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Title: The association of mothers' and fathers' insomnia symptoms with school-aged children's sleep assessed by parent report and in-home sleep-electroencephalography

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Abstract

Objective: Sleep plays an essential role for children's well-being. Because children's sleep is associated with parental sleep patterns, it has to be considered in the family context. As a first aim of the present study, we test whether parental insomnia symptoms are related to children's in-home sleep-electroencephalography (EEG). Second, we examine the association between parental insomnia symptoms and maternal and paternal perception of children's sleep using actor-partner interdependence models.

Methods: 191 healthy children enrolled in public school (96 were formerly very preterm born children) and aged 7 – 12 years took part in the study. Children underwent in-home sleep-EEG, and parents reported children's sleep-related behavior by using the German version of the Children's Sleep Habits Questionnaire. Further, parents completed the Insomnia Severity Index to report their own insomnia symptoms.

Results: Maternal but not paternal insomnia symptoms were related to less children's EEG-derived total sleep time, more stage 2 sleep, less slow wave sleep, later sleep onset time, and later awakening time. Mothers' and fathers' own insomnia symptoms were related to their reports of children's bedtime resistance, sleep duration, sleep anxiety, night wakings, and/or daytime sleepiness. Moreover, maternal insomnia symptoms were associated with paternal reports of children's bedtime resistance, sleep anxiety, and sleep disordered breathing. The associations between parental insomnia symptoms and parents' perception of children's sleep could not be explained by children's objectively measured sleep.

Conclusions: Mothers' insomnia symptoms and children's objective sleep patterns are associated. Moreover, the parents' own insomnia symptoms might bias their perception of children's sleep-related behavior problems.

Keywords: maternal and paternal insomnia; school-aged children; in-home sleep electroencephalography; parent reports; Children's Sleep Habits Questionnaire; Actor-Partner Interdependence Model

Abbreviations: TST, total sleep time; WASO, wake after sleep onset; SE, sleep efficiency; SOL, sleep onset latency; SWS, slow wave sleep; REM, rapid eye movement; CSHQ-DE, the German version of the Children's Sleep Habits Questionnaire; PSG, polysomnography; EEG, electroencephalography; ISI, Insomnia Severity Index; APIM, Actor-Partner Interdependence Model.

1. Introduction

Sleep plays an essential role for children's daytime functioning including their well-being, emotion regulation, cognitive functioning, and academic performance [1-4]. According to self- and parent reports sleep problems are frequent: 30%-40% of school-aged children seem to suffer from sleep disturbances such as difficulties initiating and maintaining sleep, as well as excessive day-time sleepiness [5,6].

There is compelling evidence that children's sleep is associated with parental sleep patterns [7-10], particularly with maternal sleep [8,9,11-14]. The association was especially salient when parents' and children's sleep were measured with the same method, e.g. when both parental and children's sleep were reported by the parents [14] or measured with actigraphy [9] or sleep-electroencephalography (EEG) [8]. However, to date no study examined the association between parental insomnia symptoms and children's sleep measured by polysomnography, the gold-standard of sleep assessment. Similarly, evidence on the association between one parent's insomnia symptoms and the co-parent's perception of child sleep is missing.

Several possible mechanisms that could account for the relationship between parents' and children's sleep have been suggested. First, children may learn sleep habits from their parents, which could lead to high correspondence between parents' and children's sleep quality [15]. Second, both parents and children could for instance be affected by poor family functioning [16-19] as well as by environmental stress related to poor socio-economic status leading to poor sleep patterns [20]. Third, children may share genetic variation with their parents that predisposes for poor sleep [21]. Fourth, in younger children, sleep difficulties can also affect parental sleep [22,23].

A methodological issue that could account for the association between parental insomnia and parent reports of children's sleep difficulties in particular, is that parental insomnia may affect their perception of children's sleep without any real underlying sleep problem of the child [24]. This can be important because parents are often the first to perceive their children's sleep problems and to seek help. Aside from causing unnecessary costs, treatment for sleep disorders without a real underlying indication might be harmful and eventually even trigger children's sleep disturbances [25,26]. This might happen in a similar fashion as described by Harvey's cognitive model of insomnia [27] which posits that selective attention and monitoring of sleep difficulties play a crucial role in triggering and perpetuating sleep disturbances.

Moreover, parent reports are often used to assess children's sleep-related behavior and sleep problems in research, since parent reports are practical and inexpensive [28], although being error-prone and possibly biased due to the parents' own sleep difficulties [24,29]. Rönnlund et al. (2016) [24] for instance studied parents of children aged 2 to 6 years and measured children's sleep by actigraphy to identify to what degree parent reports were explained by children's actual sleep. Parents, who themselves suffered from poor sleep, more often reported sleep problems in their children, including disorders of initiating and maintaining sleep, disorders of sleep-wake transitions, and excessive daytime somnolence [24]. These associations could not be explained by objective (i.e. actigraphy) measures of children's sleep [24]. A possible explanation for this pattern of results is that parents who sleep poorly themselves show a negativity bias, such that they show increased attention towards and more often remember negative stimuli related to their child's sleep [24,30,31].

As a first aim of the present study, we tested whether maternal and paternal insomnia symptoms were related to children's sleep measured objectively with in-home sleep-EEG. Compared to laboratory-based polysomnography, in-home sleep-EEG assessment has the advantage that sleep is assessed in the ecological context where it normally occurs. We hypothesized that children show worse sleep patterns including shorter sleep duration and decreased sleep continuity when their mothers and fathers had increased insomnia symptoms. As a second aim of the present study we examined the association of parents' insomnia symptoms and their perception of their children's sleep. In order to account for the degree to which this relationship is explained by the children's actual sleep, we controlled children's objectively measured sleep (i.e., the EEG sleep-indices) in an additional step. We hypothesized that parents with more insomnia symptoms also perceive more sleep-related behavior problems in their children. Because the present study aims to extend our understanding of parental perception of children's sleep, the interdependence of maternal and paternal reports of children's sleep was examined using the Actor-Partner Interdependence Model (APIM) [32]. Maternal and paternal perception of their children's sleep could be influenced by their own as well as by their partners' sleep problems. The APIM approach allows to shed light into the mutual interdependence of

maternal and paternal perception of their children's sleep by disentangling so-called actor and partner effects; in the present context, actor effects denote associations between one parent's insomnia symptoms and his/her perception of the child's sleep-related behavior problems (Figure 1, paths a), whereas partner effects reflect the associations between one parent's insomnia symptoms and the other parent's perception of the child's sleep-related behavior problems (Figure 1, paths b).

(Insert Figure 1 about here)

2. Material and methods

2.1. Study population and procedure

The data for the present study derives from the second wave (May 2013 – September 2014) of the Basel Study of Preterm Children (BSPC). Recruitment procedures have been described elsewhere in detail (see e.g. Lemola et al., 2015 [33] and Perkinson-Gloor et al., 2015 [34] for reports on the first study wave and Maurer et al., 2016 [35] for a report on the second study wave). In total, the second wave of the study included 191 healthy school-aged children (age: $M = 9.58$ years, $SD = 1.47$; range: 7.17 to 12.92 years; 109 (57.1%) were boys; see Table 1). Children underwent one night of in-home sleep-EEG, and parents completed the German version of the Children's Sleep Habits Questionnaire (CSHQ-DE; Schwerdtle & Hautzinger, 2010 [36]) to rate children's sleep-related behavior and the Insomnia Severity Index (ISI; Morin, 1993 [37,38]) to rate their own insomnia symptoms. The total sample included 96 (50.3%) children born very preterm (<32 weeks of gestation) and 95 (49.7%) age and sex matched children born at term. Since children born very preterm and full-term differ in various sleep indices [34,35] the statistical analyses were controlled for preterm birth status. All children attended compulsory primary or secondary school in Switzerland. The study was approved by the Ethics Committee of Basel, assent was obtained from the children and written informed consent was obtained from the parents for each participant.

Post hoc power analysis using G*Power was conducted to evaluate the statistical power given the sample size of the study [39]. Post hoc calculations regarding correlations revealed a power of .80 to detect small to medium effect sizes (i.e., $r = 0.20$) at a 0.05 alpha level (two-sided; based on the total sample size). Therefore, the present study was deemed sufficiently powered to detect effect sizes of $r = 0.20$ [40].

2.2. Sleep assessment

Sleep was assessed using the Somté PSG (Compumedics, Singen, Germany), a portable sleep-monitoring device during a single night on a regular school day at the children's home. Sleep-EEG signals C3/A2 and C4/A1, right and left electrooculogram and bipolar submental electromyogram were obtained. The sleep-EEG reports were analyzed by two experienced raters according to standard procedures [41]. The following sleep indices were evaluated: Total sleep time (TST; time in bed minus time spent awake in hours). Sleep continuity: Sleep efficiency (SE; $TST/\text{time in bed} \times 100$), sleep onset latency (SOL; min), and wake after sleep onset (WASO; the amount of time awake from the initial sleep onset to the last awakening; min). Sleep architecture (%): Stage 1 sleep, stage 2 sleep, slow wave sleep (SWS), rapid-eye-movement (REM) sleep, and REM latency (min). In addition sleep onset time and awakening time are reported. Sleep-EEG data were available for 146 children.

To evaluate if there was a first night effect due to sleep-EEG assessment, children also reported whether they had a quiet night of sleep for the sleep-EEG night as well as for the following six nights. While for the sleep-EEG night 89.0% reported that they had a quiet night, this figure was on average 94.2% for the following six nights.

2.3. Parent reports of children's sleep-related behavior

Parents completed the CSHQ-DE [36], a retrospective questionnaire to examine sleep-related behavior in children regarding a "typical" recent week. In most cases parents completed the questionnaire during the afternoon or early evening before sleep-EEG assessment was conducted. Items were answered on a three-point scale with the response options *usually* (5-7/week), *sometimes* (2-4/week), or *rarely* (0-1/week). Eight scales including one to eight items and reflecting the following sleep domains were calculated: Bedtime Resistance, Sleep Onset Delay, Sleep Duration problems, Sleep Anxiety, Night Wakings, Parasomnias, Sleep-Disordered Breathing, and Daytime Sleepiness. In addition an overall Sleep Disturbance Score was calculated. Higher scores indicate greater sleep-

related behavior problems. Table 2 shows Cronbach's alpha for maternal and paternal reports of the CSHQ-DE scales, intraclass correlations (*ICC*) for maternal and paternal reports as an index of interrater agreement, and *t*-values for mean comparisons between maternal and paternal reports. Cronbach's alpha ranged from $\alpha = .41$ to $\alpha = .72$. Interrater agreements were fair to excellent for all scales and ranged from $ICC = .54$ to $ICC = .88$. On average, maternal reports of children's Sleep Duration problems and Daytime Sleepiness were higher than paternal reports. For 185 children maternal reports and for 154 children paternal reports of sleep-related behavior were available.

2.4. Assessment of parental insomnia symptoms

Parents reported their current (i.e. last 2 weeks) insomnia symptoms on the German version of the ISI [37,38], a questionnaire consisting of 7 items, rated on a five-point Likert scale. A higher ISI score indicates greater insomnia severity. Table 2 also shows Cronbach's alpha, intraclass correlation (*ICC*), and *t*-value for the ISI scale. For 184 children the mothers' reports and for 153 children the fathers' reports of their own insomnia symptoms were available. For 142 children both maternal reports of insomnia symptoms and children's sleep-EEG indices were available. For 123 children both paternal reports of insomnia symptoms and children's sleep-EEG indices were available.

2.5. Statistical analysis

Multiple regression analyses were conducted with parental insomnia symptoms as independent variable and children sleep-EEG as dependent variable, to examine whether maternal and paternal insomnia symptoms are related to children's sleep measured objectively with in-home sleep-EEG (hypothesis 1). All analyses were controlled for children's age, sex, and prematurity status if not stated otherwise. Multiple regression analyses were performed with IBM® SPSS® Statistics 22 (IBM Corporation, Armonk NY, USA) for Apple Mac® and standardized betas, adjusted *t* values, and adjusted *p* values (two-tailed) are reported. To test hypothesis 2, that is whether parents with more insomnia symptoms reported more sleep-related behavior problems in their children than parents with less insomnia symptoms, we computed APIMs with the lavaan package in R for each CSHQ-DE scale. APIMs were controlled for children's age, sex, and prematurity status. Further, we calculated APIMs that were additionally controlled for sleep-EEG patterns (TST and SE) to examine the degree to which effects of parental insomnia can be explained by children's actual sleep. For APIM analyses, unstandardized regression coefficients and adjusted *p* values are reported. By z-standardization of the scales before the analyses, interpretation of the unstandardized regression coefficients is facilitated. The APIM analyses were conducted with the full sample of 191 parental couples. Full-information maximum likelihood estimation was used to deal with missing values, which is considered a more reliable procedure compared to other more conventional methods [42].

3. Results

3.1. Descriptive statistics and preliminary analyses

Table 1 shows descriptive statistics of background variables and children's sleep-EEG indices, while Table 2 shows descriptive statistics for parent-reported children's sleep-related behavior (CSHQ-DE scales), and parental insomnia symptoms (ISI scale).

Child age was negatively related to EEG-derived TST ($r = -.48, p < .001$) and positively related to sleep onset time ($r = .40, p < .001$). Regarding maternal-reported sleep-related behavior, child age was negatively related to Bedtime Resistance ($r = -.17, p = .019$), Sleep Anxiety ($r = -.21, p = .005$), Night Wakings ($r = -.17, p = .024$), and Sleep Disturbance Score ($r = -.16, p = .030$). Regarding paternal-reported sleep-related behavior, child age was negatively related to Sleep Anxiety ($r = -.18, p = .025$) and positively related to Sleep Duration problems ($r = .19, p = .021$). Girls showed shorter EEG-derived REM latency ($t(144) = 2.35, p = .020$), earlier awakening time ($t(144) = 3.06, p = .003$), and more maternal and paternal reported Sleep Onset Delay than boys ($t(183) = -2.14, p = .034$; $t(150) = -2.49, p = .014$). Very preterm children showed earlier sleep onset times compared to term-born children, while there were no further significant group differences regarding sleep-EEG indices (for a detailed report see Maurer et al., 2016 [35]). Mothers of very preterm born children reported less Sleep Duration problems ($t(183) = 2.72, p = .007$) and more Sleep Anxiety ($t(183) = -2.95, p = .011$) than mothers of full-term children. The remaining maternal-reported CSHQ-scales and paternal-reported sleep-related behaviors did not differ between children born very preterm and full-term.

(Insert Table 1 and 2 about here)

3.2. Associations of parental insomnia symptoms with children's in-home sleep-EEG

Table 3 shows the association between parental insomnia symptoms and children's sleep-EEG indices. Increased maternal insomnia symptoms were associated with less children's sleep-EEG TST ($\beta = -.17, t = -2.20, p = .029$), which is in line with our first hypothesis. There was no significant association between maternal insomnia and children's sleep-EEG continuity (SE, SOL, WASO). Moreover, increased maternal insomnia symptoms were associated with more stage 2 sleep ($\beta = .21, t = 2.50, p = .014$), less SWS ($\beta = -.17, t = -2.05, p = .043$), later sleep onset time ($\beta = .25, t = 3.34, p = .001$), and later awakening time ($\beta = .20, t = 2.44, p = .016$) in children. No significant association between paternal insomnia symptoms and children's sleep-EEG was found.

(Insert Table 3 about here)

3.3. Associations of parental insomnia symptoms with parental perception of children's sleep-related behavior (APIM-Analyses)

Table 4 shows the association between parental insomnia symptoms and children's sleep-related behavior as reported by the parents. Significant actor and partner effects emerged. For mothers, their own insomnia levels predicted their perception of children's Bedtime Resistance ($b = .28, p < .001$), Sleep Anxiety ($b = .23, p = .001$), Night Wakings ($b = .18, p = .015$), and overall Sleep Disturbance Score ($b = .23, p = .001$) (actor effects). Moreover, maternal insomnia symptoms were positively linked to paternal perception of children's Bedtime Resistance ($b = .33, p < .001$), Sleep Anxiety ($b = .21, p = .009$), Sleep Disordered Breathing ($b = .19, p = .004$), and overall Sleep Disturbance Score ($b = .19, p = .006$) (partner effects). All these associations remained significant when controlling children's sleep-EEG indices and were thus not explained by objective sleep measures.

For fathers, insomnia symptoms were positively associated with their perception of children's Sleep Duration problems ($b = .29, p < .001$), Daytime Sleepiness ($b = .18, p = .021$), and the overall Sleep Disturbance Score ($b = .17, p = .021$) (actor effects). These associations also remained significant when controlling children's sleep-EEG indices. However, paternal insomnia symptoms were unrelated to maternal perception of children's sleep-related behavior (partner effects) in the first step of the analyses (i.e. not controlling children's sleep-EEG indices). A significant association between paternal insomnia symptoms and mothers' perception of children's Daytime Sleepiness emerged only in the model that additionally controlled the children's sleep-EEG indices ($b = .17, p = .046$).

(Insert Table 4 about here)

4. Discussion

This is the first study that reports associations between parental insomnia symptoms and children's sleep with in-home sleep-EEG. Further, it analyzes for the first time actor and partner effects regarding the association between mothers' and fathers' insomnia symptoms and their perception of children's sleep. The key findings were that maternal but not paternal insomnia was related to children's EEG-derived sleep. Moreover, maternal insomnia symptoms were associated with both maternal and paternal reports of children's bedtime resistance and sleep anxiety. Paternal insomnia was related to the father's own reports of children's sleep duration problems and daytime sleepiness. These associations were not explained by the children's sleep-EEG indices.

4.1. Associations of parental insomnia symptoms with children's in-home sleep-EEG

In accordance to our first hypothesis, maternal insomnia was related to children's sleep duration measured by EEG. In addition, maternal insomnia was associated with children's sleep-EEG architecture including more stage 2 sleep and less SWS and sleep timing including later sleep onset and awakening time. However, maternal insomnia was unrelated with children's sleep continuity measured by sleep-EEG. Paternal insomnia and children's sleep-EEG indices were unrelated. These findings are in line with prior studies reporting associations between maternal and children's sleep patterns as measured both by sleep-EEG [8] or by actigraphy [9] but not between paternal and

children's sleep patterns [9]. A possible explanation for a stronger association between maternal insomnia symptoms and children's objective sleep patterns might be that mothers in Switzerland tend to spend more time with their children than fathers [43,44]. Maternal and children's sleep may therefore also influence each other more strongly.

4.2. Associations of parental insomnia symptoms with parental perception of children's sleep-related behavior

Consistent with our second hypothesis and the results from Rönnlund et al. (2016) [24] we found that parents with more insomnia symptoms also reported more sleep-related behavior problems for their children. Mothers with higher levels of insomnia symptoms perceived more bedtime resistance, sleep anxiety, and night wakings in their children (maternal actor effects for CSHQ-DE scales). In addition to these actor effects, maternal insomnia problems were also associated with paternal perception of bedtime resistance, sleep anxiety, and sleep disordered breathing (partner effects). In turn, fathers with higher levels of insomnia symptoms perceived more sleep duration problems and daytime sleepiness (actor effects). However, paternal insomnia symptoms were not associated with maternal perception of children's sleep duration problems and daytime sleepiness (partner effects). The only partner effect, which was between paternal insomnia symptoms and maternal perception of child's daytime sleepiness, emerged after controlling children's sleep-EEG.

Possible explanations for the association of maternal insomnia with children's bedtime resistance and sleep anxiety include interactions between children's and parental sleep related behavior. Children's sleep difficulties and subsequent behavior could prevent parents from a good night's sleep or vice versa [10,11]. Further, it is possible that some of the families' homes provided generally unfavorable sleep environments, e.g. due to environmental noise [20]. Moreover, shared genetic risk could account for vulnerabilities to sleep difficulties in parents and children [21]. A possible explanation for a greater number of partner effects related to maternal insomnia symptoms might again be that mothers spend more time with their children than fathers and that there might be more mutual influences [43,44]. On the other hand it is also possible that mothers report their perception of children's sleep difficulties to their partners more often than vice versa. These reports, however, can already be influenced by maternal sleep problems.

Interestingly, none of the above-mentioned associations between parental insomnia symptoms and children's sleep-related behavior problems, as reported by the parents, could be explained by objective measures of children's sleep. Therefore, one alternative explanation of the association between parental sleep difficulties and their reports of children's sleep is over-reporting of children's sleep problems – parents with insomnia symptoms may exhibit an attention bias towards negative sleep-related stimuli including their children's sleep problems [30,31]. This interpretation is generally in line with a prior study reporting an association between poor parental sleep and parent ratings of their 2-6 year old children's sleep controlling children's objective sleep measured by actigraphs [24]. In opposition to this interpretation, however, it can be argued that one night of in-home sleep-EEG may not accurately represent children's common sleep behavior. Nevertheless, a prior study using in-home sleep-EEG in children showed relatively high stability across 18 months particularly regarding EEG-derived sleep duration and sleep architecture [45]. In sum, it is possible that parental insomnia leads to over-reporting regarding their children's sleep problems. Moreover, it is possible that parental selective attention and monitoring of children's sleep difficulties may in some cases even trigger children's sleep disturbances in a similar fashion as described by Harvey's cognitive model of insomnia [27]. From a clinical perspective it therefore appears important to carefully diagnose children's sleep difficulties taking the family context into account.

4.3. Strengths and Limitations

The major strength of our study is that children's sleep was measured by in-home sleep-EEG, which increases the ecological validity of sleep assessment compared to laboratory based PSG. In addition, information from both parents was available and actor and partner effects regarding the association between parental insomnia and parental perception of children's sleep could be examined.

However, the current study is not without limitations. First, in-home sleep-EEG was only conducted during one single night, which may decrease reliability compared to assessment across multiple nights. Relatedly, there was no EEG-adaptation night and therefore it is not possible to rule out first-night effects. By contrast, parents referred to a longer period of time when rating their

children's sleep (i.e., 1 week) and their own insomnia symptoms (i.e., 2 weeks). Second, the study sample included very preterm children and age and sex matched controls, therefore, the findings may not necessarily apply to the general population. Third, parental insomnia symptoms were assessed with self-report questionnaires, which do not necessarily reflect clinically relevant insomnia.

5. Conclusions

The present study implies that it is important to consider children's sleep in the family context. In particular, maternal insomnia appears to be associated to children's objectively measured sleep duration, sleep architecture (stage 2 sleep and SWS), and sleep onset and awakening time. These associations may reflect a mutual interdependence between maternal and children's sleep. Parental insomnia symptoms were also associated with parental perception of children's sleep related behavior problems, in particular regarding bedtime resistance, sleep duration problems, sleep anxiety, night wakings, and daytime sleepiness. On the one hand, these associations may also be seen in the light of the mutual interdependence of parental and children's sleep. On the other hand, these associations may at least partly reflect parental overrating of children's sleep-related behavior problems due to a negativity bias in parents, who suffer from insomnia symptoms themselves, as the associations were not explained by objective measures of children's sleep. Eventually, it is possible that selective attention and increased monitoring of children's sleep alongside unindicated treatment attempts may even pose a risk for children's sleep.

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Conflicts of interest: none

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Table 1		
Descriptive statistics for background variables and sleep-EEG indices.		
	M/N	(SD/%)
Background variables		
Age, years ¹	9.58	(1.47)
Sex, male	109	(57.10)
Prematurity status, born very preterm	96	(50.30)
Gestational age, weeks	34.50	(5.21)
Sleep-EEG indices ²		
Total sleep time (h)	8.93	(0.69)
Sleep efficiency (%)	93.40	(2.85)
Sleep onset latency (min)	18.50	(8.59)
WASO (min)	18.77	(14.18)
Stage 1 sleep (%)	3.51	(2.30)
Stage 2 sleep (%)	47.53	(4.98)
Slow wave sleep (%)	21.46	(4.81)
REM sleep (%)	24.96	(3.86)
REM latency (min)	111.29	(42.20)
Sleep onset time	21:19:42	(0:39:32)
Awakening time	6:34:14	(0:23:06)

Note: EEG = electroencephalography; WASO = wake after sleep onset; REM = rapid eye movement.

¹ refers to 191 children, the total sample size.

² refers to 146 children who underwent in-home sleep-EEG.

Table 2

Descriptive statistics for parent reports of children's sleep-related behavior and the parents' own insomnia symptoms.

	Maternal report			Paternal report			intra-class correlations	
	M	(SD)	Cronbach's α	M	(SD)	Cronbach's α	<i>ICC</i>	<i>t-value</i>
Children's sleep patterns (CSHQ-DE) ¹								
Bedtime Resistance	6.81	(1.46)	.61	6.79	(1.33)	.55	.85	-1.27
Sleep Onset Delay	1.36	(0.62)	n/a	1.41	(0.68)	n/a	.75	-.29
Sleep Duration problems	3.66	(1.08)	.59	3.43	(0.87)	.66	.54	2.90**
Sleep Anxiety	4.90	(1.44)	.64	4.75	(1.30)	.62	.80	.67
Night Wakings	3.53	(0.92)	.57	3.56	(0.84)	.41	.77	-.69
Sleep Disordered Breathing	3.35	(0.87)	.72	3.28	(0.75)	.69	.88	1.51
Parasomnias	8.43	(1.55)	.55	7.98	(1.22)	.44	.72	3.54
Daytime Sleepiness	12.54	(2.39)	.59	12.19	(2.37)	.57	.73	2.18*
Sleep Disturbance Score	42.17	(5.02)	.71	41.09	(4.77)	.70	.82	3.27
Parental insomnia problems (ISI) ²	14.07	(4.23)	.74	13.92	(3.66)	.67	.08	0.62

Note: CSHQ-DE = the German version of the Children's Sleep Habits Questionnaire; ISI = Insomnia Severity Index.

¹ refers to 185 children with maternal reports and 154 children with paternal reports.

² refers to 184 children with maternal reports and 153 children with paternal reports.

* $p < .05$, ** $p < .01$ (two-tailed).

Table 3

Association between parental insomnia symptoms and children's sleep-EEG indices.

	Maternal insomnia problems ¹		Paternal insomnia problems ²	
	β	t	β	t
Total sleep time (h)	-.17*	-2.20	.07	.84
Sleep efficiency	-.12	-1.39	.18	1.96
Sleep onset latency	.06	.70	-.17	-1.85
WASO (min)	.08	.90	-.10	-1.07
Stage 1 sleep (%)	-.03	-.32	.03	.34
Stage 2 sleep (%)	.21*	2.50	.03	.29
Slow wave sleep (%)	-.17*	-2.05	-.05	-.51
REM sleep (%)	-.03	-.36	.02	.20
REM latency (min)	.01	.17	.09	1.01
Sleep onset time	.25**	3.34	.03	.39
Awakening time	.20*	2.44	.12	1.29

Note: EEG = electroencephalography; WASO = wake after sleep onset; REM = rapid eye movement.

Data adjusted for children's age, sex, and prematurity status.

¹ refers to 142 children with both sleep-EEG and maternal reports of insomnia symptoms.

² refers to 123 children with both sleep-EEG and paternal reports of insomnia symptoms.

* $p < .05$, ** $p < .01$ (two-tailed).

Table 4

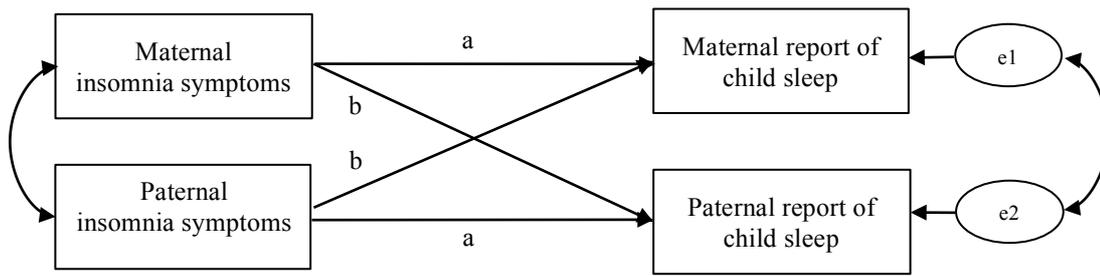
Results of actor-partner interdependence models (APIM) for maternal and paternal insomnia predicting maternal and paternal reports of sleep-related behavior of their children.

	Maternal report of child sleep		Paternal report of child sleep	
	Actor effects (Maternal insomnia as predictor)	Partner effects (Paternal insomnia as predictor)	Actor effects (Paternal insomnia as predictor)	Partner effects (Maternal insomnia as predictor)
Bedtime Resistance	.28***/.28***	.06/.05	.09/.07	.33***/.32***
Sleep Onset Delay	.07/.02	-.13/-.06	-.13/-.08	.13/.09
Sleep Duration problems	.12/.10	.05/.03	.29***/.29***	.03/.01
Sleep Anxiety	.23**/.24**	.02/.01	.00/-.03	.21**/.21**
Night Wakings	.18*/.17*	.03/.02	.08/.09	.15/.14
Sleep Disordered Breathing	.13/.12	-.14/-.17	.03/.06	.19**/.19*
Parasomnias	.07/.06	.03/.03	.12/.10	.07/.08
Daytime Sleepiness	.09/.08	.15/.17*	.18*/.19*	.05/.06
Sleep Disturbance Score	.23**/.23**	.05/.08	.17*/.19*	.19**/.22**

Note: Effects are unstandardized estimates. Due to the z-standardization of scales prior to analyses they may be interpreted similar to standardized β -regression coefficients. Estimates before the dash (/) are controlled for child's age, sex, and prematurity status. Estimates after the dash (/) are additionally controlled for sleep-EEG patterns (total sleep time and sleep efficiency). Partner effects regarding maternal reports are the effects of paternal insomnia on the mothers' perception of children's sleep-related behavior. Partner effects regarding paternal reports are the effects of maternal insomnia reports on fathers' perception of children's sleep-related behavior.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Fig. 1.



a = actor effect: effect of one parent's insomnia symptoms on his/her own report of the child's sleep; b = partner effect: effect of one parent's insomnia symptoms on the other parent's report of the child's sleep.

Fig. 1. Actor-Partner Interdependence Model with maternal and paternal insomnia symptoms predicting maternal and paternal perception of children's sleep-related behavior problems.