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Research Article

Sleep Patterns in Children of Alcoholics and the Relationship with Parental Reports

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Abstract

Parental ratings of poor sleep have been associated with early onset of substance use and substance-related problems during adolescence and young adulthood. Children of alcohol-dependent parents have an increased risk for depression as well as substance-related problems. We conducted the current study to describe sleep -wake patterns in children of alcoholics (COAs) compared to non-alcoholic families (NCOAs), to explore the relationships among sleep assessment methods by groups, and to report rates of agreement between methods by group. We assessed subjective (sleep diaries) and objective (actigraphy-measured) sleep in children with (N=68) and without (N=24) a parental history of alcohol use disorder between 7.2 and 12.9 (mean 10.2 +/- 1.2) years of age. Children were instructed to complete the sleep diaries with assistance from parents only if needed. Parents provided descriptions of their child's sleep in a Pediatric Sleep Questionnaire (PSQ). Results showed that COAs reported sleeping slightly less time at night and were more likely to nap during the day. Actigraphy captured fewer hours of sleep and more nighttime motor activity. Sleep diary variables were highly correlated with parent reports on the PSQ, except for bedtimes in some COAs, which were later than parent's estimations.

Conclusion: School- aged COAs showed small but significant differences in their sleep characteristics compared to NCOAs. COA self-reported sleep diaries agreed with parental reports, but differed significantly from actigraphy measurements of sleep continuity. Insufficient sleep in COAs may be an additional risk factor as they approach adolescence.

Keywords: Sleep; Children; Napping; Alcoholism; Actigraphy; Behavior; Sleep diaries

Abbreviations

COA: Children of Alcoholic; NCOA: Not a Child of an Alcoholic; SOL: Sleep Onset Latency; WASO: Wake time After Sleep Onset; TST: Total Sleep Time; SE: Sleep Efficiency

Introduction

Several studies have now shown that sleep disturbances at an early age can predict psychiatric disorders, such as depression [1-4] suicide [5-7] and drug and alcohol use [8-10]. The etiology of sleep problems in children are multifactorial, but can be associated with social and cultural family environment as well as marital conflict [11]. Sleep and family environment has been evaluated over time in children [12,13] however, whether the sleep disruption is an endogenous phenomenon (i.e. a genetic influence), an exogenous one (i.e. chaotic home environment), or both, is still unclear. To our knowledge, few studies have examined sleep in children of an alcoholic parent. Objective sleep measures, such as polysomnography (PSG) between in children of alcoholics and healthy controls have not revealed differences in objective markers, such as sleep stages [14,15]. This study sought to understand subjective sleep patterns in children of alcoholics and parental reports of their children's sleep in this understudied population.

Identifying sleep problems early on in life may have important implications for prevention and intervention for mood and substance use disorders. There are a myriad of potential factors that could contribute to the early onset of substance use, including, additive and interactive genetic variations, environmental factors, and childhood psychiatric disorders. Genetic factors account for about 50% of the risk for alcoholism [16]. Children of alcohol-dependent parents have an increased risk for depression as well as substance-related problems [17]. Differences in sleep or circadian rhythms may predispose to early substance use in children deemed at high risk [18], but it is not clear how such differences predispose to the development of substance related symptoms once drinking has begun.

Methodological examination of sleep in the pediatric population

The use of actigraphy is a method to assess normal and disturbed sleep-wake patterns in children. It is easy to use in the home environment compared to PSG. Actigraphy has been well validated as a way to measure sleep-wake patterns, sleep quality, and quantity in normal healthy children [19,20]. Studies on actigraphy in children (ages ranging from infants to 12 years of age) are typically accompanied by a sleep diary completed by the parent(s) [19,21-24]. High correlations have been found between actigraphy and parent reported sleep start and stop times [24] and with "trouble sleeping"

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[25], but parents also overestimate their child's total sleep time and underestimate wake time after sleep onset and nighttime awakenings [24,26]. When compared to polysomnography, actigraphy is poor at detecting wake time after sleep onset [20,27]. No study prior to the current study has assessed children of alcoholic's own estimations of their sleep time using a sleep diary and compared this to parental reports.

The current study

One of the limitations in studies assessing the relationship between sleep and behavior in children is that sleep characteristics are often only reported by the parent and the sleep problem is defined by response to a few items on a questionnaire [10,28-31]. While a number of recent studies have utilized actigraphy to examine the impact of home environment on sleep actigraphy was often used at nighttime only [12,32-36] instead of the more accepted 24 hours a day for at least one week. The aims of the current study were as follows:

Aim 1: To describe sleep-wake patterns in children of alcoholics (COAs). We hypothesized that sleep would be worse in COAs compared to non-COAs (NCOAs).

Aim 2: To explore the relationships among sleep variables in parent and child. We hypothesized that there would be a significant difference between children's estimates of sleep variables from their parents and from actigraphy.

Material and Methods

Sample recruitment

Participants were recruited from the Michigan Longitudinal Study (MLS), an ongoing, prospective community-recruited study of families with parental alcohol use disorder along with a contrast sample of nonalcoholic families drawn from the same neighborhoods [37,38]. The study has been ongoing for approximately 25 years. Families in which the target child exhibited signs of fetal alcohol syndrome (FAS) were excluded from the original ascertainment. Exclusionary FAS characteristics included prenatal or postnatal growth retardation or both, central nervous system involvement, and characteristic facial dysmorphology [39,40]. Full details on the prospective assessment and data collection protocol in the MLS can be found elsewhere [37].

MLS participants were originally recruited via (a) drunk driving records of all district courts in the four counties surrounding Lansing, Michigan, a medium size Midwestern city; and (b) community canvassing in the neighborhoods where the court alcoholics and control families lived. The court alcoholic sample consists of convicted male drunk drivers with a blood alcohol concentration of 0.15% or higher (0.12% or higher if this was a second or more documented drinking-related legal problem), who had a biological son between the ages of 3-5 currently living with them, and who were living with the boy's biological mother at the time of first contact. Mother's substance use disorder history was not constrained. Of the drunk drivers identified through the courts, 79% agreed to be contacted and 92% of those agreed to participate in the study. Control families were recruited from the same neighborhoods where the alcoholic families resided. This way, many other variables are a function of their diagnosis, including their selection of the neighborhood. In this context, if one controls for socioeconomic status (SES), and lower SES is a result of the alcoholism i.e., downward mobility [41] then one is removing some of the alcoholism-specific variance. The families were interviewed at 3-year intervals, beginning when children were 3-5 years old (Time1: 3-5 years old, Time 2: 6-8 years old, Time 3: 9-11 years old, Time 4: 12-14 years old). Sample retention rate was approximately 85 %. This study sample includes children ages 9-11 participating in the Time 3 sample interval of the longitudinal study. The study was approved by the University of Michigan Review Board (IRB) as well as the IRB from Michigan State University, a collaborating institution for the larger MLS. Informed consent was obtained from parents and all children assented to participation in the study.

Study sample

Parents

All alcoholic families were required to have at least one parent with a lifetime history of an alcohol use disorder. Diagnoses were made via an interview with a study psychologist or social worker. In the families from the MLS (n=57), the diagnosis of lifetime history of alcohol dependence was made based on a combination of criteria from the Diagnostic and Statistical Manual, 4th edition (DSM-IV) as determined from the Diagnostic Interview Schedule.

Children

The study population consisted of 68 COAs (age range 7.2-12.9 years; mean=10.1, SD=1.2) and 24 NCOAs (age range 8-12.9 years; mean=10.2, SD=1.3). The study was restricted to this age range because the median age of a first drink in the U.S. is 14 [42] and our goal was to capture sleep-wake patterns before drinking behavior began. For all participants, inclusion criteria were willingness and ability to complete the study protocol. Exclusion criteria included any medical problem that restricted the child's mobility, significant previous or concurrent medical illness, lifetime history of any head injury with loss of consciousness, independent /intrinsic sleep disorder based on history, a current diagnosis of any Axis I disorder according to the DSM-IV criteria, use of stimulants, and the presence of either fetal alcohol syndrome or any fetal alcohol effects. No child had current psychiatric or developmental disorder. No child was taking any medication at the time of the study.

Study measurements

Daily sleep diaries

The sleep diary packet included 7 pages. Each page reflected sleep patterns for each night. The diary was explained to each child directly with the mother, father, or legal guardian present. At least one adult took responsibility for assisting the child in completing the diary as needed. Each morning, children (with guidance from their parents only if requested), were asked to record the day and date, the time when lights were turned out the previous night ("What time was lights out"), how long it took to fall asleep after the lights were turned out (sleep onset latency, SOL), rise time ("The time you got out of bed"), quality of sleep (the child circled the words good, fair, or poor), number of nighttime awakenings, total wake time after sleep onset in the night (WASO) ("If you woke up, how long were you awake for each time", and napping for the previous day ("If you nap during

Table 1: Sample characteristics.

Variable	NCOAs (n=24) Mean (SD)	COAs (n=68) Mean (SD)	t	*p*[a]
Age	10.2 (1.3)	10.1 (1.2)	.36	.72
Sex (% male)	75.0	58.8	---	.15
Race (% white)	82.6	45.3	---	.01
Tanner stage[b]	1.6 (0.9)	2.0 (1.0)	-1.6	.12
Body Mass Index	23.5(6.1)	23.4 (5.2)	-.26	.80
Alcoholism (% father)	0	50.0	---	<.001
Alcoholism (% mother)	0	10.3	---	<.001
Alcoholism (% both)	0	39.7	---	<.001
Parent sleep problems (count)[c]				
--Sleep apnea	2	2		
--"Sleep disturbance"[d]	1	11		
--Restless Legs Syndrome	0	1		
--Hypnotic dependence	0	1		
--Hypersomnolence	0	1		
--Parasomnia	1	0		
Total count	4	16		
Child sleep problems[e]				
Insomnia[f]	.52	.91	-1.5	.14
Restlessness[f]	.44	.56	-.48	.63
Parasomnias[f]	.39	.49	-.60	.56
Daytime sleepiness[f]	.53	.77	-1.1	.28
Breathing problems in sleep[g]	2.9	2.0	.82	.41

Note: [a]=Likelihood ratio is presented for Chi Square test. [b]=Tanner refers to a measure of pubertal development with scores ranging from 1-5, (1=no clinical signs of puberty to 5=completed puberty). [c]=number of sleep disorders reported by both parents about themselves on the Pediatric Sleep Questionnaire (PSQ). [d]= "Sleep disturbance" is a term used to group various descriptions of disturbed sleep e.g. "insomnia" or "sleep problems." [e]=rated by parents on the PSQ. [f] =Means reflect parental response to the respective sleep problems in a 0=no, 1= yes format. [g]= In addition to no/yes questions about breathing in sleep, means include responses to a 4 item-Likert scale inquiring about child's hyperactivity symptoms during the day, 0=does not apply, 1=applies just a little, 2=applies quite a bit, and 3=definitely applies.

the day about what time did the nap start and end." The child also indicated on the bottom of the sleep diary whether the actigraph was removed, for how long, for what purpose, and if any problems arose with the watch. Unlike a typical sleep diary, this diary was colorful and had cartoon pictures of children sleeping to make it more fun for the child. All children maintained their usual sleep-/wake schedule. Two dependent variables were calculated from the sleep diaries: total sleep time (TST) and sleep efficiency (SE %). TST was obtained by subtracting SOL and WASO from reported time in bed (TIB). SE is a percentage calculated from dividing TST by TIB multiplied by 100.

Actigraphy

The actiwatch (Actiwatch-L, Mini Mitter, Respironics, Bend, OR) was worn on the non-dominant wrist of each participant for at least five days. Actigraphs were set to begin recording data shortly after the meeting with the study coordinator (typically between the hours of 9 am and 5 pm). Epoch length was set to 1 minute for all recordings. Activity data were derived from the number of movements that exceeded a threshold of 0.01 g (gravitational force) per minute of recording. A sleep epoch was defined as a minute in which movements did not exceed 80counts/minute. Given that light is the primary zeitgeber to the endogenous circadian pacemaker; we were interested in characterizing the amount of light exposure across the day and sleep period between the two groups. Therefore, the actiwatches were equipped with a photoconductive cell to record light level exposure > 10 lux. Actigraphy was used to provide information about sleep-wake habits, sleep continuity (SOL, WASO, TST, and SE %), light exposure (daytime light and sleep time light), and level of activity (activity during daytime and activity during sleep time) and amplitude of rest/activity cycles (amplitude) expressed in z-scores.

Actigraphy data was downloaded using an Actireader™ device (Actiware 5 Philips/Respironics, Bend, OR). Activity and light levels were obtained for active (day) and sleep (night) periods. An "active" period is defined by the average of all valid physical activity counts per 1 minute from the start time to the end time of the given interval. Average daily light exposure was defined by the average of all valid light data for all 1-min epochs from the start time to the end time of the given period. All analyses were conducted using SPSS version 17.0 (PASW, Inc., San Ramon, CA).

The pediatric sleep questionnaire (PSQ)

The PSQ [43] is a 45-item instrument that is completed by the parent and used to identify several types of sleep problems, particularly sleep-related breathing problems when polysomnography is not feasible. The PSQ will also be referred herein as the parent completed questionnaire. The PSQ was also used in this study to rule out the presence of sleep-related breathing disorders. The questionnaire was explained directly to the parent(s), and they were asked to complete it at home at a time that was most convenient. The child did not complete any part of the PSQ. There are 22 items that relate to sleep-related breathing disorder (SRBD). These questions ask specifically about the child having difficulty breathing in sleep, e.g. "Does your child snore more than half the time?", but also about unrefreshing sleep, e.g. "Does your child wake up feeling unrefreshed in the morning?" and about attention/hyperactivity problems, "Is your child easily distracted by extraneous stimuli?" The presence of SRBD is indicated when 33% of the 22 items are answered affirmatively. Parents responded in a yes/no format to the questions. Items were first grouped according to general sleep complaint: sleep-disordered breathing, restlessness (e.g. "does your child have restless sleep, describe restlessness in bed, brief kicks of one or both legs, and repeated kicks or jerks"), insomnia (e.g. "does your child have difficulty falling asleep, have difficulty with routines at bedtime, wake up >2 times/night, have trouble falling back to sleep, wake up early and have difficulty going back to sleep, do bedtimes vary?"), excessive daytime sleepiness (e.g. " have a problem with sleepiness during the day, complain of feeling sleepy during the day, does your child nap during the day?"), and parasomnias (e.g. "has your child ever walked during sleep, have nightmares, wake up screaming?"). Parents were allowed free space at the end of the PSQ to list if they or other family members had a sleep disorder. Parent sleep characteristics are presented in Table 1.

Procedures

Once the families met study criteria, the participating

Table 2: Overview of Sleep Variables Assessed by Actigraphy, Diary, and Questionnaire.

Mean (SD)									
	Actigraphy			Child Diary			Parent Questionnaire		
Variable	NCOA	COA	df[b] (t)	NCOA	COA	df[b] (t)	NCOA	COA	df[b] (t)
BT[a]	---	---	---	22:13(00:59)	22:26 (01:11)	89 (-.81)	22:03 (00:40)	22:00 (00:48)	88 (.26)
WT[a]	---	---	---	07:48 00:59)	07:56 (1:08)	89 (-.46)	07:56(00:47)	07:53 (00:58)	85 (.23)
Sleep latency	37.5 (25.4)	34.9 20.3)	75 (.46)	15.1 (9.1)	18.0 (11.4)	83 (-1.1)	15.9 (9.4)	19.8 (14.2)	87 (.23)
Wake after sleep onset	40.6 (16.6)	44.2 (15.2)	79 (.13)	2.2 (2.3)	2.0 (2.5)	83 (.42)	.00 (.00)	.11 (.31)	4.3[c] (.04)*
Total sleep time	8.6 (0.7)	7.9 (0.7)	79 (3.2)**	9.4 (0.7)	9.1 (0.7)	88 (2.5)*	9.6 (.95)	9.6 (.92)	84 (.22)
Sleep efficiency	80.5 (8.0)	80.6 (5.0)	72 (-.01)	97% (1.5)	96% (2.1)	84 (1.9)	97% (.02)	97% (.02)	84 (.98)
Nap duration (min)	---	---	---	7.1 (17.9)	28.4 (72.0)	90 (-2.2)*	.53[d] (.74)	.77[d] (.10)	88[d] (-1.0)
Sleep quality	---	---	---	2.6[e] (0.3)	2.8[e] (0.3)	88 (-1.5)	0.5[f] (.90)	0.9[f] (1.1)	88 (-1.5)
Amplitude	58.0 (2.1)	57.3 (6.6)	.42 (.67)	---	---	---	---	---	---

Note: Bedtime (BT); Waketime (WT); [a]=Bedtimes and Waketimes for actigraphy were based on the information provided in the sleep diaries. [b]= Degrees of freedom for independent sample t-tests by COA vs. NCOA. Levene's tests for equality of variance were not significant. [c]= Chi-square likelihood ratio for parental questionnaire, scores are for the item, "Does your child have trouble falling back asleep if awoken?" [d]= for parental questionnaire data, summary scores for items endorsed relating to daytime sleepiness were used. [e]= Sleep quality scores on diaries ranged from 1 (poor), 2 (fair), and 3 (good). [f]=for parental questionnaire, summary scores for endorsed items relating to insomnia were used. *p<.05, **p<.01.

families met with one of the study investigators (DC or IH) at the University of Michigan for a full explanation of the study. The study investigator first explained the purpose of the study to the parent(s) and child. Once the family agreed to participate, they underwent a comprehensive informed consent procedure. The study investigator explained directly to the child how to complete the sleep diary. The role of the parent in this process was to provide assistance to the child per his or her request. The diaries used in this study were written in child friendly language and included cartoon pictures of getting into and out of bed. The actigraph was then shown to the child and the functionality of the watch was explained. Children were asked to remove the actigraph for sporting events and during bathing. The parent was then given the Pediatric Sleep Questionnaire and asked to complete the questionnaire at a convenient time at some point over the next week.

Study compliance

Following the initial meeting with the study investigators, the parent and child were sent home with the study materials, a pre-paid Federal Express® envelope, and a list of Federal Express® drop off locations closest to the family's home. After one week of wearing the actiwatch, the family placed the questionnaires and actiwatch in the envelope and mailed it back to the laboratory investigators. Once received, the researchers evaluated the integrity of the data. In the case of missing diary or questionnaire data or data that appeared suspect, the researcher contacted the family to obtain any missing information.

Data analysis

To describe sleep-wake patterns in children of alcoholics, we conducted independent t-tests of sleep variables collected by the diaries, actigraphy, and parental sleep questionnaire data separated by family history of alcoholism. ANCOVAs were used to examine the effect of family history on sleep variables while controlling for age, gender, and pubertal status. To explore the relationships among sleep variables by groups, we conducted partial correlations and multiple regressions between subjective (diary and questionnaire) measures and actigraphy. To report rates of agreement of between methods by group, we presented Bland-Altman plots.

Bland altman charts

Bland Altman Charts provide an interval in which 95% of the differences between measurements are expected to lie (limits of agreement). The information is displayed as scatter plots with the difference of the two measurements for each sample on the vertical axis and the average of the two measurements on the horizontal axis. Three superimposed lines are also on the scatter plot. The middle line represents the average difference between the measurements. The top and bottom line are the upper and lower limits of plus and minus 1.96* sigma (sigma=the standard deviation of the measurement differences). If the two methods are comparable, then differences should be small and the mean of the differences is close to zero. If it is not close to zero, this indicates that the two methods are producing different results. They should show no systematic variation with the mean of the two measurements. One also looks for a trend, i.e., if the difference between methods tend to get larger (or smaller) as the average increases and if the variability is consistent across the graph. A small difference is an amount that is clinically insignificant for the factor being measured [44]. If the differences within mean +/- 1.96 SD are not clinically important, the two methods may be used interchangeably.

Results

Participant characteristics

A total of 92 children participated (34 females and 58 males). There were 24 children from control families (6 female and 18 males) and 68 from alcoholic families (28 females and 40 males). There were no differences in age between groups. The ethnic distribution of the sample was 48 Caucasian, 14 African American, 1 Native American, 14 Hispanic, and10 biracial. Five families did not report their ethnicity. There was no difference in family income between alcoholic and control families (mean family income for COA families $73, 000 (SD=31, 000) vs. $80, 000 (SD=16, 000) for NCOA, [t=1.0, p=.29].

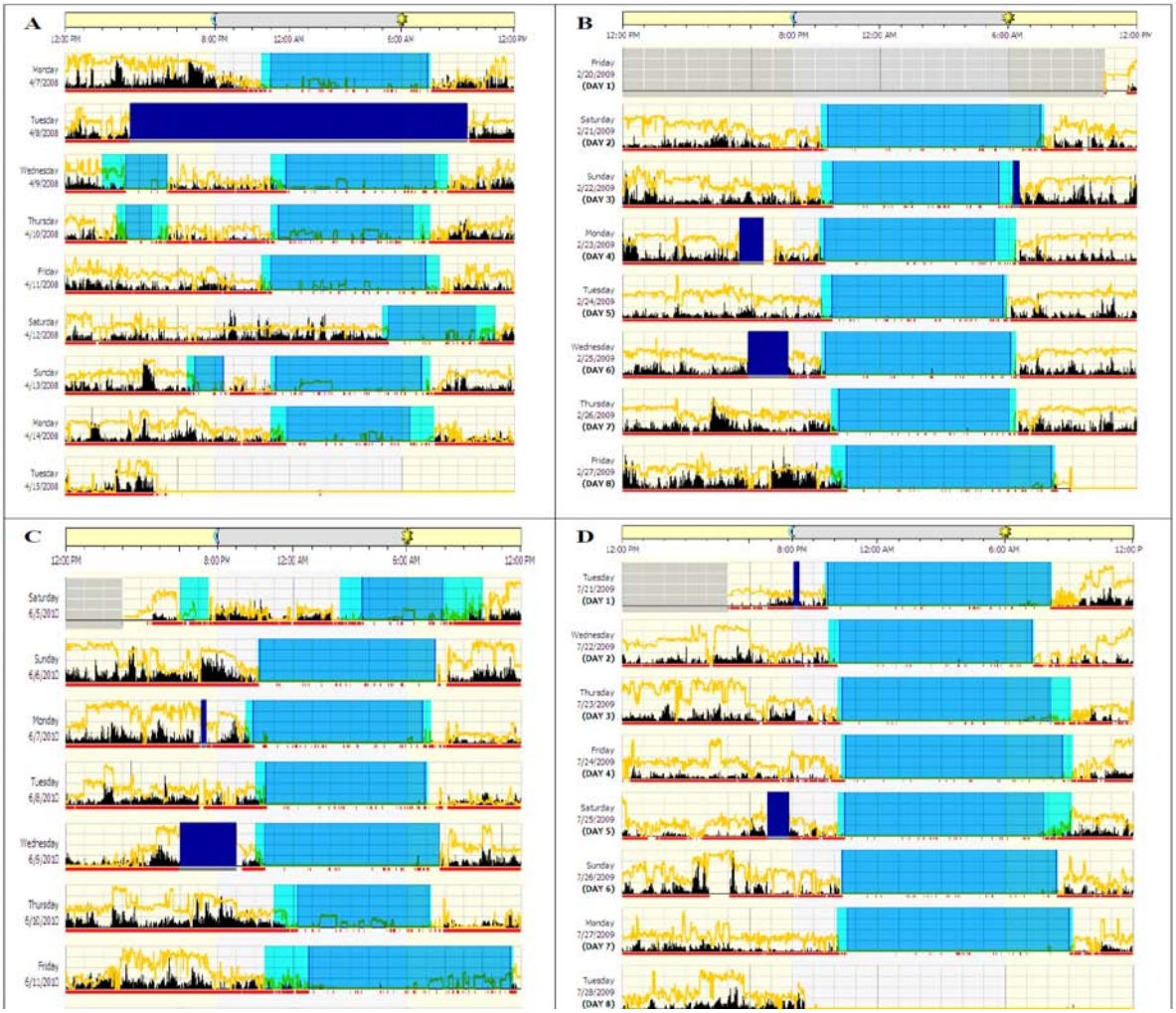

Figure 1: Actigraphy data in a male COA (A), female COA (B), male NCOA (C) and female NCOA (D).
Actigraphy with sleep periods (turquois bars), time watch was removed (navy blue bars), activity (black vertical lines and red bars along bottom of each row), and light levels (vertical yellow lines along middle of row); A) Top left-male COA 9.7 years old, B) Top right-male NCOA 9.6 years old, C) Bottom left – female COA 9.3 years old, D) Bottom right-female NCOA 10.2 years old. Gray bars in the first rows of B, C and D indicate the time before the watch was schedule to start. COAs with napping and most discrepant sleep patterns were chosen to present pictorially.

The average height of the subjects was 4 feet 5.5 inches and weight was 86.6 pounds (SD=24.2), yielding an average BMI of 21.7. The mean Tanner scores, a measure of pubertal development with scores ranging from 1-5, (1=no clinical signs of puberty to 5=completed puberty) was stage 2 and did not differ between groups. These scores were derived by asking the parent to circle the appropriate stage of their child's pubertal development from a series of pictures (breast development and pubic hair for females and development in size of male genitalia and pubic hair in males). Thirteen COA parents identified having a sleep disorder compared to three NCOAs parents (Table 1). COA parents reported that their child was slightly more likely to demonstrate symptoms of insomnia (Cohen's *d*=.28) and daytime sleepiness (*d*=.39) compared to NCOAs. While the children reported more frequent napping in their sleep diaries, parents did not describe their children as typical "nappers." A Chi-square analysis revealed that only one COA parent reported that their child was a "napper", but no NCOA parent endorsed napping in their child. A greater percentage of COA parents reported that their child drank caffeinated beverages (88%) compared to NCOAs (12%). Across the entire sample, parental reports of insomnia and parasomnias were correlated with total time spent napping (r=.50, p=.029 and r=.53, p=.020, respectively). When the group was split according to COA vs. NCOAs, the duration of daytime napping in NCOAs was correlated with parental reports of insomnia only (r=.68, p<.01). The duration of daytime napping in COAs was correlated with parental reports of insomnia (r=.30, p=.01), restlessness during sleep (r=.29, p=.05), and parasomnias (r=.31, p=.01).

Comparison of sleep characteristics by family history

Sleep diaries revealed a significant difference in TST between the COA group and NCOA groups, although perhaps a small clinical difference of 18 minutes. COAs reported napping for longer than NCOAs (see Table 2). Fifteen (22%) COAs took 1-4 naps per week that ranged from 10-375 minutes, while five (21%) NCOAs took 1 nap per week that ranged from 10-75 minutes. Actigraphy was used to provide a secondary measure of sleep as well as information on

Table 3: Partial correlation between sleep variables assessed by actigraphy, diary, and Pediatric Sleep Questionnaire (PSQ) completed by parent.

r (P value)		
	Actigraphy	Diary
Bedtime	NA	.67 (.0005)
Waketime	NA	.57 (.0005)
Sleep latency	.12 (.30)	.68 (.0005)
Wake after sleep onset[a]	.00 (.99)	.28 (.012)
Total sleep time	.32 (.005)	.32 (.003)
Napping[b]	----	.20 (.060)[c]

Note: [a]=for parental questionnaire data, scores for the item "does your child have trouble falling back asleep if awoken?" was used. [b]= PSQ summary score of items relating to daytime sleepiness were used to correlate total nap time reported by the child in the diary. [c]= when *not* controlling for family history, this relationship becomes significant (r=.21, p=.04).

motor activity, light exposure, and rest/activity patterns. Figure 1 displays individual graphs from actigraphy from four participants matched by age and sex. COAs with the most extreme pattern deviations and presence of naps were selected. Naps are shown by the shorter turquoise bars in the two COAs. Analyses revealed higher average nocturnal motor activity in COAs, 53.7 (23.9) counts per minute versus 39.8 (21.9) in NCOAs, t=-2.3, (p=.03). No differences were found in light exposure between groups.

To explore the effects of family history on sleep variables while controlling for age, gender, and pubertal status, we ran ANCOVAs. After controlling for these variables, sleep diary sleep quality was significantly different between COAs and NCOAs [F (1, 50) =5.7, p=.02, eta squared =.10], with COAs reporting better sleep quality. For the sleep diary, there was a significant effect of age on bedtime [F (1, 51) =6.5, p=.014, eta squared =.11)], such that the older children went to bed later and had more wake after sleep onset [F (1, 47) =9.7, p=.003, eta squared =.17]. For actigraphy, there was an effect of age on activity during sleep [F (1, 44) =4.7, p=.036, eta squared =.10) and a significant effect of both age [F (1, 44) =4.5, p=.041, eta squared=.10] and Tanner stage [F (1, 44) =4.1, p=.049, eta squared=.10] on wake time after sleep onset. For the parent rated PSQ, there was a significant effect of Tanner stage on PSQ calculated sleep efficiency [F (1, 48) =4.6, p=.038, eta squared =.09] and an effect of age [F (1, 57) =9.2, p=.004, eta squared=.15] and Tanner stage [F (1, 57) =10.5, p=.002, eta squared=.17] on PSQ rated daytime sleepiness.

Interrelations between and within methods by group

Table 3 displays the partial relationship between parental report of sleep (via the PSQ), actigraphy, and sleep diaries while controlling for family history. Preliminary analyses were performed to ensure no violation of the assumptions of normality, linearity, and homoscedasticity. Significant relationships were found between parent reports and 5 out of 6 of the corresponding sleep diary measures and 1 out of 3 of the corresponding actigraphy sleep measures. The one sleep diary measure that did not show a relationship to parental report was total daytime napping. An inspection of the zero order correlation [r=.21, n=88, p=.04] suggested that family history did have an effect on the strength of the relationship between napping reported by the child and daytime sleepiness reported by the parent. Actigraphically defined sleep latency and wake time after sleep onset were not consistent with parental reports of difficulty staying asleep.

Multiple regressions of diary-reported sleep quality and other measures of sleep continuity revealed that sleep quality was predicted by the number of awakenings during the night, (beta=-.33; p=.01) in both groups, but with a slightly stronger relationship in NCOAs (beta=-.46, p=.03). Sleep quality was predicted by wake time after sleep onset in COAs (df= 1, 62, beta=-.29, p=.02), but not in NCOAs, (df=1, 20, beta=-.30, p=.19).

Agreement between measurement methods by FH status

Bland Altman plots are displayed for three variables: Bedtime, Waketime and Wake after sleep onset so that the reader can visually inspect for the subtle differences between groups. Figure 2 shows that child- and parent-reported bedtime (graphs A and B) and waketime (graphs C and D) are generally comparable because the mean differences are relatively small, are close to zero, and there is no systematic variation with the mean of the two measurements. However, there is a slight trend in the COA data with parent and child reports of bedtime becoming more discrepant the later bedtime becomes. Indeed, parent-rated bedtimes were significantly different from child-rated bedtimes in COAs, but not NCOAs. There were no differences between parents- and -child-rated waketimes.

Mean differences between sleep diaries compared to actigraphy ranged from 15.5 minutes and 63.5 minutes. The Bland Altman plot of wake time after sleep onset in Figure 2 shows that the mean differences are not close to zero, there is a clear trend in the data, as the difference between methods gets larger, the average increases, and the variability is consistent across the graph. Therefore, the two methods are producing different results.

Discussion

The purpose of the present study was to describe sleep-wake patterns in the children of alcoholics (COAs), to explore relationships between sleep variables between COAs to children of control families (NCOAs), and to explore the relationships among sleep variables by groups.

These preliminary results suggest that COAs napped more during the day than NCOAs, had statistically significantly less nocturnal sleep time, and had more motor activity during sleep. Children's sleep diary reports appear to correlate well with parental reports, except for bedtime in some COAs, where in some cases, parents rated bedtime earlier than their children. There were also discrepancies between sleep diaries and actigraphy-derived sleep measurements in both groups. Compared to sleep diaries, actigraphy overestimated the amount of nocturnal awakening by approximately 30 minutes.

COAs reported more napping compared to NCOAs. Additional assessments of where and under what circumstances the naps were taking place e.g. scheduled versus spontaneous may have provided more insight into this. Most children (34.5%) stop taking natural/unscheduled napping by age 4 [23]. According to a Sleep in America Poll, out of a sample of 635 school-aged children in the US, only 2% slept 1-2 hours in the daytime [45]. Environmental or family factors may be at play, e.g. the COA's attempt to seek refuge in bed from a disturbing home environment or fewer after school activities. Interestingly, parents were not aware of the frequency of their child's daytime naps. Given the common genetic risk for alcoholism and mood disorders in first degree relatives of alcoholics [17], this

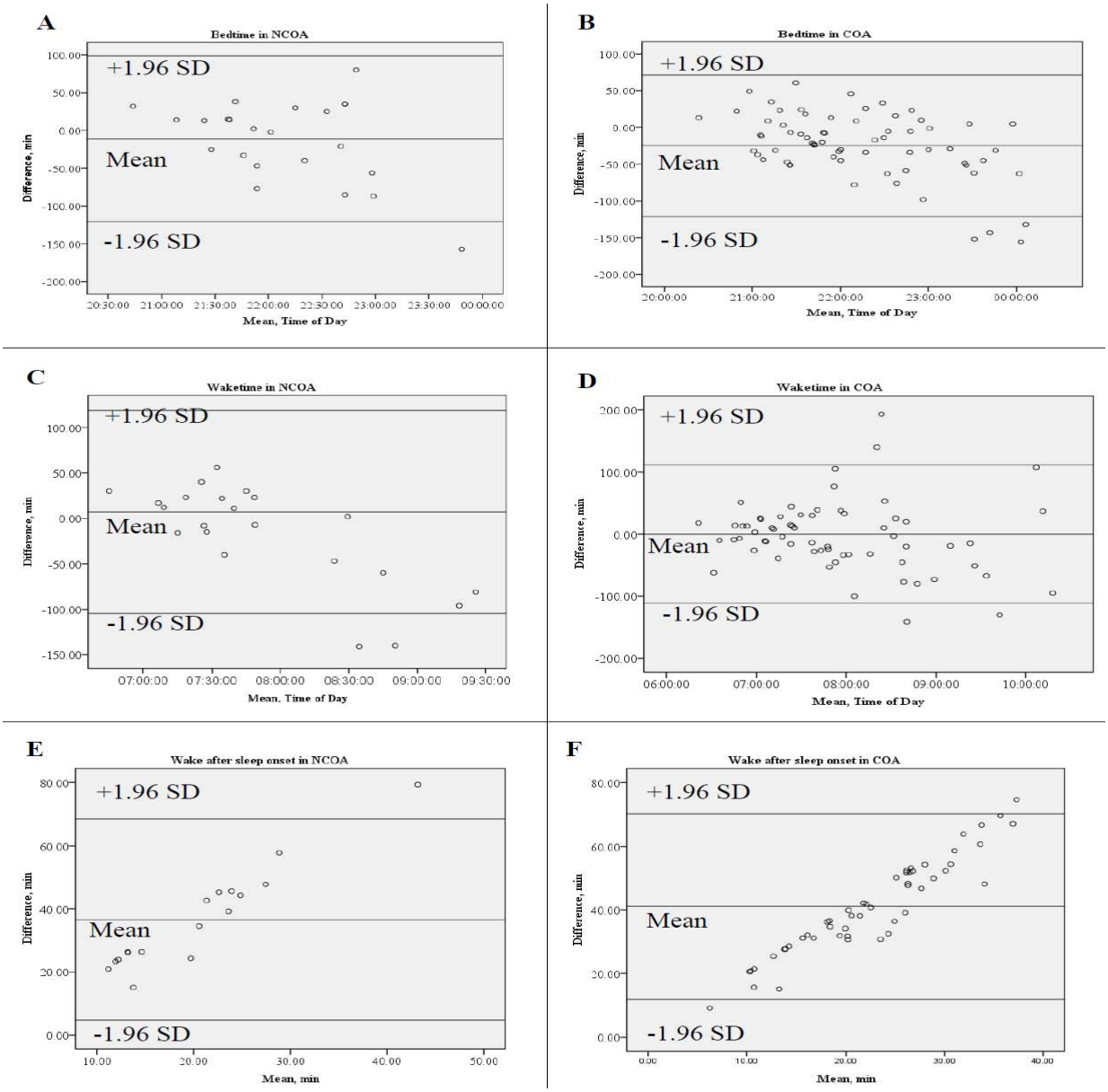

Figure 2: Bland Altman Plots for Bedtime, Waketime, and Waketime after sleep onset by group.
Bland Altman plots of the differences between parental report of bedtime plotted against bedtime reported by the child in the sleep diary in NCOAs (A) and COAs (B); differences between parental report of waketime plotted against waketime reported by the child in the sleep diary in NCOAs (C) and COAs (D); differences of Waketime after sleep recorded in the sleep diary plotted against wake after sleep onset measured by actigraphy in NCOAs (E) and COAs (F).

may reflect, at least in part, the early signs of mood disorders, (e.g. hypersomnolence [46]). Other studies have shown that COAs between the ages of 6-17 are twice as likely as controls to present with subclinical symptoms of psychiatric problems and four times more likely to have a diagnosis of any psychiatric disorder [47].

A higher level of motor activity was captured by actigraphy during sleep in the COAs compared to NCOAs. This may have been due to actigraphy detecting subtle motor movements in sleep that is not perceived as wakefulness. In a comparison study between actigraphy and polysomnography, it was found that time awake after sleep onset was overestimated with actigraphy by 26.3 +/-34.4 minutes [23]. Moreover, our findings suggest that in this sample, actigraphy and sleep diaries may be producing different results. Nevertheless, should the COA group have subtle sleep disturbance, the sleep disturbances could be associated with future problems.

Interrelationships between methods by group

We hypothesized that there would be a significant difference between children's estimation of sleep from their parents estimations as well as from an objective measure (actigraphy). In general, the study findings did not support this hypothesis, but rather that child and parent reports are similar. However, there was a high degree of inter-individual variability. A similar study in children (ages 4-7) using Bland Altman plots [24] to describe discrepancies between actigraphy and sleep diary used a range of <30 minutes between

measures to be considered a useful measure. The authors found that actigraphically determined sleep start, sleep end, and "assumed sleep" were comparable to parent reports which were made every 15 minutes across 6-8 nights of actigraphy. Our study differed from Werner et al not only in terms of the age of our participants, but because parent participants made a one-time assessment of typical bedtime and waketime and this was compared to the mean of a 7-day sleep diary. In addition, we relied on child-reported bedtimes in the sleep diary and not actigraphically defined sleep start and end times. Future studies should examine sleep start and end times as derived by actigraphy alone in this population.

Study limitations

There were a number of limitations to the study. Therefore, the results should be interpreted with caution. First, the sample size was quite small. Second, anxiety and depressive symptoms were not included in this study and therefore we cannot statistically test whether depressive mood symptoms acted as a mediator to explain differences in sleep observed between COAs and NCOAs. Third, we did not determine if the parent that helped guide the child's sleep diary was an alcoholic or not. Fourth, this study utilized child's own reports of sleep in the sleep diaries instead of parental reports, which may have accounted for the high degree of variability in our results. Fifth, we did not have an equal distribution of males to females in both groups to examine our measures by sex. This is important given the sex difference in vulnerability to develop alcoholism. Sixth, we had a smaller control group than study group, which may have led to the absence of associations between parental reports of sleep and daytime napping. Last, the study did not include sleep electroencephalography (EEG), so possible differences in sleep architecture could not be studied. Future studies may further explore sleep EEG characteristics, including homeostatic regulation of sleep, temporal coherence between EEG and actigraphy, and biological circadian markers (e.g. dim light melatonin onset). Studies should also compare raw actigraphy to actigraphy combined with bedtimes and waketimes from sleep logs in this population. These finite analyses in combination with assessments of the child's mood or early traumatic events may provide a better understanding of the neurophysiological predisposition to a healthy or problem outcome later in life.

Conclusions

In summary, children with a high risk status based on their family history of alcoholism slept more during the day and slightly less at night compared to control children. Self-report of sleep was correlated strongly with parental reports, except for bedtime reports in some COAs. Diary and actigraphy assessments of sleep did not agree well and therefore these were not interchangeable methods of assessing sleep in this sample. A focus on achieving sufficient sleep in COAs at this age group may be important, since sleep hours are likely to be further curtailed when these children approach adolescence due to biological (puberty, circadian rhythm changes) and environmental (earlier school start times) factors. Behavioral sleep interventions may also worthwhile to consider, given their potential to also affect clinical course.

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References

1. Baglioni C, Battagliese G, Feige B, Spiegelhalder K, Nissen C, Voderholzer U, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord. 2011; 135: 10-19.
2. Breslau N. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biological Psychiatry. 1996; 39: 411-418.
3. Gregory AM, Buysse DJ, Willis TA, Rijsdijk FV, Maughan B, Rowe R, et al. Associations between sleep quality and anxiety and depression symptoms in a sample of young adult twins and siblings. J Psychosom Res. 2011; 71: 250-255.
4. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? JAMA. 1989; 262: 1479-1484.
5. Goldstein TR, Bridge JA, Brent DA. Sleep disturbance preceding completed suicide in adolescents. J Consult Clin Psychol. 2008; 76: 84-91.
6. Koyawala N. Sleep problems and suicide attempts among adolescents: A case-control study. Behavioral Sleep Medicine. 2014; 12: 1-11.
7. Wong MM, Brower KJ, Zucker RA. Sleep problems, suicidal ideation, and self-harm behaviors in adolescence. J Psychiatr Res. 2011; 45: 505-511.
8. Hasler BP, Martin CS, Wood DS, Rosario B, Clark DB. A longitudinal study of insomnia and other sleep complaints in adolescents with and without alcohol use disorders. Alcohol Clin Exp Res. 2014; 38: 2225-2233.
9. Wong MM, Brower KJ, Zucker RA. Childhood sleep problems, early onset of substance use and behavioral problems in adolescence. Sleep Med. 2009; 10: 787-796.
10. Wong MM, Brower KJ, Fitzgerald HE, Zucker RA. Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. Alcohol Clin Exp Res. 2004; 28: 578-587.
11. Gulewitsch M. Brief hypnotherapeutic-behavioral intervention for functional abdominal pain and irritable bowel syndrome in childhood: a randomized controlled trial. Eur J Pediatr. 2013; 172: 1043-1051.
12. Buckhalt JA, El-Sheikh M, Keller PS, Kelly RJ. Concurrent and longitudinal relations between children's sleep and cognitive functioning: the moderating role of parent education. Child Dev. 2009; 80: 875-892.
13. El-Sheikh M, Kelly R. Sleep in children: Links with marital conflict and child development. In: Sleep and development: Familial and socio-cultural considerations. El-Sheikh M, editor. Oxford University Press: New York. 2011; 3-28.
14. Tarokh L, Carskadon MA. Sleep electroencephalogram in children with a parental history of alcohol abuse/dependence. J Sleep Res. 2010; 19: 165-174.
15. Tarokh L, Van Reen E, Acebo C, LeBourgeois M, Seifer R, Fallone G, et al. Adolescence and parental history of alcoholism: insights from the sleep EEG. Alcohol Clin Exp Res. 2012; 36: 1530-1541.
16. Köhnke MD. Approach to the genetics of alcoholism: a review based on pathophysiology. Biochem Pharmacol. 2008; 75: 160-177.
17. Edenberg HJ, Foroud T. The genetics of alcoholism: identifying specific genes through family studies. Addict Biol. 2006; 11: 386-396.
18. Hasler B, Clark D. Circadian misalignment, reward-related brain function, and adolescent alcohol involvement. Alcohol Clin Exp Res. 2013; 37: 558-565.
19. Dayyat EA, Spruyt K, Molfese DL, Gozal D. Sleep estimates in children: parental versus actigraphic assessments. Nat Sci Sleep. 2011; 3: 115-123.

20. Spruyt K, Gozal D, Dayyat E, Roman A, Molfese DL. Sleep assessments in healthy school-aged children using actigraphy: concordance with polysomnography. J Sleep Res. 2011; 20: 223-232.

21. Paavonen EJ, Fjällberg M, Steenari MR, Aronen ET. Actigraph placement and sleep estimation in children. Sleep. 2002; 25: 235-237.

22. Sadeh A. Evaluating night wakings in sleep-disturbed infants: a methodological study of parental reports and actigraphy. Sleep. 1996; 19: 757-762.

23. Tikotzky L, Sadeh A. Sleep patterns and sleep disruptions in kindergarten children. J Clin Child Psychol. 2001; 30: 581-591.

24. Werner H, Molinari L, Guyer C, Jenni OG. Agreement rates between actigraphy, diary, and questionnaire for children's sleep patterns. Arch Pediatr Adolesc Med. 2008; 162: 350-358.

25. Gregory A. Sleep items in the Child Behavior Checklist: A comparison with sleep diaries, actigraphy and polysomnography in a sample of anxious, depressed, and control youth. J Am Acad Child Adoles Psychiatry. 2011; 50: 499-450.

26. Sadeh A, Sharkey KM, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. Sleep. 1994; 17: 201-207.

27. Biggs SN, Lushington K, van den Heuvel CJ, Martin AJ, Kennedy JD. Inconsistent sleep schedules and daytime behavioral difficulties in school-aged children. Sleep Med. 2011; 12: 780-786.

28. Gregory AM, Caspi A, Eley TC, Moffitt TE, Oconnor TG, Poulton R, et al. Prospective longitudinal associations between persistent sleep problems in childhood and anxiety and depression disorders in adulthood. J Abnorm Child Psychol. 2005; 33: 157-163.

29. Gregory AM, O'Connor TG. Sleep problems in childhood: a longitudinal study of developmental change and association with behavioral problems. J Am Acad Child Adolesc Psychiatry. 2002; 41: 964-971.

30. Johnson EO, Chilcoat HD, Breslau N. Trouble sleeping and anxiety/depression in childhood. Psychiatry Res. 2000; 94: 93-102.

31. Lofthouse N, Fristad M, Splaingard M, Kelleher K. Parent and child reports of sleep problems associated with early-onset bipolar spectrum disorders. J Fam Psychol. 2007; 21: 114-123.

32. Dahl RE, El-Sheikh M. Considering sleep in a family context: introduction to the special issue. J Fam Psychol. 2007; 21: 1-3.

33. El-Sheikh M, Buckhalt JA, Keller PS, Cummings EM, Acebo C. Child emotional insecurity and academic achievement: the role of sleep disruptions. J Fam Psychol. 2007; 21: 29-38.

34. Keller PS, El-Sheikh M, Buckhalt JA. Children's attachment to parents and their academic functioning: sleep disruptions as moderators of effects. J Dev Behav Pediatr. 2008; 29: 441-449.

35. Kelly RJ, El-Sheikh M. Marital conflict and children's sleep: reciprocal relations and socioeconomic effects. J Fam Psychol. 2011; 25: 412-422.

36. Rauer AJ, Kelly RJ, Buckhalt JA, El-Sheikh M. Sleeping with one eye open: marital abuse as an antecedent of poor sleep. J Fam Psychol. 2010; 24: 667-677.

37. Zucker R. Other evidence for at least two alcoholisms II: Life course variation in antisociality and heterogeneity of alcoholic outcome. Development and Psychopathology. 1996; 8: 831-848.

38. Zucker R. The clinical and social ecology of childhood for children of alcoholics: Description of a study and implications for a differentiated social policy. Children of addiction: Research, health and policy issues. Fitzgerald H, Lester B, Zuckerman B, editors. New York: Routledge Falmer. 2000; 109-141.

39. Loukas A, Fitzgerald HE, Zucker RA, von Eye A. Parental alcoholism and co-occurring antisocial behavior: prospective relationships to externalizing behavior problems in their young sons. J Abnorm Child Psychol. 2001; 29: 91-106.

40. Sokol RJ, Clarren SK. Guidelines for use of terminology describing the impact of prenatal alcohol on the offspring. Alcohol Clin Exp Res. 1989; 13: 597-598.

41. Buu A, Mansour M, Wang J, Refior SK, Fitzgerald HE, Zucker RA, et al. Alcoholism effects on social migration and neighborhood effects on alcoholism over the course of 12 years. Alcohol Clin Exp Res. 2007; 31: 1545-1551.

42. Johnston L. Monitoring the Future national survey results on drug use, 1975-2003. National Institute on Drug Abuse. Secondary School Students. 2004; 1.

43. Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. Sleep Med. 2000; 1: 21-32.

44. Bland J, Altman D. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet. 1986; 1: 307-310.

45. National Sleep Foundation, Children and sleep. 2004.

46. Hawkins DR, Taub JM, Van de Castle RL. Extended sleep (hypersomnia) in young depressed patients. Am J Psychiatry. 1985; 142: 905-910.

47. Díaz R, Gual A, García M, Arnau J, Pascual F, Cañuelo B, et al. Children of alcoholics in Spain: from risk to pathology. Results from the ALFIL program. Soc Psychiatry Psychiatr Epidemiol. 2008; 43: 1-10.

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