ELSEVIER

Journal of Affective Disorders 96 (2006) 1 – 8

JOURNAL OF AFFECTIVE DISORDERS

www.elsevier.com/locate/jad

Research report

Early childhood sleep and eating problems as predictors of adolescent and adult mood and anxiety disorders

Say How Ong [a,*], Priya Wickramaratne [b,c], Min Tang [b], Myrna M. Weissman [b,c]

[a] *Department of Child and Adolescent Psychiatry, Child Guidance Clinic, Institute of Mental Health, Singapore*
[b] *Department of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, NY, USA*
[c] *Division of Clinical and Genetic Epidemiology, New York State Psychiatric Institute, New York, NY, USA*

Received 5 April 2006; received in revised form 23 May 2006; accepted 25 May 2006
Available online 17 July 2006

Abstract

Background: Recent studies have suggested that eating and sleep problems during early childhood may pose as risk factors for mood and anxiety disorders in later life. We aim to study the associations between early childhood sleep and eating problems, specifically high motor activity during sleep and irregularities in sleep/eating schedules, and lifetime history of mood and anxiety disorders.
Methods: We followed up 164 offspring, who were at high and low risk for major depression by virtue of their parental history (at least one parent had Major Depressive Disorder). Target sleep and eating problems were measured using Dimensions of Temperament Survey (DOTS). The offspring were blindly assessed at 3 times over 20 years using a structured diagnostic interview.
Result: Irregularities in sleeping and eating schedules in childhood (low rhythmicity) was associated with adolescent-onset major depression and anxiety disorder, as well as childhood-onset anxiety disorder. High motor activity level during sleep was associated with both childhood-onset and adolescent-onset dysthymic disorder. Neither childhood sleep nor eating irregularities were associated with adult onset psychopathology.
Limitations: Retrospective reports of childhood sleep and eating patterns were derived from parent-reports. Reported problems may overlap with clinical diagnoses.
Conclusion: Clinicians should be alerted to parental reports of children's sleep and eating problems suggesting low rhythmicity, as well as high motor activity levels during sleep. These early behaviors may be predictive of subsequent mood and anxiety disorders in childhood and adolescence.

Keywords: Sleep; Eating; Rhythmicity; Activity level; Mood disorder; Anxiety disorder

1. Introduction

Mood and anxiety disorders are debilitating psychiatric conditions often requiring long-term treatment and monitoring, with the first onset often occurring in childhood and adolescence. With no known biological markers available

* Corresponding author. Child Guidance Clinic, Health Promotion Board Building, 3 Second Hospital Avenue, #03-01, Singapore 168937. Tel.: +65 64353867; fax: +65 65343677.
E-mail address: say_how_ONG@imh.com.sg (Say How Ong).

0165-0327/$ - see front matter © 2006 Elsevier B.V. All rights reserved.
doi:10.1016/j.jad.2006.05.025

to diagnose mood and anxiety disorders, the use of behavioral signs that occur during early childhood to predict subsequent psychopathology could be useful. Extensive research has been conducted to study the risk for adult internalizing problems and symptoms based on adolescent symptomatology (Pine et al., 1998) and childhood neurological soft signs (Shaffer et al., 1985; Pine et al., 1993), including somatic illnesses and health problems during early childhood (Jacobsen et al., 1983; Wells et al., 1985; Cohen et al., 1989; Katon and Sullivan, 1990). However, health problems during childhood may go undetected by parents and sometimes even by clinicians, so the search for readily observable and reportable signs as possible identified risk factors is warranted.

Parents are usually knowledgeable about their children's sleep and eating habits, and are thus more likely to notice and complain about any irregularities of daily living routines and their related difficulties. Children on the other hand are poor reporters of appetite problems and to a lesser extent, sleep difficulties (Waters and Storm, 1985). Sleep problems during childhood have generated a reasonable amount of interest in child psychiatry as it has been implicated in several childhood psychopathologies, including anxiety/depression (Ryan et al., 1987; Johnson et al., 2000), attention problems (Dahl et al., 1991; Yuen et al., 1999; Gruber et al., 2000), hyperactivity and conduct problems (Dahl, 1996; Aronen et al., 2000). In a longitudinal study by Gregory and O'Connor (2002) on 490 children, parental reports of sleep problems at age 4 predicted behavioral/emotional problems in mid-adolescence. Similarly, motor activity (measured using objective measurements such as actigraphy or polysomnography) and its influence on childhood psychopathologies, have been studied in children and adolescents with nonseasonal depression (Teicher et al., 1993), major depressive disorder (Armitage et al., 2004), and ADHD (Small et al., 1971; Busby et al., 1981; Porrino et al., 1983; Tirosh et al., 1993). Pediatric sleep-related involuntary movements, identified through sleep questionnaires completed by parents, have been associated with both ADHD and to a greater extent, separation anxiety (Corkum et al., 1999). These studies however measure motor movements of children with existing psychiatric conditions and not premorbid sleep motor disturbances. The presence of sleep disturbances could consequently confer an increased risk for new incidences of psychiatric disorders in children and later in their life. To our knowledge, no studies have yet linked high motor activity during sleep in early childhood with lifetime psychiatric diagnoses. In addition, few studies had specifically looked into sleep and eating habits, both of which constitute part of a more observable and enduring host of temperamental traits during early childhood, that may persist over time and ultimately serve as risk factors for later psychopathology.

Available studies on pediatric sleep and eating disturbances and the correlation with psychiatric diagnoses often were based on shorter follow up periods ending in late childhood or mid-adolescence (Stoléru et al., 1997; Gregory and O'Connor, 2002; Gregory et al., 2004) except for one study (Gregory et al., 2005) which followed up very young children up to young adulthood. The pathological significance of the relationship between early childhood behaviors and adolescent and adult psychiatric diagnoses thus remains interesting. In our study, we hypothesized that those children with early sleep and eating irregularities (low rhythmicity), and high motor activity during sleep are at a higher risk of developing lifetime diagnoses for Major Depressive Disorder (MDD), Dysthymic Disorder (DD) or Anxiety Disorder (ANX) than children without the reported problems. The opportunity to study this relationship arose in our earlier longitudinal study of offspring from high-risk families followed up over 20 years.

The specific questions we propose to address in this study are:

(1) Is there an association between early childhood low rhythmicity and high motor activity during sleep with lifetime MDD/DD/ANX?
(2) What is the relationship between early childhood low rhythmicity and high motor activity during sleep with age of onset of MDD/DD/ANX?
(3) Is there a difference in the rates of maternal reporting of offspring sleep and eating problems in the high and low risk groups?

2. Methods

2.1. Design

This study is derived from a multi-generational high-risk study. In the original study, adult probands with moderate to severe MDD were selected from outpatient clinical specialty settings for the psychopharmacologic treatment of mood disorders. Non-depressed adult probands were also selected, at the same time, from an epidemiologic sample of adults from the same community. They were required to have no lifetime history of psychiatric illness based on several interviews. See Weissman et al. (1987, 1997, 2005) for a full description of methods and materials. This paper reports data from probands reporting on their children at Wave 1 (intake interview), aged 6 to 23 years. These children were later assessed at Wave 10 (10 years later) and Wave 20 (approximately 20 years later) to detect lifetime MDD,

Table 1
Association between parental report on offspring's sleep/eating variables at Wave 1 and parental MDD status

Sleep/eating variables	One or more parents depressed (n = 108)		Neither parent depressed (n = 56)		n (%) (N = 164)	P-value
'My child wakes up at different times'						
Yes	37	34.3%	12	21.4%	49 (29.9%)	0.09
No	71	65.7%	44	78.6%	115 (70.1%)	
'There is no set time when my child goes to sleep'						
Yes	46	42.6%	18	32.1%	64 (39.0%)	0.19
No	62	57.4%	38	67.9%	100 (61.0%)	
'My child eats about the same amount for dinner whether he/she is home, visiting someone, or traveling'						
Yes	71	66.4%	36	64.3%	107 (65.6%)	0.79
No	36	33.6%	20	35.7%	56 (34.4%)	
'My child moves a great deal in his/her sleep'						
Yes	30	28.3%	9	16.1%	39 (24.1%)	0.08
No	76	71.7%	47	83.9%	123 (75.9%)	
'My child seems to get sleepy just about the same time every night'						
Yes	71	67.0%	41	73.2%	112 (69.1%)	0.41
No	35	33.0%	15	26.8%	50 (30.9%)	
'When my child is away from home, he/she still wakes us at the same time each morning'						
Yes	54	51.4%	31	55.4%	85 (52.8%)	0.63
No	51	48.6%	25	44.6%	76 (47.2%)	
'My child eats about the same amount of breakfast from day to day'						
Yes	84	78.5%	48	85.7%	132 (81.0%)	0.27
No	23	21.5%	8	14.3%	31 (19.0%)	
'My child moves a lot in bed'						
Yes	32	30.2%	9	16.1%	41 (25.3%)	0.05
No	74	69.8%	47	83.9%	121 (74.7%)	
'My child eats about the same amount at supper from day to day'						
Yes	80	74.8%	44	78.6%	124 (76.1%)	0.59
No	27	25.2%	12	21.4%	39 (23.9%)	
'My child doesn't move around much at all in his/her sleep'						
Yes	68	64.2%	43	76.8%	111 (68.5%)	0.10
No	38	35.9%	13	23.2%	51 (31.5%)	
'My child's appetite seems to stay the same day after day'						
Yes	74	69.2%	43	76.8%	117 (71.8%)	0.30
No	33	30.8%	13	23.2%	46 (28.2%)	

DD and ANX. The interview and assessment procedures were kept similar across all Waves, with few exceptions, to avoid introducing methods bias. The proband, spouse and offspring were interviewed independently and blind to the clinical status of the previous generations and their previous history.

2.2. Sample

At the initial interview in 1982 (Wave 1), the sample consisted of 220 offspring aged 6 to 23 years from 91 families. Only those offspring with their Dimensions of Temperament Survey (DOTS) completed by their parents and who were followed up at Wave 10 and/or Wave 20 were included in this study, resulting in the final total of 74 families with 164 eligible offspring. One offspring had to be removed from the study due to incomplete data. At Wave 10 and Wave 20, approximately 10 years and 20 years respectively after first interview, all 164 offspring were contacted and reassessed for lifetime diagnosis of mental disorders. Attrition rates did not differ by parental diagnosis and there were no significant differences in age and gender of offspring by proband groups. The families from which the offspring were derived were comparable demographically. All waves of interviews were approved by the Institutional Review Board at the New York State Psychiatric Institute/Columbia University.

2.3. Assessment and diagnosis

The diagnostic interviews across all waves were conducted using a semi-structured diagnostic assessment

Table 2
Association between identified childhood sleep/eating problems and later offspring disorders (N=164)

Offspring disorder	AOR*	(95% CI)	P-value
MDD			
High activity level—sleep	1.1	(0.85, 1.47)	0.41
Low rhythmicity—sleep	1.3	(1.04, 1.72)	0.02
Low rhythmicity—eating	1.2	(0.92, 1.52)	0.18
DD			
High activity level—sleep	1.5	(1.11, 1.90)	0.006
Low rhythmicity—sleep	0.9	(0.73, 1.21)	0.63
Low rhythmicity—eating	1.1	(0.83, 1.36)	0.64
ANX			
High activity level—sleep	1.1	(0.87, 1.51)	0.32
Low rhythmicity—sleep	1.2	(0.95, 1.58)	0.11
Low rhythmicity—eating	1.3	(1.03, 1.73)	0.03

* AOR: Odds ratio (OR) adjusted for parental depression status, gender of child and age of child at interview.

— Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L) (Mannuzza et al., 1986) for adults, modified to include Research Diagnostic Criteria (RDC) (Puig-Antich, 1982) with impairment and DSM-III-R (APA, 1987) criteria, and the child version of SADS (K-SADS-E) (Orvaschel et al., 1982) for offspring younger than 18 years of age. The SADS-L is a semi-structured interview providing detailed information on a variety of diagnoses including lifetime anxiety disorders (Generalized Anxiety Disorder, Obsessive Compulsive Disorder, Post Traumatic Stress Disorder, Panic Disorder, Agoraphobia, Simple Phobia, Social Phobia) and affective disorders and symptoms. Information on offspring was derived a parent informant, typically the mother. A life chart was used during interview to enable identification of developmental patterns in the offspring.

Early childhood sleep and eating patterns were elicited from the mothers, based on their general impression of their children's sleeping and eating behaviors up to six years of age, using the Dimensions of Temperament Survey (DOTS) (Lerner et al., 1982). DOTS is a 34-item self-administered report scale in a true–false format, which examines the five dimensions of temperament: activity level (activity during sleep); attention span (task persistence); adaptability (response to new situations); rhythmicity (regularity of eating and sleeping habits); reactivity/irritability (reactivity on sensory stimuli). In our study, only 2 dimensions were assessed, namely items concerning sleep and eating rhythmicities, and motor activity level during sleep. The time frame of reporting was also modified to capture early childhood behavior instead of current expression of behavior. DOTS had been previously used in an earlier high-risk study on depressed parents and their offspring. Moderate to good stability (Pearson correlation r, 0.45–0.58) was found for maternal reports on offspring temperament over two years and the reports were less influenced by the age or sex of the child compared with child self-reports (Mufson et al., 1990). Though primarily used as a measurement of a child's temperament, DOTS has broad age coverage for early childhood behaviors (Maziade et al., 1986) as well as consistency from early childhood to young adulthood. The "activity level — sleep" component addresses motor activity and movement of the child during sleep and the "rhythmicity" component measures regularities in the biological diurnal activities, namely sleep (e.g., consistency in sleep–wake cycle) and eating (e.g., appetite and quantity consumed) activities. The summed responses for the "rhythmicity" statements would suggest how "predictable" the child was in terms of his/her daily sleep and eating habits. Composite measures of all the DOTS components were not calculated because we did not intend to look into all the five temperament dimensions. To adjust for the possibility of over-reporting bias by depressed parents in the high-risk families, we controlled for parental depression status in all our analyses. All interviews were conducted by clinically trained mental health professionals. The final best-estimate diagnosis (Leckman et al., 1982) was made independently, by a psychiatrist or clinical psychologist blinded to the diagnoses of the probands and the offspring's previous assessments at all the 3 times of interview.

2.4. Statistical analyses

Differences in demographic characteristics between depressed and non-depressed probands as well as differences in demographic characteristics of their offspring were tested using t-tests for continuous variables and Chi-squared tests for categorical variables. The association between positive reports of specific DOTS items in offspring and parental MDD status was also tested using Chi square analysis. Rates of psychiatric disorder for each development phase were computed for offspring (Wickramaratne and Weissman, 1998) as follows: Childhood rates were computed by dividing the number of cases with first onset before age 13 years by the total number of offspring. Adolescent rates were computed by dividing the number of cases with first onset between age 13 and 19 years by the number of offspring at risk for an adolescent onset disorder, i.e. by the number who at last interview were 13 years or older and who had not had a first onset before age 13. Adult rates were computed similarly to adolescent rates, except using age 20 instead of age 13. The adult group consisted of offspring age 20 and older at time of last assessment.

Table 3
Association between identified childhood sleep/eating problems and onset of disorders by offspring developmental phase

Offspring disorders	AOR*	(95% CI)	*P*-value
Childhood (age<13)-onset disorders			
MDD			
High activity level—sleep	0.8	(0.55, 1.27)	0.41
Low rhythmicity—sleep	1.2	(0.87, 1.77)	0.23
Low rhythmicity—eating	0.9	(0.63, 1.34)	0.65
DD			
High activity level—sleep	1.4	(1.03, 1.93)	*0.03*
Low rhythmicity—sleep	1.1	(0.79, 1.45)	0.67
Low rhythmicity—eating	1.0	(0.75, 1.39)	0.88
ANX			
High activity level—sleep	1.2	(0.93, 1.61)	0.16
Low rhythmicity—sleep	1.2	(0.93, 1.55)	0.15
Low rhythmicity—eating	1.5	(1.15, 1.92)	*0.003*
Adolescent (age 13–19)-onset disorders			
MDD			
High activity level—sleep	1.2	(0.90, 1.70)	0.20
Low rhythmicity—sleep	1.3	(1.00, 1.81)	*0.05*
Low rhythmicity—eating	1.3	(0.96, 1.67)	0.10
DD			
High activity level—sleep	1.6	(1.04, 2.45)	*0.03*
Low rhythmicity—sleep	0.9	(0.58, 1.35)	0.56
Low rhythmicity—eating	1.1	(0.78, 1.70)	0.48
ANX			
High activity level—sleep	1.1	(0.53, 2.42)	0.75
Low rhythmicity—sleep	2.7	(1.14, 6.60)	*0.02*
Low rhythmicity—eating	0.8	(0.30, 2.08)	0.63
Adult (age>=20)-onset disorders			
MDD			
High activity level—sleep	1.1	(0.70, 1.65)	0.74
Low rhythmicity—sleep	1.2	(0.80, 1.79)	0.37
Low rhythmicity—eating	1.2	(0.78, 1.72)	0.46
DD			
High activity level—sleep	1.1	(0.46, 2.46)	0.89
Low rhythmicity—sleep	0.8	(0.37, 1.83)	0.62
Low rhythmicity—eating	1.2	(0.57, 2.52)	0.63
ANX			
High activity level—sleep	0.9	(0.58, 1.55)	0.83
Low rhythmicity—sleep	1.0	(0.62, 1.51)	0.88
Low rhythmicity—eating	1.1	(0.62, 1.79)	0.84

* AOR: Odds ratio (OR) adjusted for parental depression status, gender of child and age of child at interview.

A series of logistic regression analyses were used to determine the effect of each of the composite DOTS sleeping/eating activity and rhythmicity variables on offspring diagnoses, for lifetime as well as each of the development phases. In these analyses, offspring diagnoses were treated as a dichotomous outcome variable and the sum of positive DOTS items for each of these composite variables was treated as the independent variable. As the number of low-risk children with diagnoses was small, the data for both the high and low risk offspring were combined, and age, and gender of offspring as well as parental depression status were treated as potential confounding variables and controlled for in the analysis.

3. Results

3.1. Demographic profile of proband

Of the 74 probands, 51 parents were in the depressed (high-risk) group and 23 were in the non-depressed (low-risk) group. The parent groups did not vary at the beginning of the study by age (mean age, 39.6 ± 7.7 years), sex (56.8% female), marital status (79.7% married), occupation (97.3% skilled worker and above) and educational level attained (93.3% high school and above).

3.2. Age and gender of offspring by proband group

Of the 164 offspring from the 74 probands in the study, there were 108 children from the depressed probands and 56 children from the non-depressed control probands. The children did not vary by parents' depression status, sex (53.7% female) and age at the beginning of the study (mean age, 16.7 ± 4.7 years). Thirty-four children were between 6 and 12 years old; 75 were 13–19 years old; and 55 were between 20 and 23 years old. At the end of follow-up at Wave 20, the mean age of offspring was 34.1 ± 5.1 years.

3.3. Parental reports of sleep/eating problems on offspring

Table 1 gives the number and percentage of depressed and non-depressed parents reporting on their offspring's sleep and eating problems at Wave 1. While parents reported sleeping and eating behaviors in their offspring, the offspring were assessed independently. There were no statistically significant difference between the two groups, except for the item "My child moves a lot in bed" where a greater number of depressed parents reported significantly more movement in their children during sleep, $p = 0.05$.

At the end of the three times of assessment spanning over approximately 20 years, 41(25%) offspring with MDD had their first onset during adolescence (age 13 to 19 years) while 28 (17.1%) had their onset of DD during childhood (age < 13 years). Likewise, the majority of offspring (56, 34.2%) with any anxiety disorder had their first onset during childhood.

The overall association between sleep/eating problems and offspring disorder is summarized in Table 2. By combining responses of individual DOTS items under each component (activity level, sleep rhythmicity and eating rhythmicity), the effect of each behavior variable on offspring disorders became more pronounced. The results showed that low sleep rhythmicity was predictive of offspring MDD ($p = 0.02$, adjusted odds-ratio, AOR = 1.3). Low eating rhythmicity on the other hand exerted itself prominently on anxiety disorder ($p = 0.03$, AOR = 1.3). High activity level during sleep was predictive of Dysthymic Disorder ($p = 0.006$, AOR = 1.5).

3.4. Psychiatric diagnoses

Table 3 summarizes the association between each sleep/eating problem with onset of disorder by offspring developmental phase.

3.4.1. Major depressive disorder

Low sleep rhythmicity was predictive of adolescent-onset MDD ($p = 0.05$, AOR = 1.3). No association was found between low sleep rhythmicity and MDD of childhood and adulthood onsets. No associations were found between high activity level during sleep and offspring MDD and between low eating rhythmicity and offspring MDD.

3.4.2. Dysthymic disorder

High activity level during sleep was predictive of both childhood-onset ($p = 0.03$, AOR = 1.4) and adolescent-onset DD ($p = 0.03$, AOR = 1.6). Association between high activity level and adult-onset DD was not observed. No associations were found between low sleep/eating rhythmicities and offspring DD.

3.4.3. Anxiety disorder

Low eating rhythmicity was predictive of childhood-onset ANX ($p = 0.003$, AOR = 1.5). Low sleep rhythmicity was however predictive of adolescent-onset ANX ($p = 0.02$, AOR = 2.7). No association between any of the sleep/eating problems and adult-onset ANX was found.

4. Discussion

Our study findings suggest that specific early childhood sleeping and eating behaviors may be predictive of childhood and adolescent-onset mood and anxiety disorders but not adult-onset mood and anxiety disorders. Specifically, low sleep rhythmicity was predictive of both adolescent-onset MDD and ANX while high motor activity during sleep was predictive of childhood and adolescent-onset DD. Low eating rhythmicity, on the other hand, was predictive of childhood-onset ANX. The lack of association between low sleep rhythmicity and MDD in childhood and adulthood may be due to the low rates of MDD in these two developmental phases in our study, resulting in a lack of statistical power to detect any association. An alternative explanation could be that low sleep rhythmicity may belong to a separate group of early childhood problems that have yet to reach threshold levels in offspring with MDD of childhood and adult onsets.

The predictive power is stronger for adolescent-onset disorders than for childhood-onset disorders because it is likely that adolescent-onset illnesses are actual incidences and independent of reported sleep and eating problems, and any childhood sleep/eating problems reported in parental reports would have predated the diagnoses made in adolescence or adulthood. It is difficult to determine whether symptoms of childhood-onset disorders overlapped with childhood behaviours captured with the parental reports, i.e. whether the reported sleep/eating problems during childhood were a consequence or symptom manifestation of childhood-onset mood and anxiety disorders. This problem is shared by two other studies: one study on sleep problems in children of affectively ill mothers (Stoléru et al., 1997) and another study on temperament characteristics of bipolar offspring (Chang et al., 2003). Authors of the latter study further highlighted how temperament constructs and psychopathology interrelate and the difficulty in teasing them apart because of the retrospective design of their study.

Our overall findings are nevertheless consistent with other studies (Aronen et al., 2000; Gruber et al., 2000; Johnson et al., 2000; Gregory et al., 2004), which found sleep disturbances in childhood being associated with anxiety/depression and a range of other behavioral and emotional problems in children. The lack of association between early childhood sleep problems and adult-onset disorders concurs with the finding by Gregory et al. (2005), which found no association between childhood sleep disturbances and adult depression. Their study however looked at 'current' episodes of depression in adulthood and not onsets, having also combined depression with dysthymia as one mood disorder entity.

Some prospective studies, e.g. Johnson et al. (2000), which correlated sleep problems and anxiety/depression at 6 years and 11 years, have a limited follow-up period, thus negating the possibility of anxiety/depression developing at a later age. Despite their finding that the relative risk for anxiety/depression at age 11 associated with sleep problems at age 6 was of the same magnitude as that in adults (Breslau et al., 1996), the result was not statistically significant. Our study of the two generations

thus differs by possessing the strength of having a 20-year follow-up longitudinal data and an opportunity to observe a high-risk sample as they proceed through the risk period of developing mood and anxiety disorders.

Early childhood sleep and eating problems could thus constitute an expression of an underlying vulnerability of offspring to psychiatric disorders. The findings that these problems may predict an increased risk for future major depressive disorder, dysthymic disorder and anxiety disorder therefore have important clinical implications. In addition, it might be pertinent to consider firstly, whether eating and sleep problems in early childhood could also be predictive of bipolar spectrum disorders, which share common biological factors as the disorders we have studied. Secondly, whether such childhood problems might not necessarily be antecedents of mood and anxiety disorders but may be actual manifestations of these illnesses. Because sleep and eating problems are common complaints and information on their severity and persistence can be readily elicited from parents, without relying on the use of actigraphy or polysomnography, health professionals should consider including parental accounts of children's sleep and eating patterns into their interview routine in their clinics. An increased awareness in the possibility of developing mood and anxiety disorders in later life could help pave the way for more appropriate assessment, closer monitoring and timely intervention for the more at-risk children. Future research could further address the possibility of reducing these biological risks of mood and anxiety disorders in at-risk children through regularization of sleep and eating schedules, daily life routines and physical activities.

4.1. Limitations

The number of low-risk offspring developing mood and anxiety disorders was too small for separate meaningful analysis. As such, data from high and low-risk offspring were combined and controlled for parental depression status in the analysis. In addition, the majority of depressed offspring had an onset in childhood or adolescence; consequently the lack of an association in sleeping and eating disorders in childhood and adult-onset psychopathology could be due to low incidence and hence reduced power to detect such an association. The degree of overlap between assessment of childhood-onset psychopathology and sleep/eating problems elicited by DOTS remains largely unknown. This confounding factor reflects the difficulty of correlating temperament-derived variables and constructs, including sleep and eating rhythmicity problems, to psychopathology.

We did not look into the presence of environmental, family and marital problems, which could be potential predisposing factors for early childhood habits and behaviors. Irregularities in sleep and eating habits could stem either from a combination of inconsistent child-rearing practices (Wolin et al., 1980) and family stressors, including interparental marital disorder (Rutter and Pickles, 1991; Davies and Cummings, 1994), or from a product of family disorganization and ongoing family stressors, which interact with a child's neuropsychological and neurophysiological activities, leading to sleep–wake cycle irregularities and other similar difficulties (Davies and Windle, 2001).

Another limitation of the study was the use of retrospective data collection of childhood behaviors by the parent that could be subjected to memory distortion and parental mental state at time of reporting. The child's own self-report of sleeping and eating problems was not used for this study, though Luby and Steiner (1993) did show that parental reports of children's temperament in psychiatric populations are very similar to that of the child's own self-report. Further research could utilize more objective measurements to prospectively examine the influence of early childhood problems on psychopathology.

Acknowledgements

This work was supported in part by NIMH grant R01 MH036197 (Dr. Weissman). The first author is also grateful to the National Healthcare Group (Singapore) for funding his research fellowship in New York State Psychiatric Institute/Columbia University under its Health Manpower Development Plan (HMDP), 2004.

References

APA, 1987. The Diagnostic and Statistical Manual of Mental Disorders, Third Edition Revised. American Psychiatric Association, Washington, DC.

Armitage, R., Hoffmann, R., Emslie, G., Rintelman, J., Moore, J., Lewis, K., 2004. Rest–activity cycles in childhood and adolescent depression. J. Am. Acad. Child Adolesc. Psych. 43, 761–769.

Aronen, E.T., Paavonen, E.J., Fjallberg, M., Soininen, M., Torronen, J., 2000. Sleep and psychiatric symptoms in school-age children. J. Am. Acad. Child Adolesc. Psych. 39, 502–508.

Breslau, N., Roth, T., Rosenthal, L., Andreski, P., 1996. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol. Psychiatry 39, 411–418.

Busby, K., Firestone, P., Pivik, R.T., 1981. Sleep patterns in hyperkinetic and normal children. Sleep 4, 366–381.

Chang, K.D., Blasey, C.M., Ketter, T.A., Steiner, H., 2003. Temperament characteristics of child and adolescent bipolar offspring. J. Affect. Disord. 77, 11–19.

Cohen, P., Velez, C.N., Brook, J.S., Smith, J., 1989. Mechanisms of the relation between perinatal problems, early childhood illness, and psychopathology in late childhood and adolescence. Child Dev. 60, 701–709.

Corkum, P., Moldofsky, H., Hogg-Johnson, S., Humphries, T., Tannock, R., 1999. Sleep problems in children with attention deficit-hyperactivity disorder: impact on subtype, comorbidity, and stimulant medication. J. Am. Acad. Child Adolesc. Psych. 38, 1285–1293.

Dahl, R., 1996. The regulation of sleep and arousal: development and psychopathology. Dev. Psychopathol. 8, 3–27.

Dahl, R., Pelham, W., Wierson, M., 1991. The role of sleep disturbances in attention deficit disorder symptoms: a case study. J. Pediatr. Psychol. 6, 229–239.

Davies, P.T., Cummings, E.M., 1994. Marital conflict and child adjustment: an emotional security hypothesis. Psychol. Bull. 116, 387–411.

Davies, P.T., Windle, M., 2001. Interpersonal discord and adolescent adjustment trajectories: the potentiating and protective role of intrapersonal attributes. Child Dev. 72, 1163–1178.

Gregory, A.M., O'Connor, T.G., 2002. Sleep problems in childhood: a longitudinal study of developmental change and association with behavioral problems. J. Am. Acad. Child Adolesc. Psych. 41, 964–971.

Gregory, A.M., Eley, T.C., O'Connor, T.G., Plomin, R., 2004. Etiologies of associations between childhood sleep and behavioral problems in a large twin sample. J. Am. Acad. Child Adolesc. Psych. 43 (6), 744–751.

Gregory, A.M., Caspi, A., Eley, T.C., Moffitt, T.E., O'Connor, T.G., Poulton, R., 2005. Prospective longitudinal associations between persistent sleep problems in childhood and anxiety and depression disorders in adulthood. J. Abnorm. Child Psychol. 33, 157–163.

Gruber, R., Sadeh, A., Raviv, A., 2000. Instability of sleep patterns in children with attention-deficit/hyperactivity disorder. J. Am. Acad. Child Adolesc. Psych. 39, 495–501.

Jacobsen, R.H., Lahey, B.B., Strauss, C.C., 1983. Correlates of depressed mood in normal children. J. Abnorm. Child Psychol. 11, 29–40.

Johnson, E.W., Chilcoat, H.D., Breslau, N., 2000. Trouble sleeping and anxiety/depression in childhood. Psychiatry Res. 94, 93–102.

Katon, W., Sullivan, M.D., 1990. Depression and chronic medical illness. J. Clin. Psychiatry 51, 3–11.

Leckman, J.F., Sholomskas, D., Thompson, D., Belanger, A., Weissman, M.M., 1982. Best estimate of lifetime psychiatric diagnosis: a methodological study. Arch. Gen. Psychiatry 39, 879–883.

Lerner, R.M., Palermo, M., Spiro, A., Nessleroade, J., 1982. Assessing the dimensions of temperament individuality across the life span: the Dimensions of Temperament Survey (DOTS). Child Dev. 53, 149–159.

Luby, J.L., Steiner, H., 1993. Concordance of parent–child temperament ratings in a clinical sample of adolescent girls. Child Psychiatry Hum. Dev. 23, 297–305.

Mannuzza, S., Fyer, A.J., Klein, D.F., Endicott, J., 1986. Schedule for affective disorders and schizophrenia-lifetime version modified for the study of anxiety disorders (SADS-LA): rationale and conceptual development. J. Psychiatr. Res. 20, 317–325.

Maziade, M., Cote, R., Boudreault, M., Thiverge, J., Boutin, P., 1986. Family correlates of temperament continuity and change across middle childhood. Am. J. Orthopsychiatr. 56, 195–203.

Mufson, L., Fendrich, M., Warner, V., 1990. The stability of temperament by child and mother reports over two years. J. Am. Acad. Child Adolesc. Psych. 29, 386–392.

Orvaschel, H., Puig-Antich, P., Chambers, W., Tabrizi, M.A., Johnson, R., 1982. Retrospective assessment of prepubertal major depression with Kiddie-SADS-E. J. Am. Acad. Child Psych. 21, 392–397.

Pine, D.S., Shaffer, D., Schonfeld, I.S., 1993. Persistent emotional disorder in children with neurological soft signs. J. Am. Acad. Child Adolesc. Psych. 32, 1229–1236.

Pine, D.S., Cohen, P., Gurley, D., Brook, J., Ma, Y., 1998. The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. Arch. Gen. Psychiatry 55, 56–64.

Porrino, L.J., Rapoport, J.L., Behar, D., Sceery, W., Ismond, D., Bunny, W.E., 1983. A naturalistic assessment of the motor activity of hyperactive boys. Arch. Gen. Psychiatry 40, 681–687.

Puig-Antich, P., 1982. The use of RDC criteria for major depressive disorder in children and adolescents. J. Am. Acad. Child Adolesc. Psych. 21, 291–293.

Rutter, M., Pickles, A., 1991. Person–environment interactions: concepts, mechanisms, and implications for data analysis. In: Wachs, T.D., Plomin, R. (Eds.), Conceptualization and Measurement of Organism–Environment Interaction. American Psychiatric Association, Washington, DC, pp. 105–136.

Ryan, N.D., Puig-Antich, J., Ambrosini, P., Rabinovich, H., Robinson, D., Nelson, B., Iyengar, S., Twomey, J., 1987. The clinical picture of major depression in children and adolescents. Arch. Gen. Psychiatry 44, 854–861.

Shaffer, D., Schonfeld, I.S., O'Connor, P.A., Stokman, C., Trautman, P., Shafer, S., Ng, S., 1985. Neurological soft signs. Their relationship to psychiatric disorder and intelligence in childhood and adolescence. Arch. Gen. Psychiatry 42, 342–351.

Small, A., Hibi, S., Feinberg, I., 1971. Effects of dextroamphetamine sulfate on EEG sleep patterns of hyperactive children. Arch. Gen. Psychiatry 25, 369–380.

Stoléru, S., Nottelmann, E.D., Belmont, B., Ronsaville, D., 1997. Sleep problems in children of affectively ill mothers. J. Child Psychol. Psychiatry 38, 831–841.

Teicher, M.H., Glod, C.A., Harper, D., Magnus, E., Brasher, C., Wren, F., Pahlavan, K., 1993. Locomotor activity in depressed children and adolescents: I. Circadian dysregulation. J. Am. Acad. Child Adolesc. Psych. 32, 760–769.

Tirosh, E., Sadeh, A., Munvez, R., Lavie, P., 1993. Effects of methylphenidate on sleep in children with attention-deficit hyperactivity disorder. Am. J. Dis. Child 147, 1313–1315.

Waters, B.G.H., Storm, V., 1985. Depression in pre-pubertal children. Aust. N. Z. J. Psychiatry 19, 6–17.

Weissman, M.M., Gammon, D., John, K., Merikangas, K.R., Warner, V., Prusoff, B.A., Sholomskas, D., 1987. Children of depressed parents: increased psychopathology and early onset of major depression. Arch. Gen. Psychiatry 44, 847–853.

Weissman, M.M., Warner, V., Wickramaratne, P., Moreau, D., Olfson, M., 1997. Offspring of depressed parents: ten years later. Arch. Gen. Psychiatry 54, 932–940.

Weissman, M.M., Wickramaratne, P., Nomura, Y., Warner, V., Verdeli, H., Pilowsky, D.J., Grillon, C., Bruder, G., 2005. Families at high and low risk for depression: a 3-generation study. Arch. Gen. Psychiatry 62, 29–36.

Wells, V.E., Deykin, E.Y., Klerman, G.L., 1985. Risk factors for depression in adolescence. Psychiatr. Dev. 3, 83–108.

Wickramaratne, P.J., Weissman, M.M., 1998. Onset of psychopathology in offspring by developmental phase and parental depression. J. Am. Acad. Child Adolesc. Psych. 37, 933–942.

Wolin, S.J., Bennett, L.A., Noonan, D.L., Teitelbaum, M.A., 1980. Disrupted family rituals: a factor in the intergenerational transmission of alcoholism. J. Stud. Alcohol 41, 199–214.

Yuen, K., Pelayo, R., Biederman, J., 1999. Sleep disorders and attention-deficit/hyperactivity disorder. JAMA 281, 794–797.