhood to adolescence was associated with drug-related problems. It is possible, therefore, that the two sleep items operate through different pathways to affect the risk for adverse alcohol and drug outcomes in adolescence.

Our findings have several implications for prevention and intervention programming. First, these findings underscore the importance of increasing public awareness of the significance of sleep problems in children and of their potential effect on response inhibition and later substance use and abuse. Parents, educators, and counselors are encouraged to help children deal with sleep deprivation and poor sleep. Seeking help from health care professionals may also be necessary to deal with persistent sleep problems. Second, substance abuse prevention and intervention programs need to consider the role of both sleep problems and response inhibition as possible core mechanisms operating earlier in the pathway to substance abuse. Thus, an appropriate intervention target would be to educate youth about the potentially serious consequences of sleep problems. Skills related to developing better sleep hygiene and self-control could also easily and appropriately be taught. Recent research on sleep intervention trials among adolescents, including those who abuse substances, shows promising results (Haynes et al., 2006; Moseley and Gradisar, 2009; Stevens et al., 2007). Third, health care providers may need to become more sensitized to the potential serious ramifications of childhood sleep problems and provide appropriate treatment or referral for sleep disturbances when clinically indicated. Such treatments would include sleep hygiene, cognitive-behavioral therapy, and/or medication.

Clearly, the longitudinal relation between sleep problems and response inhibition observed here needs to be replicated in other studies. An issue that remains to be addressed is whether the sleep problems-response inhibition link has an effect on other psychiatric problems such as depression and anxiety. Another issue is whether childhood sleep problems affect other types of EFs besides response inhibition.

The current study has several limitations. First, our analyses did not include any information prior to 3 to 8 years of age. Data pertaining to prenatal development and sleep-wakefulness information in the first 3 years of life are necessary to thoroughly characterize the developmental pathways related to the emergence of sleep problems in early childhood. Second, our sample of alcoholic and control families was deliberately selected so that offspring were at the middle-to-high end of the risk continuum for alcohol-use

disorders. Moreover, the numbers of alcoholic and control families, as well as the number of boys and girls were unequal. Thus findings in this study need to be evaluated in other samples at high risk for substance abuse using a balanced design, as well as in general population samples. Third, our measures of sleep problems were limited to two items and thus did not fully capture the spectrum of childhood sleep problems. In addition, the sleep problem indicators we used were maternal ratings. Although earlier work by our group has shown that maternal sleep ratings predicted adolescent substance use even when one important maternal factor—her depression—was controlled (Wong et al., 2004); it is still essential to establish that the observed differences reflect true sleep problems, rather than a parent's subjective experience of them. Similarly, an adolescents' self-report of trouble sleeping might be influenced by their mother's perception of their childhood sleep. Studies using polysomnographic or actigraphic measures in addition to sleep questionnaires are needed. Finally, our study lacks data on caffeine use, one of the most common psychoactive substances in both children and adults (Roehrs and Roth, 2000, 2008; Temple, 2009). Caffeine use may disrupt sleep and increase daytime sleepiness (Roehrs and Roth, 2000) and has also been linked to use of both tobacco (Istvan and Matarazzo, 1984; Martin et al., 2008) and alcohol (Istvan and Matarazzo, 1984).

This study provides preliminary evidence on the relationships between childhood sleep problems and substance use and substance-related problems in young adulthood. Childhood sleep problems appear to have both direct and indirect effects (via sleep trouble and response inhibition in adolescence) on subsequent substance-related outcomes. Examining the effects of sleep problems during this period of time may contribute to a greater understanding of the many developmental issues confronting children, adolescents, and young adults, including the important decisions about the use of alcohol and other drugs that lead to problems.

ACKNOWLEDGMENTS

This work was supported in part by grants from the National Institute on Alcohol Abuse and Alcoholism to M.M. Wong (R21 AA016851), K.J. Brower (K24 AA00304), and R.A. Zucker (R37 AA07065 and R01 AA12217). We are indebted to all participating families for their willingness to engage in the study. We are grateful to Susan Refior, Director of Field Operations in the Michigan Longitudinal Study, for her commitment and skill in maintaining this study's viability over a long time. Last but not least, we also thank Deirdre Conroy for her comments on the interpretation and discussion of the actigraphy data.

REFERENCES

Achenbach T (1991a) Manual for the Child Behavior Checklist/4–18 and 1991 Profile. University of Vermont Department of Psychiatry, Burlington, VT.

- Achenbach T (1991b) Manual for the Youth Self-Report Form and 1991 Profile. University of Vermont Department of Psychiatry, Burlington, VT.
- Barkley RA (1997) Behavioral inhibition, sustained attention, and executive functioning: constructing a unifying theory of ADHD. *Psychol Bull* 121:65–73.
- Breslau N, Roth T, Rosenthal L, Andreski P (1996) Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol Psychiatry* 39:411–418.
- Brower KJ (2001) Alcohol's effects on sleep in alcoholics. *Alcohol Res Health* 25:110–125.
- Carskadon MA, Harvey K, Dement WC (1981) Acute restriction of nocturnal sleep in children. *Percept Mot Skills* 53:103–112.
- Caspi A, Moffitt TE, Newman DL, Silva PA (1996) Behavioral observations at age 3 predict adult psychiatric disorders: longitudinal evidence from a birth cohort. *Arch Gen Psychiatry* 53:1033–1039.
- Chuah YML, Venkatraman V, Dinges DF, Chee MWL (2006) The neural basis of interindividual variability in inhibitory efficiency after sleep deprivation. *J Neurosci* 26:7156–7162.
- Deckel AW, Hesselbrock V (1996) Behavioral and cognitive measurements predict scores on the MAST: a 3-year prospective study. *Alcohol Clin Exp Res* 20:1173–1178.
- DeWit DJ, Adlaf EM, Offord DR, Ogborne AC (2000) Age at first alcohol use: a risk factor for the development of alcohol disorders. *Am J Psychiatry* 157:745–750.
- Durmer JS, Dinges DW (2005) Neurocognitive consequences of sleep deprivation. *Semin Neurol* 25:117–129.
- Ford DE, Kamerow DB (1989) Epidemiologic study of sleep disturbance and psychiatric disorders. *J Am Med Assoc* 262:1479–1484.
- Grant BF, Dawson DA (1997) Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. *J Subst Abuse* 9:103–110.
- Gregory AM, O'Connor TG (2002) Sleep problems in childhood: a longitudinal study of developmental change and association with behavioral problems. *J Am Acad Child Adolesc Psychiatry* 41:964–971.
- Harrison Y, Horne JA (2000) The impact of sleep deprivation on decision making: a review. *J Exp Psychol Appl* 6:236–249.
- Hasin DS, Stinson FS, Ogburn E, Grant BF (2007) Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Arch Gen Psychiatry* 64:830–842.
- Haynes PL, Bootzin RR, Smith L, Cousins J, Cameron M, Stevens S (2006) Sleep and aggression in substance-abusing adolescents: results from an integrative behavioral sleep-treatment pilot program. *Sleep* 29:512–520.
- Istvan J, Matarazzo J (1984) Tobacco, alcohol, and caffeine use: a review of their interrelationships. *Psychol Bull* 95:301–326.
- Johnson EO, Breslau N (2001) Sleep problems and substance use in adolescence. *Drug Alcohol Depend* 64:1–7.
- Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE (2008a) Monitoring the Future: National Survey Results on Drug Use, 1975–2002. Volume I: Secondary School Students. National Institute on Drug Abuse, Bethesda, MD.
- Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE (2008b) Monitoring the Future: National Survey Results on Drug Use, 1975–2002, Volume II: College Students and Adults Ages 19–45. National Institute on Drug Abuse, Bethesda, MD.
- Kataria S, Swanson MS, Trevathan GE (1987) Persistence of sleep disturbances in preschool children. *J Pediatr* 110:642–646.
- Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE (2005) Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry* 62:593–602.
- Logan GD (1994) On the ability to inhibit thought and action: a users' guide to the stop signal paradigm, in *Inhibitory Processes in Attention, Memory, and Language* (Dagenbach D, Carr TH eds), pp. 189–239. Academic Press, San Diego, CA.

- Logan GD, Cowan WB (1984) On the ability to inhibit thought and action: a theory of act and control. *Psychol Rev* 91:295–327.
- Logan GD, Schachar RJ, Tannock R (1997) Impulsivity and inhibitory control. *Psychol Sci* 8:60–64.
- MacKinnon DP (2008) *Introduction to Statistical Mediation Analysis*. Lawrence Erlbaum Associates, New York.
- MacKinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V (2002) A comparison of methods to test mediation and other intervening variable effects. *Psychol Methods* 7:83–104.
- Martin CA, Cook C, Woodring JH, Burkhardt G, Guenther G, Omar HA, Kelly TH (2008) Caffeine use: association with nicotine use, aggression, and other psychopathology in psychiatric and pediatric outpatient adolescents. *Sci World* 22:512–516.
- McArdle JJ, Epstein D (1987) Latent growth curves within developmental structural equation models. *Child Dev* 58:110–133.
- Meeker WQ, Cornwell LW, Aroian LA (1981) The product of two normally distributed random variables. *Selected Tables in Mathematical Statistics, Vol. VII*. American Mathematical Society, Providence, RI.
- Meredith W, Tisak J (1990) Latent curve analysis. *Psychometrika* 55:107–121.
- Mischel W, Shoda Y, Rodriguez ML (1989) Delay of gratification in children. *Science* 244:933–938.
- Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A (2000) The unity and diversity of executive functions and their contributions to complex 'frontal lobe' tasks: a latent variable analysis. *Cogn Psychol* 41:49–100.
- Moldofsky H, MacFarlane JG (2005) Fibromyalgia and chronic fatigue syndromes, in *Principles and Practice of Sleep Medicine* (Kryger MH, Dement WC eds), pp 1225–1236. Saunders, Philadelphia, PA.
- Molina BSG, Pelham WE (2003) Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. *J Abnorm Psychol* 112:497–507.
- Morrison D, McGee R, Stanton W (1992) Sleep problems in adolescence. *J Am Acad Child Adolesc Psychiatry* 31:94–99.
- Moseley L, Gradisar M (2009) Evaluation of a school-based intervention for adolescent sleep problems. *Sleep* 32:334–341.
- Muraven M, Tice DM, Baumeister RF (1998) Self-control as a limited resource: regulatory depletion patterns. *J Pers Soc Psychol* 74:774–789.
- Muthen B (2000) Methodological issues in random coefficient growth modeling using a latent variable framework: applications to the development of heavy drinking ages 18–37, in *Multivariate Applications in Substance Use Research: New Methods for New Questions* (Rose JS, Chassin L, Presson CC, Sherman SJ eds), pp 113–140. Erlbaum, Mahwah, NJ.
- Muthen BO, Satorra A (1995) Complex sample data in structural equation modeling. *Sociol Methodol* 25:267–316.
- National Institute of Health (2006) Public Announcement. PA-06-238. National Institute of Health, Bethesda, MD.
- National Sleep Foundation (2006) *Sleep in America Poll*. National Sleep Foundation, Washington, DC.
- Nigg JT (2000) On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychol Bull* 126:220–246.
- Nigg JT, Glass JM, Poon E, Wong MM, Fitzgerald HE, Puttler LI, Jester JM, Adams KM, Zucker RA (2004) Neuropsychological executive functioning in children at elevated risk for alcoholism: findings in early adolescence. *J Abnorm Psychol* 113:302–314.
- Nigg JT, Wong MM, Martel MM, Jester JM, Puttler LI, Glass J, Adams KM, Fitzgerald HE, Zucker RA (2006) Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *J Am Acad Child Adolesc Psychiatry* 45:468–475.
- Pennington BF, Ozonoff S (1996) Executive functions and developmental psychopathology. *J Child Psychol Psychiatr* 37:51–87.
- Peterson J, Finn P, Pihl RO (1992) Cognitive dysfunction and the inherited predisposition to alcoholism. *J Stud Alcohol* 53:154–160.
- Pilcher JJ, Huffcutt AJ (1996) Effects of sleep deprivation on performance: a meta-analysis. *J Sleep Res Sleep Med* 19:318–326.
- Randazzo AC, Muehlbach MJ, Schweitzer PK, Walsh JK (1998) Cognitive function following acute sleep restriction in children ages 10–14. *Sleep* 21:861–868.
- Raudenbush SW, Bryk AS (2002) *Hierarchical Linear Models: Applications and Data Analysis Methods*. Sage, Newbury Park, CA.
- Robins LN, Helzer JE, Croughan JL, Ratcliff KS (1980) The NIMH Diagnostic Interview Schedule: Its History, Characteristics and Validity. Washington University School of Medicine, St Louis, MO.
- Roehrs TA, Roth T (2008) Caffeine: sleep and daytime sleepiness. *Sleep Medicine Reviews* 12:153–162.
- Roehrs TA, Roth T (2000) Depressants, sedatives, and hypnotics, in *Encyclopedia of Psychology, Vol 2* (Kazdin AE ed), pp 470–471. American Psychological Association, Oxford University Press, Washington, DC and New York, NY.
- Sadeh A, Gruber R, Raviv A (2002) Sleep, neurobehavioral functioning, and behavior problems in school-age children. *Child Dev* 73:405–417.
- Sadeh A, Gruber R, Raviv A (2003) The effects of sleep restriction and extension on school-age children: what a difference an hour makes. *Child Dev* 74:444–455.
- Selzer ML, Vinokur A, van Rooijen L (1975) A self-administered Short Michigan Alcoholism Screening Test (SMAST). *J Stud Alcohol* 36:117–126.
- Shaffer D, Fisher P, Lucas C, Dulcan MK, Schwab-Stone M (2000) NIMH Diagnostic Interview Schedule for Children, Version IV (NIMH DISC-IV): description, differences from previous versions, and reliability of some common diagnoses. *J Am Acad Child Adolesc Psychiatry* 39:28–38.
- Stevens S, Haynes P, Ruiz B, Bootzin R (2007) Effects of a behavioral sleep medicine intervention on trauma symptoms in adolescents recently treated for substance abuse. *Subst Abuse* 28:21–31.
- Temple J (2009) Caffeine use in children: what we know, what we have left to learn, and why we should worry. *Neurosci Biobehav Rev* 33:793–806.
- Tsai LL, Young HY, Hsieh S, Lee CS (2005) Impairment of error monitoring following sleep deprivation. *Sleep* 28:707–713.
- Tynjala J, Kannas L, Levalahti E (1997) Perceived tiredness among adolescents and its association with sleep habits and use of psychoactive substances. *J Sleep Res* 6:189–198.
- Vignau J, Bailly D, Duhamel A, Vervaecke P, Beuscart R, Collinet C (1997) Epidemiologic study of sleep quality and troubles in French secondary school adolescents. *J Adolesc Health* 21:343–350.
- Weissman MM, Greenwald S, Nino-Murcia G, Dement WC (1997) The morbidity of insomnia uncomplicated by psychiatric disorders. *Gen Hosp Psychiatry* 19:245–250.
- Wolfson AR, Carskadon MA (1998) Sleep schedules and daytime functioning in adolescents. *Child Dev* 69:875–887.
- Wong MM, Brower KJ, Fitzgerald HE, Zucker RA (2004) Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. *Alcohol Clin Exp Res* 28:578–587.
- Wong MM, Brower KJ, Zucker RA (2009) Childhood sleep problems, early onset of substance use and behavioral problems in adolescence. *Sleep Med* 10:787–796.
- Zucker RA, Fitzgerald HE (1991) Early developmental factors and risk for alcohol problems. *Alcohol Health Res World* 15:18–24.
- Zucker RA, Fitzgerald HE (2002) Family Study of Risk for Alcoholism Over the Life Course. Appendix 9.3 Assessment protocol: Description of instruments and copies of contact schedules.
- Zucker RA, Fitzgerald HE, Refior SK, Puttler LI, Pallas DM, Ellis DA (2000) The clinical and social ecology of childhood for children of alcoholics: description of a study and implications for a differentiated social policy, in *Children of Addiction: Research, Health, and Policy Issues* (Fitzgerald HE, Lester BM, Zuckerman BS eds), pp 109–141. Routledge Falmer, New York.
- Zuckerman B, Stevenson J, Bailey V (1987) Sleep problems in early childhood: continuities, predictive factors, and behavioral correlates. *Pediatrics* 80:664–671.