A R T I C L E

SLEEP PATTERNS OF 7-WEEK-OLD INFANTS AT FAMILIAL RISK FOR ATTENTION DEFICIT HYPERACTIVITY DISORDER

RIVKA LANDAU
Ben Gurion University

AVI SADEH
Tel Aviv University

PAZ VASSOLY, ANDREA BERGER, NAAMA ATZABA-PORIA, AND JUDITH G. AUERBACH
Ben Gurion University

ABSTRACT: Sleep patterns of 26 seven-week-old boys at familial risk for attention deficit hyperactivity disorder (ADHD) and 18 control infants were compared by objective (actigraph) and subjective (maternal sleep diary) measures, over five consecutive 24-hr periods. Actigraph findings indicated that the groups differed on stability (*SD*) of quiet sleep only during the day. Reports in maternal sleep diaries indicated that they also differed on stability of waking and stability of sleep duration, again only during the day. No group differences were found in terms of average scores, whether calculated for the entire 24-hr periods, for nights, or for days. Mothers in the risk group reported that fathers were less involved in infant care than did those in the control group. These findings suggest that as early as 7 weeks of age, infants at risk for ADHD differ from controls only on stability of their sleep patterns during the day, when environmental regulatory factors are more intensive.

RESUMEN: Los patrones de dormir de 26 niños de siete semanas de edad y con riesgos familiares del Trastorno de Hiperactividad y Déficit de Atención (ADHD) y de 18 infantes en un grupo de control fueron comparados por medio de medidas objetivas (el actígrafo) y subjetivas (el diario sobre patrones de dormir escrito por la madre), dentro de cinco períodos consecutivos de 24 horas. Los resultados del actígrafo indicaron que los grupos difirieron en cuanto a la estabilidad (desviación estándar) del dormir tranquilo solamente durante el día. Los reportes de los diarios maternales indicaron que ellos también diferían en cuanto a la estabilidad del despertar y la estabilidad de la duración del sueño, otra vez solamente durante el día. No se encontraron diferencias entre los grupos en términos de los puntajes promedio, ya fueran estos calculados para los períodos de 24 horas completos, para las noches, o para los días. Las madres dentro del grupo de bajo riesgo reportaron que los papás estuvieron menos involucrados en el cuidado del infante de lo que mostraron los papás dentro del grupo de control. Estos resultados sugieren que, tan pronto como siete semanas de edad, los infantes bajo riesgo de ADHD difieren de los grupos de control solamente en cuanto

This study was supported by the Israel Science Foundation, Grants 756/98–01 and 869–01, and by the Yael-Ana Ben-Aaron grant.

Direct correspondence to: Rivka Landau, Department of Psychology, Ben Gurion University of the Negev, Beer Sheva, Israel; e-mail: Landau@bgu.ac.il.

INFANT MENTAL HEALTH JOURNAL, Vol. 31(6), 630–646 (2010)

View this article online at wileyonlinelibrary.com.
DOI: 10.1002/imhj.20275

a la estabilidad de sus patrones de dormir durante el día, cuando los factores ambientales de regulación son más intensivos.

RÉSUMÉ: Les patterns du sommeil de 26 garçons âgés de sept semaines à risque familial pour le TDA/H ou trouble du déficit de l'attention/hyperactivité ont été comparés grâce à des mesures objectives (actigraphe) et subjectives (journal de sommeil maternel), sur cinq périodes consécutives de 24 heures. Les résultats actigraphes ont indiqué que les groupes différaient pour ce qui concerne la stabilité (écart type) du sommeil tranquille uniquement durant la journée. Les rapports dans les journaux maternels de sommeil ont indiqué que les groupes différaient également pour ce qui concerne la stabilité du réveil et la stabilité de la durée du sommeil, à nouveau uniquement durant la journée. Aucune différence de groupe n'a été trouvée pour ce qui concerne les scores moyens, que ce soit calculés pour toutes les périodes de 24 heures, pour les nuits ou pour les journées. Les mères du groupe à risque ont indiqué que les pères étaient moins impliqués dans le soin du bébé que ne l'ont fait celle du groupe de contrôle. Ces résultats suggèrent que, aussi tôt qu'à l'âge de sept semaines, les bébés à risque pour le TDA/H diffèrent des contrôles uniquement pour ce qui concerne la stabilité de leurs patterns de sommeil durant la journée, lorsque les facteurs regulateurs environnementaux sont plus intenses.

ZUSAMMENFASSUNG: Die Schlafmuster von 26 sieben Wochen alten Jungen mit erblich bedingtem erhörtem Risiko für ADHS und 18 Kinder einer Kontrollgruppe wurden durch objektive (Actigraph Vergleich) und subjektive (mütterliches Schlaftagebuch) Messinstrumente, über fünf aufeinander folgende 24-Stunden-Zeiträumen ermittelt. Die Actigraph Ergebnisse zeigen nur Gruppenunterschiede bezüglich der Stabilität (Standardabweichung) während des Tagschlafs. Die Ergebnisse der Schlaftagebücher der Mutter deuten zusätzlich auf Stabilitätsunterschiede der Aufwachhäufigkeit und der Dauer des Schlafes, jedoch wieder nur im Tagschlaf. Es gab keine Unterschiede zwischen den Gruppen bezüglich der Durchschnittswerte, sowohl berechnet für die gesamte 24-Stunden-Zeiträume, als auch für die Nächte oder Nächte. Mütter in der Risiko-Gruppe berichtet, dass die Väter weniger an der Säuglingspflege beteiligt als in der Kontrollgruppe. Die Ergebnisse zeigen, dass sieben Wochen alte Säuglinge mit erhörtem Risiko für ADHS im Vergleich mit einer Kontrollgruppe nur Unterschiede der Schlafstabilität im Tagschlaf vorliegen, wenn die durch Umweltbedingungen bedingten Faktoren intensiviert werden.

抄録：ADHDの家族的なリスクのある26人の生後７週の男児と、18人の対照児の睡眠パターンが、5日間連続した24時間単位で、客観的(アクティグラフactigraph)および主観的（母親の睡眠日記）測定法により比較された。アクティグラフの所見は、日中についてだけ静かな睡眠の安定性（標準偏差）に差があることを示した。母親の睡眠日記の報告でも、ここでも日中についてだけ、覚醒の安定性と睡眠持続の安定性に差があったことを示した。平均値に関しては、24時間全体を計算しても、夜間あるいは日中について計算しても、群間に差は認められなかった。リスク群の母親は、対照群の母親よりも、父親が乳児の世話に関わることが少ないと報告した。これらの所見から、早くも生後７週から、環境の調節因子がより強力であれば、ADHDのリスクのある乳児は、日中の睡眠パターンの安定性についてだけ、対照と違いがあることが示唆される。

* * *

Attention deficit hyperactivity disorder (ADHD) is characterized by impaired cognitive functioning, hyperactivity, and impulsivity, which are associated with problems in behavioral and emotional regulation according to the *Diagnostic and Statistical Manual of Mental Disorders, fourth edition* (*DSM-IV*; American Psychiatric Association, 1994). The theory is that a genetic neurodevelopmental vulnerability underlies this disorder, especially when it persists into adolescence and adulthood (Barkley, 1998; Biederman et al., 1995; Faraone, Biederman,

& Monuteaux, 2000; Price et al., 2005; Smalley et al., 2000; Taylor, 1999). Impaired cognitive functioning and modulation of impulses also were found to characterize sleep restriction or deprivation (Dahl, 1996). In addition, Thoman, Denenberg, Sievel, Zeidner, and Becker (1981) found inconsistent sleep–wake patterns in early infancy in the general population to be associated with medical and behavioral problems later in life.

In light of the similarity in symptoms found in children with ADHD and healthy children when deprived of proper sleep, as well as the physiological component that underlies the development of sleep–wake organization into stable cycles, it seems reasonable to assume a link between the sleep disruption prevalent in children with ADHD and the genetic neurodevelopment impairment of the ADHD disorder. However, this connection has been challenged lately. A few studies of preschool and school-age children with ADHD suggest that sleep systems and ADHD do not share the same physiological structures (Gruber & Sadeh, 2004; Mick, Biederman, Jetton, & Faraone, 2000).

Research on the association between ADHD and sleep problems has applied both subjective and objective methods and has yielded inconsistent findings (Corkum, Tannock, & Molodofsky, 1998; Dahl, 1996; Gruber & Sadeh, 2004; Gruber, Sadeh, & Raviv, 2000; Sadeh, Pergamin, & Bar-Haim, 2006). A survey by Corkum et al. (1998) found that when subjective measures were used by parents and clinicians, the sleep of children with ADHD differed from the sleep of regular children in the following ways: The children with ADHD had more problems going to bed, more night wakings, and more short naps during the day. However, in most studies where objective measures were used, no differences in sleep measures were found between the groups of children with ADHD and comparison groups, with the exception of activity level during sleep. One of the studies that used an actigraph (not included in the Corkum et al., 1998, survey) found that sleep efficiency and quiet sleep were lower in children with ADHD than that in the comparison group (Dagan et al., 1997). In addition, Sadeh et al. (2006), in a recent meta-analysis of available polysomnographic studies of children with ADHD, found that the only significant difference between them and controls was the prevalence of periodic limb movements in sleep; however, most children with ADHD do not have significant periodic limb movements in sleep.

Most studies on sleep patterns over a few nights use averages to calculate sleep measures. According to Halperin, Maclean, and Baumeister (1995) and Parmelee and Stern (1972), organization of the sleep patterns into *consistent* sleep–wake cycles better reflects the proper neurodevelopment of sleep. Thus, night-to-night consistency of each measure seems to be an important indicator of proper development. In studies by Gruber and Sadeh (2004) and Gruber et al. (2000), sleep was recorded for 5 nights using an actigraph. When means across all nights were calculated, no differences were found between children with ADHD and a control group. However, when night-to-night intraindividual consistency (i.e., the variance between nights) was examined, a higher variability was found in the group of children with ADHD for sleep onset, sleep duration, and true sleep (i.e., sleep without waking).

These studies were all of school-age children. Yet, patterns of sleep are established during the first years of life. In addition to the physiological factors underlying their development, children are strongly influenced by environmental regulation of sleep (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 1991; Anders, Halpern, & Hau, 1992; Beltramini & Hertzing, 1983; Johnson, 1991; Keener, Zeanah, & Anders, 1988; Mindell, Telofski, Wiegand, & Kurtz, 2009; Sadeh & Anders, 1993; Sadeh, Mindell, Luedtke, & Wiegant, 2009; Scher, 2001; Scher et al., 1995). Moreover, after these patterns are established, they tend to persist and may develop into chronic disorder (Kataria, Swanson, & Trevathan, 1987; Richman, Stevenson, & Graham, 1982;

Zuckerman, Stevenson, & Baily, 1987). As indicated by Gruber et al. (2000), the inconsistency in the sleep–wake system found in studies with school-age children may represent a regulatory disorder resulting from inadequate parenting with regard to sleep procedures during the first years of life. Alternatively, it could be attributed to a genetic neurodevelopmental vulnerability of the sleep system, which may be linked to the neurodevelopmental vulnerability associated with ADHD.

The studies cited earlier did not assess parental ADHD. Yet, ADHD is a developmental disorder that often persists into adolescence and adulthood (Barkley, 1990; Campbell, 2002; Hechtman, 2000; Price et al., 2005). Not only does having a parent with ADHD increase a genetic neurodevelopmental risk for disrupted sleep patterns but the ADHD symptoms of inattention, impulsivity, and overactivity (American Psychiatric Association, 1994) may result in less optimal parental monitoring of the sleep schedule from infancy. Such nonoptimal monitoring of sleep in the first years of life may increase the expression of a genetic liability linked to ADHD. This may even cause night-to-night inconsistency in the sleep measures of children with ADHD with no family history of ADHD.

In recent years, more attention has been directed to exploring differences in parenting behavior between adults with and without ADHD. Murray and Johnston (2006) found that mothers with ADHD were poorer at monitoring child behavior and less consistent disciplinarians than were those without the disorder. The former also scored lower on the effectiveness or quality of solutions generated for problems and the level of planning involved in the solutions. This direction of findings persisted when controlling for conduct behavior and oppositional disorder in the children and for comorbidity in the mothers. In a similar vein, Harvey, Danforth, McKee, Ulaszek, and Friedman (2003) found that fathers' and mothers' self-report of inattention and impulsiveness was associated with their self-report of lax parenting.

Several studies have found that ADHD in one parent changed the regulation of the whole family system and impaired family and marital functions regardless of the gender of the affected parent (Biederman, Faraone, & Monuteaux, 2002; Minde et al., 2003; Weiss, Hechtman, & Weiss, 2000). According to Weiss, Hechtman, and Weiss (2000), a parent with ADHD may have difficulties with the instrumental and organizational tasks of parenting, and when such a parent becomes anxious, it can impact the family as a whole. A new infant calls for reorganization of the family system and the behavior of each of the participants. Having a disorganized, impulsive, nonattentive, and hyperactive spouse at this time can disrupt this reorganization and impair the parenting behavior of the partner who does not have such ADHD symptoms.

As indicated, thus far, research on sleep patterns related to ADHD has been limited to preschool and school-age children with ADHD. One way to further understand whether the inconsistency in patterns of sleep found in older children with ADHD is due to a neurodevelopmental impairment or mainly an environmental regulatory outcome is to study the pattern of sleep in infants at risk for ADHD as early as possible and to longitudinally follow them. The aim of the present study, which is part of a prospective longitudinal study of infants at familial risk for ADHD (Auerbach, Atzaba-Poria, Berger, & Landau, 2004; Auerbach et al., 2005; Landau, Amiel-Laviad, Berger, Atzaba-Poria, Auerbach, 2009; Landau et al., 2010), was to assess objectively and subjectively the sleep patterns of 7-week-old boys for five continuous 24-hr periods in two groups: a group at familial risk for ADHD (due to father's ADHD symptoms) and a control group.

Two additional studies from the same longitudinal research (Landau et al., 2009, 2010) (whose samples partially overlap that of the present study) analyzed the interaction between the

infants and each of their parents at 7 months. They revealed that mothers in the risk group (due to paternal ADHD symptoms) reacted less adequately than did the control group to the needs of their infants during two kinds of free play, although the needs of the infants did not differ (Landau et al., 2009, 2010). In Landau et al. (2010), both parents in the risk group reacted less adequately than did the control group to all infants needs, especially to negative emotions.

It has been found that newborns sleep twice as much during the night as they do during daytime hours (Sadeh, Dark, & Vohr, 1996). This difference increases with age. Moreover, parents sleep at least part of the night, and most reduce interaction with their infants when they waken in those hours. Other environmental stimulation also decreases at night. Thus, it is reasonable to expect environmental factors to influence the sleep patterns of 7-week-old infants more during the day than they would during the night.

We based our research hypothesis on the findings of factors related to the development of night-to-night stability of sleep in young children, the differences in sleep duration and environmental stimulation between day and night, and the less adequate parenting found in families in which one of the spouses has ADHD. Specifically, we expected the day-to-day sleep patterns of infants in the familial risk group for ADHD to be less stable during daytime hours than the control group on both objective and subjective measures whereas no group differences in sleep patterns would be found during the full 24-hr periods and the nights.

METHOD

Participants

In the current report, data are presented for 44 six- to seven-week-old boys (M age = 45 days, SD = .15). Recruitment to the longitudinal study was conducted in the maternity ward of a medical center. The parents of all infants were native-born or immigrants who had studied in the country and spoke the language. In the families who agreed to participate, fathers were asked to complete a questionnaire assessing current ADHD symptoms. The questionnaire included 18 items referring to such symptoms, taken from the *DSM-IV* (American Psychiatric Association, 1994) and worded for use with adults (Cronbach α = .80). An additional 19 items from the Tridimensional Personality Questionnaire (Cloninger, 1987), with a similar format, were distributed among the ADHD items so that the fathers would not have to complete a questionnaire consisting only of items indexing psychopathology.

The present prospective study is not clinical-based research; rather, it consists of families recruited from the general population. Entry into the study was based on the number of positive responses (yes-no format) made by fathers to the ADHD items. The criterion for recruitment into the ADHD risk group was a score of $\geq$7; for recruitment into the comparison group, the criterion was a score of $\leq$3 with background characteristics similar to the risk group. Infants with a history of prenatal, perinatal, or postnatal complications, including low birth weight and prematurity, were excluded. A cutoff score of at least seven symptoms for the risk group was used because it was high enough above the mean to indicate moderate symptomatology, but low enough to ensure a risk sample large enough for longitudinal tracking. The validity of this sampling procedure is supported by the findings of Faraone and Biederman (2005). They found that 2.9% of their sample initially met diagnostic criteria for ADHD; however, when these criteria were broadened to include milder impairing symptoms of the disorder, prevalence increased to 16.4%—similar to that in the current study (19.11%).

Subjects were restricted to male infants because ADHD is more prevalent among boys than girls, with a ratio of 3:1 (Barkley, 1990; Danckaerts & Taylor, 1995; Szatmari, Offord, & Boyle, 1989). Moreover, according to Johnston and Mash (2001), familial and genetic factors may affect boys and girls in different ways. Indeed, most of the studies on ADHD in children have investigated boys only; the choice of male infants as subjects simplifies the connection of our results with prevalent findings on older children with ADHD.

Risk and control groups were chosen based on paternal symptoms because children are more at risk if the affected parent is of their gender (Minde et al., 2003; Rutter & Quinton, 1984). In addition, Biederman et al. (2002) found that exposure to maternal ADHD was not associated with more impairment than was exposure to paternal ADHD.

Mothers were not assessed for ADHD at the maternity ward, in which they stayed for a short time after delivery; however, when the infants were 2 to 6 months old, a psychiatric interview was conducted with each of the parents at their home. At this time, they also completed the Conners Adult ADHD Rating Scale (Conners, Erhardt, & Sparrow, 1998) on themselves and on their spouses.

Interviews were conducted by two 5th-year medical students and two clinical psychology students trained and supervised by a psychiatrist using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID [I]; First, Spitzer, Gibbon, Williams, & Benjamin, 1996). As that does not include questions on ADHD, they also administered the ADHD module from the Schedule of Affective Disorder and Schizophrenia for School-Age Children, Epidemiologic Version (K-SADS-E) (Orvashal & Puig-Antich, 1987), worded in a way appropriate for adults. The interviews were analyzed by the psychiatrist and a clinical psychologist. To establish diagnostic reliability, each of them independently diagnosed the same 32 interviews. In four cases, they disagreed on the diagnosis (12.5%). These disagreements were resolved by discussion, and the rest of the interviews were divided between them. The interviewers and the diagnosticians were blind to the previous assessment of the fathers or their group assignment.

The following mental disorders were assessed: depression, adjustment disorder, posttraumatic stress disorder, bipolar disorder, obsessive-compulsive disorder, dysthymia, panic disorder, generalized anxiety disorder, social phobia, and substance abuse. Every parent who was diagnosed with at least one of these mental disorders was considered as having psychopathology.

The larger sample of the longitudinal study from which the present study is a subsample included 186 families at the first point of assessment (at 2–3 weeks). The mean level of paternal symptoms in this larger sample was: risk group $M = 9.73$, $SD = 2.34$; control group $M = 1.47$, $SD = 1.12$. The subsample of 44 infants and their parents in the present study did not differ significantly in terms of background variables from the 142 infants and their parents who did not participate nor did the infants in these two groups differ in terms of the percentage of first-born children (37 and 31%, respectively) or the frequency of psychiatric disorders among mothers (15 and 19%, respectively) or fathers (17 and 10%, respectively). Only 1 mother in the risk group was diagnosed with mild ADHD.

Of the 44 infants who participated in the current study, 26 were in the risk group and 18 were in the control group. The mean number of symptoms of the fathers was: risk group $M = 9.58$, $SD = 2.23$; control group $M = 1.67$, $SD = 1.19$. Background data for these infants and their parents are presented in Table 1. No differences were found between risk and control group for fathers, mothers, or infants on the background variables and on infant age nor did they differ in percentage of first-born infants (risk group 29%, comparison group 33%). However, although the groups did not differ in frequency of maternal psychopathology (risk group 21%,

TABLE 1. *Background Information on Infant and Parents for Risk and Control Groups*

	Risk			Control		
	n	*M*	*SD*	*n*	*M*	*SD*
Child						
Gestational age (weeks)	23	39.75	1.48	17	39.59	1.46
Chronological age (days)	26	46.89	17.07	18	43.11	12.73
Weight at birth (gr)	25	3442.00	452.81	18	3375.00	360.31
Birth order	24	2.41	1.21	18	2.44	1.42
Mother						
Age	26	28.54	4.25	18	29.31	5.93
Years of education	26	13.29	2.13	18	13.61	2.35
Father						
Age	26	31.92	5.14	18	33.75	5.21
Years of education	26	12.42	1.84	18	13.33	2.40

control group 18%), they did differ in paternal psychopathology (17% in the risk group, none in the control group), and this difference approached significance, $\chi^2(1) = 3.32$, $p = .07$. In two families, both parents were diagnosed with some kind of psychopathology.

Measures

Actigraph. Actigraphy has been established as a valid and reliable method for studying and assessing sleep–wake patterns in infants (Sadeh, 1994, 1996; Sadeh & Acebo, 2002; Sadeh, Acebo, Seifer, Aytur, & Carskadon, 1995; Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991). In the present study, miniature actigraphs (BMA-32, Ambulatory Monitoring Inc., Ardsley, NY) were attached to the infant's left leg for a 24-hr period on 5 consecutive days. The actigraphs were initialized with amplifier setting 18 and a 1-min epoch interval according to the standard working mode for sleep–wake scoring. The Sadeh sleep–wake scoring algorithm was used to score the raw data and provide the summary sleep–wake measures (Sadeh et al., 1995).

Actigraphic sleep measures included (a) duration of sleep: time identified as sleep excluding all periods of wakefulness; (b) quiet sleep: time identified as sleep with zero activity level; and (c) longest sleep: the longest continuous sleep episode with no wake interruptions. These measures were chosen in light of the difficulty in assessing 24-hr sleep patterns in such young infants. At this age, it is practically impossible to define a nocturnal sleep period; sleep–wake cycles are still distributed around the clock, with prolonged wake periods during the night. This makes it difficult to determine other sleep measures such as onset time and sleep percent. For which sleep episode should onset time be measured – the one that starts at 8:00 p.m. and ends at 10:30 p.m. or the one that starts at 11:30 p.m. and ends at 3:00 a.m.? Is a 3-hr waking in the night considered part of night sleep or just a separate wake episode?

Sleep diary. Mothers were requested to record each time the infant woke up or fell asleep during the day and night in a sleep diary. In addition, for potential artifact control, they were asked to indicate any actigraph-removal times that exceeded 15 min and to note when externally induced motion was involved (e.g., sleeping in a car or on a stroller). When such artifacts were identified, they were excluded from data analysis.

Questionnaire. At the end of 5 days when the actigraph was collected, the mother was interviewed by the researcher, who administered a questionnaire specifically developed for the present study. Questionnaire items referred to location of sleep, soothing behavior, feeding procedure, father's participation in daily infant care, and whether parental expectations of their infant's sleep were met.

Procedure

The first visit to the infants' homes for the longitudinal study took place when they were 2 to 3 weeks old. During this visit, the longitudinal study was described to both parents in greater detail, and they signed an agreement attesting to their informed consent. Infants then were assessed using the Neonatal Behavioral Assessment Scale (Brazelton & Nugent, 1995).

By the time the sleep-pattern assessment began, about one third of the total infant sample was already over 7 weeks old and had to be excluded. The research team described the sleep study at the end of the initial home visit to the remaining families and asked them to participate; of these, 87 parents agreed. Approximately 2 to 3 weeks later, the team contacted these parents by telephone to schedule another home visit and begin the sleep assessment. At this time, only 47 of the 87 parents agreed to participate. Data for 2 infants could not be used due to technical problems, and 1 infant was excluded because of low birth weight. The proportion of the risk versus comparison group in the current sample of 44 infants was nevertheless similar to the proportion of these groups in the entire sample of 186 infants.

Immediately before the home visit for the sleep study, the actigraph was initialized on the computer in the lab. During the visit, the study team demonstrated the use of the actigraph and explained the use of the sleep diary. A second visit was scheduled 5 days later to collect the actigraph and to fill out the questionnaire with the mother. If for any reason during one of the 5 days the actigraph assessment could not be performed properly, an additional day was added to the assessment. During the 5 days, the parents were called to find out whether any problems occurred and to encourage them to continue with the assessment. Data from the actigraph were downloaded onto a computer. When large differences were found between the actigraph and the diary, parents were contacted to clarify the situation. The study team consisted of psychology students who were blind to the group participation of the infants.

Data Analysis

The age of the infants in the two groups (risk and control) did not differ significantly, but the standard deviation for age within each group was quite large. At this young age, such a difference can be meaningful for the development of sleep. Age was therefore a covariate in the analysis of actigraph data. The 24-hr data was first analyzed by a multivariate analysis of covariance (MANCOVA), with group as the independent variable, age as the covariate, and the set of sleep measures (duration of sleep, quiet sleep, and longest sleep) as dependent variables. After this analysis, separate ANCOVAs were conducted on each of these sleep measures. In light of findings that a distinction between nocturnal and daytime periods is reflected in infant sleep from the first days of life (Sadeh et al., 1996), and to capture hypothesized differences between the groups in sleep patterns during the day, two additional analyses were conducted in the same way as for the 24-hr data: one only for nighttime data (7 p.m. to 7 a.m.) and one only for daytime data (7 a.m. to 7 p.m.).

TABLE 2. *Group Means of Averaged Actigraph Sleep Measures over Five 24-hr Periods, Nights and Days, by Risk and Control Groups*

	Risk		Control		
	M	*SD*	*M*	*SD*	*F*(1, 41)
24 hr					
Duration of sleep	825.12	97.92	838.51	108.86	0.21
Quiet sleep	380.59	65.52	379.01	80.64	0.10
Longest sleep	236.20	72.45	245.90	70.72	0.59
Night (7 p.m.–7 a.m.)					
Duration of sleep	512.85	50.97	510.19	61.34	0.00
Quiet sleep	238.68	40.08	234.43	52.77	0.04
Day (7 a.m.–7 p.m.)					
Duration of sleep	312.26	75.26	328.32	69.84	0.36
Quiet sleep	141.84	35.71	144.57	40.39	0.11

For each child, two scores were calculated from the actigraph data: an average (the mean score for the five 24-hr periods, for the 5 nights, and for the 5 days) and a stability or intraindividual variability score (the *SD* between the five 24-hr periods, the 5 nights, and the 5 days). The differences between the two groups were analyzed for each of these scores. Group differences in the data obtained from psychiatric evaluation of the parents and in terms of birth order (i.e., sibling order) were analyzed by χ^2, and group differences in questionnaire responses were analyzed by a *t* test. Effect size of ANCOVA results was evaluated with partial η^2; *t*-test results were evaluated with Hedges' *g*.

RESULTS

Actigraph Data

No differences were found between risk and control groups by the MANCOVA in terms of average score for the entire 24-hr periods or when nights and days were calculated separately (Table 2). In addition, no group differences were revealed for sleep-stability scores (*SD* over 5 days) for the 24-hr periods or the nights. However, a significant difference was found in day-to-day stability in the sleep measures, $F(2, 40) = 5.38$, $p = .01$, where the risk group had less consistent daily sleep patterns than did the control group. When each of the measures was analyzed separately (ANCOVAs), day-to-day variability was greater for quiet sleep in the risk group than for that in the control group (see Table 3). The effect size was ($\eta\ p^2 = .212$). According to Cohen (1988), an effect size larger than .14 is considered to be a large effect.

For both groups, the amount of quiet sleep during the 24-hr assessment was negatively correlated with birth order: Less quiet sleep was found in first-born than in later born infants, $r = .29$, $p = .053$. In addition, a positive correlation was found between quiet sleep and age for data on the 24-hr periods and the nights, $r = .44$, $p = .002$, and $r = .57$, $p = .001$, respectively. No correlation was found between age and quiet sleep during the daytime hours.

TABLE 3. *Group Means of Intraindividual Variability of Actigraph Sleep Measures over Five 24-hr Periods, Nights and Days, by Risk and Control Groups*

	Risk		Control		
	M	*SD*	*M*	*SD*	*F*(1, 41)
24 hr					
Duration of sleep	140.10	53.20	138.38	58.75	0.89
Quiet sleep	84.60	24.50	73.58	31.53	1.32
Night (7 p.m.–7 a.m.)					
Duration of sleep	65.73	47.73	74.16	56.88	0.36
Quiet sleep	42.84	20.88	45.07	29.30	0.21
Day (7 a.m.–7 p.m.)					
Duration of sleep	75.31	26.30	64.15	20.59	2.62
Quiet sleep	41.70	12.45	20.58	13.89	11.10*

*$p = .002$, $\eta p^2 = .212$.

Data from Maternal Diaries

The mother's records yielded data on waking and sleep duration for at least 3 of the 5 nights only for 38 infants (22 in the risk group, 16 in the control group). With respect to daytime data (for at least 3 of the 5 days), records were available only for 36 infants (22 in the risk group, 14 in the comparison group). Two measures of sleep were calculated from the mother's diaries: number of wakings and sleep duration. Again, averages and *SD*s were calculated, for both the nights and the days, for each infant.

Number of wakings. Groups did not differ significantly in average number of awakenings at night or during the day (Table 4) nor were significant differences found in *SD*s for night wakings

TABLE 4. *Group Means for Wakings and Sleep Duration: Average Scores from Maternal Diaries for Nights and Days, by Risk and Control Groups*

	Risk			Control			
	n	*M*	*SD*	*n*	*M*	*SD*	*F*(1, 41)
Wakings							
Night (7 p.m.–7 a.m.)	22	2.80	0.79	16	2.54	0.94	.89
Day (7 a.m.–7 p.m.)	22	3.84	0.85	14	3.96	0.99	.11
Duration of Sleep							
Night (7 p.m.–7 a.m.)	22	490.32	54.75	16	477.60	54.85	.53
Day (7 a.m.–7 p.m.)	22	376.47	62.62	14	382.29	52.18	.16

TABLE 5. *Group Means for Wakings and Sleep Duration: Intraindividual Variability Scores from Maternal Diaries for Nights and Days, by Risk and Control Groups*

	Risk			Control			
	n	*M*	*SD*	*n*	*M*	*SD*	*F*(1, 41)
Wakings							
Night (7 p.m.–7 a.m.)	22	0.75	0.45	16	0.75	0.31	.00
Day (7 a.m.–7 p.m.)	22	0.96	0.41	14	0.62	0.41	6.39**
Duration of Sleep							
Night (7 p.m.–7 a.m.)	22	50.43	31.07	16	58.07	39.75	.56
Day (7 a.m.–7 p.m.)	22	85.89	36.89	14	64.33	25.47	3.53*

*$p = .069$, $\eta\, p^2 = .096$.
**$p = .016$, $\eta\, p^2 = .162$.

(Table 5). However, when chronological age since birth date was the covariate, a significant group difference was obtained for day-to-day intraindividual stability, yielding a rather large effect size, $F(1, 30) = 6.39$, $p = .016$, $\eta p^2 = .162$ (see Table 5). The risk group was less stable than was the control group in terms of the day-to-day number of daytime wakenings. Note that when gestational age was a covariate, differences between the groups only approached significance, $F(1, 30) = 3.92$, $p = .057$, $\eta p^2 = .116$. In this case as well, more day-to-day instability during the daytime hours was reported by mothers in the risk group than those in the control group.

Duration of sleep. As indicated in Table 4, no group differences were found in terms of average sleep duration during the nights or days nor were there group differences in night-to-night stability (Table 5). However, the differences in day-to-day stability between the groups approached significance ($p < .07$; see Table 5). The risk group tended to be more variable than was the control group.

Questionnaire. The groups did not differ in frequency of bottle or breast feeding or in place of sleep and ways of putting infants to bed. The mothers in the two groups also did not differ on their expectations about their infant's sleep patterns. However, the groups did differ significantly in paternal involvement in caring for the infant, $t(1, 42) = 3, 23$, $p = .002$, $g = .96$. Fathers with ADHD symptoms (the risk group) were reported by their spouses to be less involved in caring for their infants than were fathers in the control group.

DISCUSSION

In the present study, sleep patterns of 7-week-old boys at familial risk for ADHD and a control group were recorded during consecutive 24-hr periods with objective (actigraph) and subjective (maternal diary) measures. When intraindividual stability was considered, no difference was found between the risk and the control group on actigraphic sleep measures during the full 24-hr periods or only during the nights. However, differences were significant during the daytime hours: The risk group was more inconsistent from day to day in terms of the amount of quiet

sleep. The risk group also was significantly less stable on the day-to-day number of daytime awakenings obtained from maternal diaries. Findings on stability of sleep duration during the days, yielded from the diaries, pointed in the same direction, but group differences only approached significance. Here, too, no group differences were found with regard to the full 24-hr periods or the nighttime hours. In other words, both the subjective findings on day-to-day stability and some of the objective findings from the actigraph support the research hypothesis.

In contrast, when averages over the nights were calculated, the risk group did not differ from the control group on the objective measures or the subjective reports. That is, the groups did not differ in the actigraphic measures of sleep duration, quiet sleep, or longest sleep episode, whether calculated for the full 24-hr periods, the nights only, or the days only nor did they differ on averages of sleep duration and number of awakenings calculated from the maternal diaries.

Note that the present findings on actigraphic averages for the 24-hr periods resemble those for sleep measures in early infancy in the general population (Sadeh et al., 1996; Sadeh et al., 2009). In a study by Sadeh et al. (1996), infants delivered at a later gestational age spent more time in quiet sleep. In the present study, for the whole sample, a significant correlation ($r = .44$) was found between amount of quiet sleep and age for the 24-hr periods and the nights, but no significant correlation was obtained for the days. Sadeh et al. (1996) also found that first-born infants spent significantly less time in quiet sleep compared to subsequent infants. In the present study, for the whole sample, a significant correlation ($r = .43$) was found between birth order and variability of quiet sleep during the 24-hr period: First-born infants were more variable than were subsequent infants.

It is reasonable to suggest that the larger difference in intraindividual variability between the groups, found in the present study when chronological age was the controlled for (compared to gestational age), reflects environmental influences. Taken together, these findings may indicate that quiet sleep is sensitive to environmental regulatory factors in addition to neurodevelopment maturational processes as early as at 7 weeks.

It has been argued that the organization of sleep patterns into consistent sleep–wake cycles reflects proper neurodevelopment of sleep (Halperin et al., 1995; Parmelee & Stern, 1972); however, those studies did not separately examine daytime sleep patterns. The present study has shown that a separate investigation of sleep patterns during the day in early infancy can help distinguish between instability that is related predominantly to physiological factors (the findings for the 24-hr period and the nights only period) and instability that can be related largely to environmental regulatory factors. The latter is reflected in findings that (a) the two groups differed on variability of quiet sleep and awakening only during the day; (b) first-born infants had reduced quiet sleep; (c) there was a greater effect on variability of quiet sleep when chronological age (rather than gestational age) was controlled for; and (d) while variability of quiet sleep decreased with age during the nights, it did not correlate with age during the day.

Our actigraph results suggest that at 7 weeks, the risk group for ADHD is similar to that of the control group in stability of the sleep measure that was found to differ in older children with ADHD (sleep duration). These findings are in line with those of Gruber and Sadeh (2004) and Mick et al. (2000) on preschool and school-age children with ADHD which suggested that sleep systems and ADHD do not share the same physiological structures.

It is reasonable to assume that the larger day-to-day inconsistency we found on quiet sleep and the subjective measures in the daytime-sleep patterns of the risk group, as compared to that of the control group, may be related to parental behavior. During infancy, mothers in most families are the main caregivers. In the present study, the mothers did not have ADHD symptomathology;

it was their spouses who had, on average, about 10 symptoms of the disorder. However, ADHD in one parent has been found to change the regulation of the entire family system and to impair familial and marital functions regardless of the gender of the affected parent (Biederman et al., 2002; Minde et al., 2003; Weiss et al., 2000). A hyperactive, impulsive, and inattentive parent may have difficulties with the instrumental and organizational tasks of parenting. Such a parent can impact the family as a whole. These effects can be more pronounced when reorganization of the family system is required upon the birth of a new infant.

This reasoning is supported by the current finding that mothers in the risk group perceived their spouses as significantly less involved in caring for their infant than were fathers in the control group. In a partially overlapping sample (59%), we also found that both parents in the risk group reacted less adequately to their 7-month-old infant's needs during free play, especially his negative emotions, even though these infants did not need more help than did the control group (Landau et al., 2010). Such findings lend support to the assumption that already during the first half-year of the infant's life, parenting is less optimal in this risk group. This can impact the way in which the sleep–wake cycle is handled, especially when negative emotions are involved. At this age, infants need more help from the parent to soothe themselves and to assuage negative emotions before falling asleep. Soothing behavior recently was found to mediate the relation between maternal cognition and infant sleep (Tikotzky & Sadeh, 2009). A less organized household may result in less consistent parental behavior, which is probably more pronounced during the day when parents have to relate to many other tasks.

How can the effects of environmental disorganized and impulsive behavior explain inconsistency of quiet sleep in infants during the day? The sleep cycles of young infants are shorter than those at older ages, and the cycles start with active sleep followed by quiet sleep. One possible explanation is that in disorganized and inconsistent households, parents interfere more with their infant's sleep patterns during the day in keeping with their own needs, which may change from hour to hour and from day to day. It is reasonable to assume that such interference may occur more frequently during the later part of the sleep cycle, after the child has slept for a while. Maternal reports of greater day-to-day instability in number of awakenings during the daytime hours in the risk group than that in the control group support such an explanation. More research is needed to investigate this hypothesis.

Sleep patterns are established during the first years of life and are strongly related to environmental regulation of sleep (Adair et al., 1991; Anders et al., 1992; Beltramini & Hertzing, 1983; Johnson, 1991; Keener et al., 1988; Sadeh & Anders, 1993; Scher, 2001; Scher et al., 1995). These patterns continue to be quite stable later in life (Kataria et al., 1987; Richman et al., 1982; Zuckerman et al., 1987). It may well be that when the majority of the infant's sleep is concentrated at night, the acquired inconsistent pattern of quiet sleep during the day will be expressed during the night. Moreover, if parental behavior that causes inconsistent daytime-sleep patterns at 7 weeks continues at bedtime, when the child is older, its accumulation can cause instability in the sleep patterns that were found to be instable in older children with ADHD. This reasoning gets some support from the findings on a follow-up at $6\frac{1}{2}$ months of 28 (64%) infants from the present study. For these children, a significant correlation was found between variability of quiet sleep during the day at 7 weeks and variability of duration of sleep at $6\frac{1}{2}$ months at night, $r(28) = .37$, $p = .05$. Duration of sleep was found to be more variable for older children with ADHD than for the controls (Gruber et al., 2000). No such significant correlation was found for variability at night at 7 weeks and nights at $6\frac{1}{2}$ months or for variability of quiet sleep during the day at 7 weeks and any average score of sleep measures at $6\frac{1}{2}$ months.

The findings of Sadeh et al. (2009) with regard to the effect of parental soothing behavior also can support such an interpretation. It therefore seems reasonable to hypothesize that at least some of the intraindividual inconsistency found in studies of adolescent children with ADHD (Gruber & Sadeh, 2004; Gruber et al., 2000) can be attributed to environmental regulatory variables.

Limitations of the Study

At this point of the longitudinal study, we cannot tell which of the children will develop ADHD. When diagnosed, it will be of interest to study the correspondence between sleep patterns at 7 weeks of the infants who developed ADHD and those of the infants who did not develop this disorder. Another limitation of the study is the loss of participants who agreed at the first home visit to participate in the sleep assessment. About half of them changed their mind and refused to participate when the experimenter called them for arranging the assessment. However, the group who agreed to participate in the sleep study did not differ from the main sample on all relevant background variables that were studied (parental demographic variables, number of father's ADHD symptoms, parental pathology, and subjects' birth order). Additional limitations are the small sample size and that only boys were assessed.

In sum, when intraindividual stability of sleep patterns was measured, the risk group for ADHD was less stable on quiet sleep (actigraph), woke more, and tended to exhibit more variability in sleep duration (mother's diaries) during the day than did the control group whereas the same measures yielded no group differences during the full 24-hr periods or nighttimes. In addition, when age since birth date was covariate, more day-to-day inconsistency was reported by mothers in the risk group than by those the control group; in both groups (just as in the general population), less quiet sleep was found in first-born than in later-born infants during the 24-hr periods. Finally, mothers reported that fathers of the risk group were less involved in caring for their infants than those in the control group. Taken together, these findings suggest that at 7 weeks of age, infants at risk for ADHD are less stable in their sleep patterns only during the day, when environmental regulatory factors are more intensive.

REFERENCES

Adair, R., Bauchner, H., Philipp, B., Levenson, S., & Zuckerman, B. (1991). Night waking during infancy: Role of parental presence at bedtime. Pediatrics, 87(4), 500–504.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Anders, T.F., Halpern, L.F., & Hau, J. (1992). Sleeping through the night: A developmental perspective. Pediatrics, 90(4), 554–560.

Auerbach, J.G., Atzaba-Poria, N., Berger, A., & Landau, R. (2004). Emerging developmental pathways to ADHD: Possible path markers in early infancy. Neural Plasticity, 11(1–2), 29–43.

Auerbach, J.G., Landau, R., Berger, A., Arbelle, S., Faroy, M., & Karplus, M. (2005). Neonatal behavior of infants at familial risk for ADHD. Infant Behavior & Development, 28, 220–224.

Barkley, R.A. (1990). Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford Press.

Barkley, R.A. (1998). Attention deficit hyperactivity disorder. Scientific American, 249(3), 66–71.

Beltramini, A.U., & Hertzing, M.E. (1983). Sleep and bedtime behavior in preschool-age children. Pediatrics, 71(2), 153–158.

Biederman, J., Faraone, S.V., Mick, E., Spencer, T., Wilens, T., Kiely, K., et al. (1995). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. American Journal of Psychiatry, 152, 431–435.

Biederman, J., Faraone, S.V., & Monuteaux, M.C. (2002). Impact of exposure to parental attention-deficit hyperactivity disorder on clinical features and dysfunction in the offspring. Psychological Medicine, 32(5), 817–827.

Brazelton, T.B., & Nugent, J.K. (1995). Neonatal behavior assessment scale. Cambridge, England: Cambridge University Press.

Campbell, S.B. (2002). Behavior problems in preschool children: Clinical and developmental issues. New York: Guilford Press.

Cloninger, C.R. (1987). A systematic method for clinical description and classification of personality variants. Archives of General Psychiatry, 44, 573–588.

Cohen, J. (1988). Statistical power analysis for the behavioral sciences (2nd ed.). Hillsdale, NJ: Erlbaum.

Conners, C.K., Erhardt, D., & Sparrow, E.P. (1998). Conners Adult ADHD Rating Scale. Toronto, Canada: Multi-Health Systems.

Corkum, P., Tannock, R., & Molodofsky, H. (1998). Sleep disturbances in children with attention-deficit/hyperactivity disorder. Journal of the American Academy of Child and Adolescent Psychiatry, 37, 637–646.

Dagan, Y., Luria, S.Z., Sever, Y., Hallis, D., Yovel, I., Sadeh, A., & Dolev, E. (1997). Sleep quality in children with attention-deficit/hyperactivity disorder. Psychiatry and Clinical Neurosciences, 51, 383–386.

Dahl, R.E. (1996). The regulation of sleep and arousal. Development and Psychopathology, 8, 3–27.

Danckaerts, M., & Taylor, E. (1995). The epidemiology of childhood hyperactivity. In F.C. Verhults & H.M. Koot (Eds.), The epidemiology of child and adolescent psychopathology (pp. 178–209). Oxford, England: Oxford Medical Press.

Faraone, S.V., Biederman, J., & Monuteaux, M.C. (2000). Toward guidelines for pedigree selection in genetic studies of attention deficit hyperactivity disorder. Genetic Epidemiology, 18, 1–15.

Faraone, S.V., & Biederman, J. (2005). What is the prevalence of ADHD? Results of a population screen of 966 adults. Journal of Attention Disorders, 9, 384–391.

First, M. B., Spitzer, R. L., Gibbon, M., Williams, J. B., & Benjamin, L. (1996). Structured clinical interview for DSM-IV Axis I Disorders. Washington, DC: American Psychiatric Association.

Gruber, R., & Sadeh, A. (2004). Sleep and neurobehavioral functioning in boys with attention-deficit/hyperactivity disorder and no reported breathing problems. Sleep, 27(2), 267–273.

Gruber, R., Sadeh, A., & Raviv, A. (2000). Instability of sleep patterns in children with attention-deficit/hyperactivity disorder. Journal of the American Academy of Child and Adolescent Psychiatry, 39(4), 495–501.

Halperin, W.E., Maclean, L.F., & Baumeister, A.A. (1995). Infant sleep–wake characteristics: Relation to neurological status and the prediction of developmental outcome. Developmental Review, 15, 255–291.

Harvey, A., Danforth, J.S., McKee, T.E., Ulaszek, W.R., & Friedman, J.L. (2003). Parenting of children with attention-deficit/hyperactivity disorder (ADHD): The role of parental ADHD symptomatology. Journal of Attention Disorders, 7(1), 31–42.

Hechtman, L. (2000). Assessment and diagnosis of attention deficit/hyperactivity disorder. Child and Adolescent Psychiatric Clinics of North America, 9(3), 481–498.

Johnson, M. (1991). Infant and toddler sleep: A telephone survey of parents in the community. Journal of Developmental and Behavioral Pediatrics, 12, 108–114.

Johnston, J., & Mash, E.J. (2001). Families of children with attention-deficit/hyperactivity disorder: Review and recommendations for future research. Clinical Child and Family Psychology Review, 4(3), 183–207.

Kateria, S., Swanson, M., & Trevathan, G. (1987). Persistence of sleep disturbances in preschool children. Journal of Pediatric Psychology, 5, 295–303.

Keener, M.A., Zeanah, C.H., & Anders, T.F. (1988). Infant temperament, sleep organization, and nighttime parental intervention. Pediatrics, 81(6), 762–771.

Landau, R., Amiel-Laviad, R., Berger, A., Atzaba-Poria, N., & Auerbach, J.G. (2009). Parenting of 7-month-old infants at familial risk for ADHD during infant's free play, with restrictions on interaction. Infant Behavior & Development, 32, 173–182.

Landau, R., Avital, M., Berger, A., Atzaba-Poria, N., Arbelle, S., Faroy, M., & Auerbach, J.G. (2010). Parenting of 7-month-old infants at familial risk for ADHD. Infant Mental Health Journal, 31(2), 141–158.

Mick, E., Biederman, J., Jetton, J., & Faraone, S.V. (2000). Sleep disturbances associated with attention deficit hyperactivity disorder: The impact of psychiatric co-morbidity and pharmacotherapy. Journal of Child and Adolescent Psychopharmacology, 10, 223–231.

Minde, K., Eakin, L., Hechtman, L., Ochs, R., Bouffard, R., Greenfield, B., & Looper, K. (2003). The psychosocial functioning of children and spouses of adults with ADHD. Journal of Child Psychology and Psychiatry, 44(4), 637–646.

Mindell, J.A., Telofski, L.S., Wiegand, B., & Kurtz, E.S. (2009). A nightly bedtime routine: Impact on sleep in young children and maternal mood. Sleep, 32(5), 599–606.

Murray, C., & Johnston, C. (2006). Parenting in mothers with and without attention-deficit/hyperactivity disorder. Journal of Abnormal Psychology, 155(1), 52–61.

Orvashal, H., & Puig-Antich, J. (1987). Schedule for Affective Disorders and Schizophrenia for School-Age Children, Epidemiologic Version. Ft. Lauderdale, FL: Nova University, Center for Psychological Study.

Parmelee, A.H., Jr., & Stern, E. (1972). Development of states in infants. In C. Clemente, D. Purput, & F.E. Mayers (Eds.), Sleep and the maturing nervous system (pp. 199–228). New York: Academic Press.

Price, T.S., Simonoff, E., Asherson, P., Curran, S., Kuntisi, J., Waldman, I., & Plomin, R. (2005). Continuity and change in preschool ADHD symptoms: Longitudinal genetic analysis with contrast effects. Behavior Genetics, 35(2), 121–132.

Richman, N., Stevenson, J., & Graham, P. (1982). Preschool to school: A behavioral study. London: Academic Press.

Rutter, M., & Quinton, D. (1984). Parental psychiatric disorder: Effects on children. Psychological Medicine, 14, 858–880.

Sadeh, A. (1994). Assessment of intervention for infant night waking: Parental reports and activity-based home monitoring. Journal of Consulting Clinical Psychology, 62, 63–68.

Sadeh, A. (1996). Evaluating night wakings in sleep disrupted infants: Methodological study of parental reports and actigraph. Sleep, 19, 757–762.

Sadeh, A., & Acebo, C. (2002). The role of actigraphy in sleep medicine. Sleep Medicine Reviews, 6, 113–124.

Sadeh, A., Acebo, C., Seifer, R., Aytur, S., & Carskadon, M.A. (1995). Activity-based assessment of sleep–wake patterns during the first year of life. Infant Behavior & Development, 18, 329–337.

Sadeh, A., & Anders, T.F. (1993). Infant sleep problems: Origins, assessment, interventions. Infant Mental Health Journal, 14(1), 17–34.

Sadeh, A., Dark, I., & Vohr, B.R. (1996). Newborns' sleep–wake patterns: The role of maternal, delivery and infant factors. Early Human Development, 44, 113–126.

Sadeh, A., Lavie, P., Scher, A., Tirosh, E., & Epstein, R. (1991). Actigraph home-monitoring of sleep-disturbed and control infants and young children: A new method for pediatric assessment of sleep–wake patterns. Pediatrics, 87, 494–499.

Sadeh, A., Mindell, J.A., Luedtke, K., & Wiegant, B. (2009). Sleep and sleep ecology in the first 3 years: A web-based study. Journal of Sleep Research, 18, 60–73.

Sadeh, A., Pergamin, L., & Bar-Haim, Y. (2006). Sleep in children with attention-deficit/hyperactivity disorder: A meta-analysis of polysomnographic studies. Sleep Medicine Reviews, 10, 381–398.

Scher, A. (2001). Attachment and sleep: A study of night waking in 12-month-old infants. Developmental Psychobiology, 38, 274–285.

Scher, A., Tirosh, E., Jaffe, M., Rubin, L., Sadeh, A., & Lavie, P. (1995). Sleep patterns of infants and young children in Israel. International Journal of Behavioral Development, 18(4), 701–711.

Smalley, S.L., McGough, J.J., del'Homme, M., NewDelman, J., Gordon, E., Kim, T., et al. (2000). Familial clustering of symptoms and disruptive behaviors in multiplex families with attention-deficit/hyperactivity disorder. Journal of the American Academy of Child and Adolescent Psychiatry, 39(9), 1135–1143.

Szatmari, P., Offord, D.R., & Boyle, M.H. (1989). Ontario Child Health Study: Prevalence of attention deficit disorder with hyperactivity. Journal of Child Psychology and Psychiatry, 30, 219–230.

Taylor, A. (1999). Developmental neuropsychopathology of attention deficit and impulsiveness. Developmental Psychopathology, 11, 607–628.

Thoman, E.B., Denenberg, V.H., Sievel, J., Zeidner, L.P., & Becker, P. (1981). State organization in neonates: Developmental inconsistency indicates risk for developmental dysfunction. Neuropediatrics, 12(1), 45–54.

Tikotzky, L., & Sadeh, A. (2009). Maternal sleep-related cognitions and infant sleep: A longitudinal study from pregnancy through the first year. Child Development, 80(3), 860–874.

Weiss, M., Hechtman, L.T., & Weiss, G. (2000). ADHD in parents. Journal of the Academy of Child and Adolescent Psychiatry, 39(8), 1059–1061.

Zuckerman, B., Stevenson, J., & Baily, V. (1987). Sleep problems in early childhood: Predictive factors and behavioral correlates. Pediatrics, 80, 664–671.