Sleep problems in preschoolers and maternal depressive symptoms: An evaluation of mother- and child-driven effects

Running head: Child sleep problems and maternal depression

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Abstract

Child sleep problems are associated with maternal depressive symptoms. It is unclear to what extent the association is due to direct effects or common risk factors for mother and child. Direct effects could represent child-driven processes, where child sleep problems influence maternal depressive symptoms; or mother-driven processes, where maternal depressive symptoms influence child sleep problems. Common factors could be shared genetic and familial environmental risk. Child- and mother-driven processes are direct in the sense that they are not due to common factors. However, such processes could be mediated by a range of unmeasured variables. By using an autoregressive fixed effects model on a community based longitudinal sample comprising 956 families assessed at 1.5, 2.5, and 4 years of age, we estimated the direction of effect between, and common causes of, child sleep problems and maternal depressive symptoms. We were able to explain the association between child sleep problems and maternal depressive symptoms by both child-driven and mother-driven processes. The effect of child-driven processes was significantly larger than the effect of mother-driven processes. The clinical implication of the study is that treatment of child sleep problems will have considerable effect on maternal depressive symptoms. Furthermore, our model supports that treatment of current child sleep problems will have a direct effect on future sleep problems, and also an indirect effect on future maternal depressive symptoms. We recommend that health professionals should assess child sleep problems in mothers at risk for depression.

Keywords: Child sleep; maternal depressive symptoms; Mother Child Relations; Early Childhood Development; Longitudinal studies; Driven-effects
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57 Introduction

58 Sleep problems are prevalent throughout childhood (Byars, Yolton, Rausch, Lanphear, & Beebe, 2012; Owens, 2008; Wake et al., 2006). Although the majority of toddlers with sleep problems outgrow their problems (Hysing et al., 2014; Wake et al., 2006), many children experience that their sleep problems, such as frequent nocturnal awakenings and difficulties in settling at night, become chronic and endure well into later childhood (Byars et al., 2012; Hysing et al., 2014; Lam, Hiscock, & Wake, 2003; Wake et al., 2006).

59 The etiology of sleep problems in young children is multifactorial and both genetic and environmental factors are important (Gregory & O'Connor, 2002). Maternal depression has repeatedly been shown to be associated with sleep problems in their offspring (Martin, Hiscock, Hardy, Davey, & Wake, 2007; Zuckerman, Stevenson, & Bailey, 1987). From pregnancy and throughout the preschool years, the peak level of maternal depressive symptoms is when the child is between 1.5 and 3 years (Ystrom et al., 2014). Maternal depression is related to parental behavior, and research suggests that maternal depression may affect child development on several domains, including cognitive and language development (Grace, Evindar, & Stewart, 2003; Sohr-Preston & Scaramella, 2006), mental health problems (Goodman et al., 2011; Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002), suboptimal diet (Ystrom, 2012; Ystrom, Barker, & Vollrath, 2012), and a number of different social, emotional and behavior problems (Goodman et al., 2011; Grace et al., 2003; Nilsen, Gustavson, Røysamb, Kjeldsen, & Karevold, 2013). There is a limited number of community and population-based studies examining the association between maternal depression and sleep problems in younger children. Significant associations between maternal depression and sleep problems have been found in both infants (Bayer, Hiscock, Hampton, & Wake, 2007; Goldberg et al., 2013), as well as in older children (e.g., toddlers and preschool aged children) (Gelman & King, 2001; Martin et al., 2007; Zuckerman et al., 1987).
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of effect in these studies is less clear, and either child, mother, or common factors may be important. However, using a large sibling study, Ystrom et al. (2017) recently found support for effects going from mother to child, but not from child to mother in 1.5 year old children.

One perspective is the ”child-driven” model, which suggests that sleep problems in children contribute to maternal depressive symptoms. Most of the literature supporting this view is based upon the notion that parents of children with sleep problems sleep less than other parents, which in turn may lead to parental stress, fatigue and symptoms of depression (Lam et al., 2003; Meltzer & Mindell, 2007; Moore, Gordon, & McLean, 2012). According to this view, helping parents with their children’s sleep problems, e.g. a guided sleep intervention program, should not only lead to improved sleep for children and their parents, but also to an improvement in the parents’ psychological well-being (e.g. less maternal depressive symptoms). There are several intervention studies on infants, toddlers and preschoolers supporting this assumption (Hiscock, Bayer, Hampton, Ukoumunne, & Wake, 2008; Lam et al., 2003).

A second perspective is the “mother-driven” model of children’s sleep problems and maternal depressive symptoms suggesting that maternal depressive symptoms contribute to children’s sleep problems (Ystrom et al., 2017). This contribution could be mediated through a range of putative maternal behaviors (Gelman & King, 2001; Teti & Crosby, 2012; Warren, Howe, Simmens, & Dahl, 2006). For instance, depressive mothers tend to spend less time in positive interactions with their children, they report more negative perceptions of their children’s behaviors, and tend to be more hostile towards their children and make more negative appraisals of their children’s behaviors than non-depressive mothers (Cornish et al., 2006; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Such interactions and appraisals could influence maternal bedtime and nighttime behavior, leading to poorer self-soothing skills and
risk for sleep problems in the child (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 1991; Mindell, Telofski, Wiegand, & Kurtz, 2009; Teti, Kim, Mayer, & Countermine, 2010). A third perspective on possible explanatory factors of maternal depressive symptoms and child sleep problems is through common factors. First, biological mechanisms, such as common genetic risk factors for psychopathology and deregulation in both the child and in their mothers, have been supported by several studies (i.e. a gene-environment correlation) (Gjerde et al., 2017; McAdams et al., 2014; Scarr & McCartney, 1983). Another biological mechanism could be elevated hormonal levels in mothers with depressive symptoms, which may affect the fetus and continue to affect the child later on. Studies have found higher levels of pregnancy and perinatal cortisol and norepinephrine in depressed mothers, and suggested this as a possible explanation of the association between pre- and perinatal maternal depression and infant night waking very early in life (Azak, Murison, Wentzel-Larsen, Smith, & Gunnar, 2013; Field, 2011; Field et al., 2007). Second, some studies indicate that social and contextual stressors, including high parenting stress, stressful life events, family conflict and low family income, as well as cultural aspects, could account for the association between sleep problems and maternal mental health (El-Sheikh, Kelly, Bagley, & Wetter, 2012; Gelman & King, 2001; Goldberg et al., 2013). Finally, individual differences in the child (i.e., temperament factors) could also account for the association between children´s sleep problems and maternal depressive symptoms (Jimmerson, 1991; Owens-Stively et al., 1997). This would constitute evocative processes also known as active gene-environment correlations (i.e., a heritable phenotype in the child influences the parent) (Narusyte et al., 2008; Scarr & McCartney, 1983).

To date there are several studies advocating both child-driven and mother-driven processes as the prime mechanism for the association between maternal symptoms of depression and child sleeping problems, and a lack of studies advocating common factors as
the prime mechanism. To the best of our knowledge, no studies have integrated all three perspectives in a single model and tested the significance of each mechanism. The rationale of the co-twin control design can be applied to longitudinal data by use of the fixed-effects regression model (Boden, Fergusson, & Horwood, 2010; Hamaker & Wichers, 2017). Such analyses are indicative of the direction of effect, and both child-driven and mother-driven mechanisms could both be active, leading to reciprocal effects in a feedback loop. Structural equation models provide means to address this issue by applying a statistical model to the data that allows reciprocal effects and select the model best fitted to the data. As of today, no studies have formally tested the comparative fit of child-driven and mother-driven processes for child sleep problems and maternal depressive symptoms using longitudinal panel data.

By applying an autoregressive fixed effects model to longitudinal data from a population-based study, we aimed to estimate to what extent the association between maternal depressive symptoms and child sleep problems could be attributed to: 1) child-driven effects, where child sleep problems causes maternal depression; 2) mother-driven effects, where maternal depression causes child sleep problems; or, 3) common factors to maternal depression and child sleep problems.

Method

Sample and Procedure

In this study, we used data from the Tracking Opportunities and Problems Study (TOPP)—a prospective population-based longitudinal study focusing on the mental health of children and their parents. More than 95% of Norwegian families with children attend the public health services, which include 8–12 health screenings during the first 4 years of the child’s life. All families from 19 geographic health care areas that visited a child health clinic in 1993 for the scheduled 18-month (Time 1 [t1]) vaccination visit were invited to complete a
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questionnaire. Of the 1,081 eligible families, 939 (87%) participated at t1. The parents who
participated at t1 received a similar questionnaire when the children were 2.5 years of age
(Time 2 [t2]: n=781), and 4 years of age (Time 3 [t3]: n=750). At t2, additional 24 families
had moved to the area and were invited to join the study. The current sample comprised
participants having valid data at one or more of the three time points (n=956). The
questionnaires were administered by the health care workers (Mathiesen, Tambs, & Dalgard,
1999). All participants signed informed consent forms emphasizing the confidentiality of the
participants, and the right to withdraw from the study at any point. The Regional Committee
for Medical and Health Research Ethics, South East, approved study 2013/863
“Intergenerational Risk for Common Mental Disorders”.

Within the 19 health care areas 28% of the families lived in large cities, 55% lived in
densely populated areas, and 17% lived in rural areas. Maternal age ranged from 19 to 46
years at t1, with a mean of 30 years (SD=4.7). Data from the child health clinics showed that
non-respondents at t1 did not differ from respondents with respect to maternal age, education,
employment status, number of children, or marital status (Mathiesen et al., 1999). Additional
logistic regression analyses were conducted to examine differences between responders
versus non-responders at t3. Mothers responding at t3 were less likely to have boys compared
to girls (odds ratio (OR) = 0.63; p < .01) and were to a greater extent employed at t1 (OR =
1.58; p < 0.01) as compared to non-responders. There were no significant differences
between responders and non-responders in terms of educational level, marital status, number
of children, child sleep problems, or symptoms of depression.

Measures

Indicators of child sleep problems.
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Sleep problems were measured using four items, one item on total sleep time and three items from the sleep problems scale in the Behavioral Checklist (BCL) (Mathiesen & Sanson, 2000; Richman, 1977). The BCL consists of 19 items covering 12 behavioral categories (i.e. eating, sleeping, soiling, dependency and attention seeking, relationships with siblings and peers, activity, concentration, control problems, tempers, mood, worries, and fears). We present the content and response categories of the BCL sleep items in table 1. Factor analysis of the 19 BCL items completed by 1,047 parents of 3-year-old British children identified sleeping problems as a distinct factor (Sonuga-Barke, Thompson, Stevenson, & Viney, 1997). We combined the four items using confirmatory factor analysis (CFA) for ordinal data, also known as a graded response model within the framework of item response theory (Asparouhov & Muthén, 2016; Samejima, 1969).

Maternal symptoms of depression.

Maternal symptoms of depression were measured by the 25-item version of the Hopkins Symptom Check List (Hesbacher, Rickels, Morris, Newman, & Rosenfeld, 1980). The mothers rated how often they had experienced symptoms the last week. The reliability of the Hopkins Symptom Check List has earlier been well established in a Norwegian sample (Tambs & Moum, 1993). Two items—“thoughts of ending your life” and “loss of sexual interest or pleasure”—were excluded from the Norwegian questionnaire because some participants in the pilot-project perceived them as offensive (Mathiesen et al., 1999). We used the overall mean of the 23 items, each rated on a 4-point scale (“Not at all”, “A little”, “Quite a bit”, and “Extremely”). Cronbach’s alphas for maternal symptoms of depression at t1, t2 and t3 were .90, .89, and .90, respectively.

Statistics
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First, we tested to what extent the cross-time covariance structure of maternal depression and child sleep problems, respectively, could be explained by a) a time-invariant fixed factor, b) an autoregressive structure, or c) both (figure 1). After selecting the best fitting cross-time models for maternal depression and child sleep problems, respectively, we included them into the bivariate autoregressive fixed effect model (figure 2). The variance of the time-invariant fixed factors (ψ), which is equivalent to a random intercept in a mixture model of longitudinal data, is an estimate of the percentage of variance in the observations that is stable. The β matrix denotes the regression paths between study variables. These parameters represent direct effect between study variables. Beta effects across time are autoregressive effects, while effects within time are reciprocal effects between study variables. The θ matrix denotes the residual variance/covariance of the observed variables in question. These represent factors uncorrelated with time-invariant factors and factors present at previous time points (i.e. emerging factors). These variables are allowed to covariate within time to represent common factors for maternal depression and child sleep problems. For sleep problems there is also a measurement model (gray area in figure 2). The squares are observed indicators of sleep problems, the λ denote factor loadings, and the Δ denote scaling factors capturing heterogeneity in variance of the latent response variables for observed indicators of sleep problems across time. The equations and assumptions of this model are also explained in detail elsewhere (Boden et al., 2010). The model partitions the covariance between the two variables of interest into four effects divided into two types. The first type is causal where variable 1 causes variable 2 or vice versa (β25 and β41, figure 2). The second type is non-causal where either time-invariant common factors (Ψ, figure 2) contribute to covariance or time-variant common factors (Θ, figure 2) contribute to covariance. Importantly, only Θ25 and Θ36 at T2 and T3, respectively, can be interpreted as measures of time-invariant common factors for maternal depression and child sleep problems. Θ14 at T1 is modelled as
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a baseline total association between maternal depression child sleep problems. All of these effects can be estimated jointly.

To enhance interpretability at the same time as retaining metric across time, we standardized both maternal depression scale and the latent factor indexing child sleep problems using the variance at T1. That is all the covariance matrices and estimates from structural equation models can be interpreted as standardized results (i.e. covariances as correlations and betas as standardized betas).

We aimed to reduce the model by setting the causal paths (i.e., β25 and β41, figure 2) to zero and compare model fit. Four models were tested: 1) A reciprocal model where maternal depression causes child sleep problems and vice versa (model 0); 2) a child-driven model where only child sleep problems causes maternal depression (model 1); 3) a mother-driven model where only maternal depression causes child sleep problems (model 2); and, four, a common factor model where neither variables causes each other, but are associated due to common factors. We estimated the models with only continuous data (i.e., the SCL-25) using maximum likelihood (ML) and models including categorical data (i.e. indicators of sleeping problems) using the mean and variance adjusted diagonal weighted least squares (WLSMV) estimator. We calculated the chi-square difference of models using the mean variance adjusted diagonal weighted least squares (WLSM). We identified the model best fitted to the data by comparing comparative fit index (CFI), the root mean square of approximation (RMSEA), and the Akaike’s Information Criterion (AIC). For models estimated by ML, we also used the difference in -2loglikelihood, which has a chi-square ($\chi^2$) distribution and degrees of freedom (df) (not estimateable using WLSMV and linear model constraints, but estimateable using WLSM). A higher CFI and a lower RMSEA indicates a better fit to the data. An AIC increase greater than two indicates a poorer fit relative to the comparison model (Model 0) (Akaike, 1987). By the principle of parsimony, we chose the
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model with the best values of these fit indices. We corrected for attrition in the analyses under the missing at random assumption by including all available cases with valid data at one or more time point. All analyses were done in Mplus, version 7.0.

Results

Child sleep problems

Indicators of child sleep problems.

We subjected the four indicators of child sleep problems to a CFA for ordinal data (also known as item response theory modeling). As shown by the factor loadings in table 2, the four indicators differed in how reliable they were as indicators of child sleep problems. Co-sleeping in response to nocturnal awakenings loaded on the general factor of child sleep problems (factor loading = 0.82), difficulties to settle at nighttime and nocturnal awakenings proved to be adequate indicators of child sleep problems (factor loadings = 0.68 and 0.61), and total sleep time proved to be a modest indicator of child sleep problems (factor loading = 0.44).

Frequencies for the indicators of child sleep problems are shown in table 1. Across time fewer children were scored in the extreme categories of total sleep time (t2 vs t1 p < .01; t3 vs t1 p < .01; t3 vs t2 p = 0.05); leading to an increase in the middle category “sometimes sleep very little” at 2.5 and 4 years. Ratings of difficulties to settle at nighttime appeared to increase slightly after t2 (t2 vs t1 p < .56; t3 vs t1 p < .01; t3 vs t2 p = 0.02). While there was a slight reduction in nocturnal awakenings after t1 (t2 vs t1 p = .06; t3 vs t1 p < .01; t3 vs t2 p = 0.14), there was an increase in co-sleeping in response to nocturnal awakenings after t1 (t2 vs t1 p < .01; t3 vs t1 p < .01; t3 vs t2 p = 0.71).

The longitudinal structure of child sleep problems.
We found child sleep problems to be moderately stable during preschool age (table 3). The 1
year (i.e., 1.5 to 2.5 years), 1.5 year (i.e., 2.5 to 4 years), and 2.5 year (i.e., 1.5 to 4 years)
covariance was 0.62, 0.51, and 0.36, respectively.

We estimated models of longitudinal course of child sleep problems (figure 1) by
WLSMV. Compared to the saturated autoregressive fixed effect model (figure 1c) ($CFI = 0.983; RMSEA = 0.035, 95\% CI 0.026-0.044$), the more parsimonious autoregressive model (figure 1b) had the best fit to the data ($CFI = 0.983; RMSEA = 0.035, 95\% CI 0.026-0.043$). The more parsimonious fixed effect model (figure 1a) had a lesser fit to the data ($CFI = 0.978; RMSEA = 0.039, 95\% CI 0.031-0.048$). The autoregressive model could be further reduced without a reduction in fit by equalizing the autoregressive paths across time (i.e. $\beta_{21} = \beta_{32}$ (figure1b)) ($CFI = 0.984; RMSEA = 0.033, 95\% CI 0.024-0.042$).

We estimated the stability of child sleep problems between each interval ($\beta_{21}$ and $\beta_{32}$, figure1b) to 0.59 ($p < 0.00$). The total stable variance from one time point to the next was 41%. We estimated the relative importance of emerging factors at each time point by tracking prediction from one time point to the next in an autoregressive structural equation model. The relative importance of factors for child sleep problems present at 1.5, 2.5, and 4 years for observed child sleep problems at these time points is presented in figure 3. Sleep problems at 1.5 years accounted for 41% of the variation in sleep problems at 2.5 years, which leads us to infer that factors for child sleep problems present at 1.5 years accounted for 41% of the variance in child sleep problems at 2.5 years. Likewise, factors for child sleep problems at 1.5, 2.5, and 4 years accounted for 21%, 31%, and 48%, respectively, of the variance in child sleep problems at 4 years.

Maternal depressive symptoms
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Indicators of maternal depressive symptoms. Symptoms of maternal depression were moderate to highly stable from 1.5 to 4 years after birth (table 3). The 2.5-year stability (covariance = 0.63) was approximate to the shorter 1-year stability (covariance = 0.63) and 1.5 year stability (covariance = 0.65).

The longitudinal structure of maternal depressive symptoms.

We estimated models of longitudinal course of depressive symptoms (figure 1) by ML. Compared to the saturated autoregressive fixed effect model, the nested fixed effect model (figure 1a) had the best fit to the data ($\chi^2 = 0.51, df = 2, p = 0.77; CFI = 1.000; RMSEA = 0.000, 95\% CI 0.000-0.042$). The nested autoregressive model (figure 1b) had a poor fit to the depressive symptom data ($\chi^2 = 85.03, df = 1, p < 0.00; CFI = 0.910; RMSEA = 0.297, 95\% CI 0.245-0.352$).

In total, 66% of the variance in depressive symptoms could be attributed to time-invariant factors. Conversely, 34% of the variance in maternal depressive symptoms could be attributed to time-variant factors.

Child sleep problems and maternal depressive symptoms

We present the correlations between all items used in the following structural equation models in appendix 1. Correlations between maternal depression and the sleep problem indicators were small in magnitude, ranging from .04 to .19.

We estimated the within time covariance between child sleep problems and maternal depressive symptoms to be 0.24, 0.06, and 0.21 at 1.5, 2.5, and 4 years, respectively (table 3). The between time covariance for early child sleep problems and later maternal depressive symptoms was in average 0.19. Conversely, the between time covariance for early maternal depressive symptoms and later child sleep problems was in average 0.15.
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We tested four bivariate models for maternal depressive symptoms and child sleep problems. The baseline model (Figure 2; model 0, table 4) had the best fit to the data (table 4). Neither the path representing mother-driven effects nor the path representing child-driven effects could be dropped from the model without a reduction in fit (model 1 to model 2, table 4).

According to the best-fitting model there is a reciprocal effect where maternal depressive symptoms causes child sleep problems ($\beta = 0.09; p = 0.03$) and child sleep problems causes maternal depressive symptoms ($\beta = 0.34; p < 0.00$). However, the child-driven effect was significantly stronger than the mother-driven effect ($\chi^2 = 6.45, df = 1, p = 0.01$). Covariance between specific factors for change in child sleep problems and change in maternal symptoms of depression at 2.5 and 4 years (i.e., residual variance; $\Theta_{25}$ and $\Theta_{36}$, figure 2) indicated effect of common factors, or third variables, that account for variation in both sleep problems and depression. These covariances were negative at 2.5 years (-0.28; $p < 0.01$) and non-significant at 4 years (-0.09; $p = .06$).

Discussion

By applying an autoregressive fixed effects model on longitudinal data from a population-based study we found the association between maternal depressive symptoms and child sleep problems from infancy to pre-school age could be attributed to three processes: Mother-driven mechanisms, child-driven mechanisms, and common factors. Mother-driven mechanisms positively predicted child sleep problems, and child-driven mechanisms positively predicted maternal depressive symptoms. However, the effect of the child-driven mechanisms was significantly stronger than the mother-driven mechanisms. After accounting for mother- and child-driven mechanisms, common factors to maternal depressive symptoms
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and child sleep problems contributed negatively to covariance at 2.5 years and were non-significant at 4 years.

Indicators of child sleep problems

There was a change across time in what was the most prevalent indicator of child sleeping problems, with fewer children scoring in the extreme categories of total sleep time at 2.5 and 4 years. While there was a slight reduction in nocturnal awakenings, and an increase in co-sleeping in response to nocturnal awakenings, settling difficulties at bedtime was relatively stable from 1.5 to 4 years of age. Both a decline in the total sleep time (Iglowstein, Jenni, Molinari, & Largo, 2003), and stability of difficulties to settle at bedtime (Galland, Taylor, Elder, & Herbison, 2012; Mindell, Meltzer, Carskadon, & Chervin, 2009) is supported in previous findings. However, the literature is inconclusive with regard to the stability of nocturnal awakenings (Hysing et al., 2014; Touchette et al., 2005). We only found a slight reduction in nocturnal awakenings throughout the period, a finding not in discordance with the aforementioned literature. Although there are substantial cultural differences in co-sleeping (Blair & Ball, 2004; Hysing et al., 2014; Touchette et al., 2005; Willinger, Ko, Hoffman, Kessler, & Corwin, 2003), our finding of an increase in difficulties during preschool age could be due to the child’s ability to move from its own bed to the parent’s bed during nighttime. Further, the definition of co-sleeping in the present study was that the child slept with the parents due to nocturnal awakenings. This may differ from more descriptive co-sleeping where the child shares a bed with the parents regardless of sleep quality.

By CFA for ordinal data we found a difference in the reliability of the indicators of child sleep problems. It appeared that co-sleeping as a response to nocturnal awakenings was the best indicator for general sleeping problems. This may be understood as an indicator of severity of nocturnal awakenings that triggers parental response. Difficulties to settle at
bedtime and nocturnal awakenings were moderate indicators of child sleep problems during the preschool age. Finally, total sleep time proved to be only a modest indicator of child sleep problems during preschool age. The latter finding may not be surprising as this is an indicator of sleep duration, which is often considered a partly overlapping, but separate construct from sleep problems.

The structure of child sleep problems

We found child sleep problems to be relatively stable across 1 to 1.5 years, but also evidence of lower stability across 2.5 years. This fits well to our finding that child sleep problems follow an autoregressive covariance pattern from 1.5 to 4 years. Our finding that time-invariant factors were non-significant suggests that sleeping problems present at 1.5 years could have effect on sleep problems at 2.5 and 4 years, and sleep problems at 2.5 years effect on of sleeping problems at 4 years. We found that an autoregressive model for child sleep problem to be the model best fitted to the data. The implication of an autoregressive hypothesis is that a reduction in early sleeping problems could indeed prevent future sleeping problems. There is some support in the literature that when the children are older they are more prone to prolong the bedtime routine and attract parental attention at night problems such as increase from 1 year to 1.5 years (Beltrami & Hertzig, 1983; Byars et al., 2012).

Parent bedtime behaviors (e.g. maternal presence at night, not having a consistent bedtime routine, or letting the child sleep in the parents’ bed) is related to persistency in sleep problems (Adair et al., 1991; Burnham, Goodlin-Jones, Gaylor, & Anders, 2002; Hysing et al., 2014; Mindell, Telofski, et al., 2009; Touchette et al., 2005). How the parents handle the child’s sleep problems that emerge during preschool age would then be expected to elicit a similar pattern of parental behavior. For example, Burnham et al. (2002) suggested that the parents bedtime behavior contribute negatively to the child’s ability to self soothe. In this
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perspective, we could expect dysfunctional bedtime behavior patterns in both the child and their parents starting at 1.5 years to persist, and in part explain sleep problems at 2.5 and 4 years. By helping parents with altering their bedtime behavior early on, it is likely that the child sleep problems will improve rather than persist.

The structure of maternal depressive symptoms

We found that the stability of maternal depressive symptoms was not related to time of measurement and to followed a monotonic covariance pattern (i.e. all time points are equally associated) from 1.5 to 4 years after birth. Hence, we also found autoregressive effects to be non-significant. This conforms to a notion of maternal depression present at 1.5 years not having an effect on maternal depression at 2.5 and 4 years, and maternal depression at 2.5 years not having an effect on maternal depression at 4 years. However, we did find time-invariant factors for maternal depression to explain 66% of the variance in symptoms of maternal depression. The implication of this notion is that a reduction in time-variant factors for maternal depression will only have a curative effect on that given time-point. Only a reduction in time-invariant factors for maternal depression will have a curative effect on maternal depression across time.

The association between child sleep problems and maternal depressive symptoms

Maternal depressive symptoms were associated with child sleep problems; a finding in line with previous studies (Gelman & King, 2001; Goldberg et al., 2013; Martin et al., 2007; Zuckerman et al., 1987). Furthermore, we found early child sleep problems to be more strongly associated with later maternal depressive symptoms than early maternal depressive symptoms were associated with later child sleep problems.
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Using structural models, we estimated that the association between maternal depressive symptoms and child sleep problems could be attributed to mother-driven mechanisms, child-driven mechanisms, and partly to common factors. We also found child-driven mechanisms to be significantly stronger than mother-driven mechanisms.

Child-driven models of children’s sleep problems and maternal depressive symptoms.

The findings in this study corroborates the notion that child sleep problems have an effect on maternal depression, and, according to this notion, a reduction in child sleep problems could lead to an improvement in maternal depressive symptoms. This finding is consistent with several former findings (Hiscock et al., 2008; Lam et al., 2003; Meltzer & Mindell, 2007; Moore et al., 2012) and inconsistent with others (Gelman & King, 2001; Teti & Crosby, 2012; Warren et al., 2006). Few of these studies did however examine this in a robust manner. Although Teti and Cosby (2012) found support for the mother-driven model compared to the child-driven model using a mediation approach, they did not formally test the fit to the data for the two competing models making it unclear if the child-driven model had the best fit to the data.

Child sleep problems could affect maternal depression through different mechanisms. For instance, nightly parental interventions interfering with parental sleep might lead to parental stress, fatigue and symptoms of depression (i.e., child-driven model) (Meltzer & Mindell, 2007; Warren et al., 2006). In support of this, Moore et al. (2012) found parental stress to be a mediator of the relation between child sleep problems and parental depressive symptoms. An alternative view is that child sleep problems lead to child behavior problems, which in turn leads to parental stress, fatigue and symptoms of depression (Sivertsen et al., 2015). Giving the parents help with their children’s sleep problems, by some sort of guided sleep intervention program, should not only lead to improved sleep for children and their
MECHANISMS FOR CHILD SLEEP PROBLEMS AND MATERNAL DEPRESSIVE SYMPTOMS

parents, but also to an improvement in the parents psychological well-being (i.e., fewer maternal depressive symptoms).

We found support for both models, but there was stronger support of the child-driven model than the mother-driven model in this study. To the best of our knowledge, this is the first study that formally tests the equality of child and mother-driven processes for child sleep problems and maternal depressive symptoms. Future studies should aim to replicate this finding using population based longitudinal data with more measurement points and shorter time intervals.

Mother-driven models of children’s sleep problems and maternal depressive symptoms.

This finding corroborates a notion of maternal depression directly affects child sleep problems, and, according to this notion; a reduction in maternal psychopathology could lead to a reduction in child sleep problems. These findings are consistent with some earlier literature in the field (Gelman & King, 2001; Teti & Crosby, 2012; Warren et al., 2006; Ystrom et al., 2017), but contrary to other studies (Hiscock et al., 2008; Lam et al., 2003; Meltzer & Mindell, 2007; Mindell, Telofski, et al., 2009; Moore et al., 2012).

Maternal depression could have an effect on child sleep problems through a range of different mechanisms. How negative cognitions and emotions could affect child sleep problems mediated through different parent bedtime and nighttime behaviors are two mechanisms that have been paid close attention to. Mothers with depressive symptoms are more likely to have negative cognitions about setting limits for their children, increased doubts regarding parenting competence and worry more often about infants sleep (Teti & Crosby, 2012). These mothers more easily perceive themselves as insensitive, neglectful or even abusive towards the child if they are not highly involved at bedtime or night time, and even tends to more often interpret their children crying as a sign of anxiety or distress.
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(Morrell, 1999; Sadeh, 2005). As a result it is postulated that these negative cognitions even tend to affect maternal bedtime and nighttime behavior and could even result in scenarios like putting the child to sleep late, using an inconsistent bedtime routine, maternal presence at bedtime, or bed sharing during night. These behaviors will interfere with the development of self-soothing skills in the child, as bedtime interactions with parents are very rewarding and provide positive feedback that maintains dependence on parents (Adair et al., 1991). This has been suggested to lead to different child sleep problems (Adair et al., 1991; Mindell, Telofski, et al., 2009; Teti et al., 2010).

In addition, Morrell (1999) claims that these negative cognitions often are related to strong emotions such as guilt, shame, and anger that serve as a negative reinforcement for any change in parenting behavior. Theoretically, emotionally availability measured by sensitivity, structuring, non-intrusiveness and non-hostility will promote feelings of safety and security in children. The ability to feel safe in one’s sleep environment is essential to the ability to feel relaxed and achieve deep sleep (Dahl & El-Sheikh, 2007). The emotional availability in the mothers at bedtime has indeed shown to be related to children’s sleep problems, even more than the parental bedtime and nighttime behavior (Teti et al., 2010). It could seem less important what mothers do compared to how they do it.

Common factors for child sleep problems and maternal depressive symptoms.

We found the covariance between common factors for change in child sleep problems and change in maternal depressive symptoms to be negative at 2.5 years and non-significant at 4 years. These residual associations are indicative of third variables that account for variation in both maternal depression and child sleep problems. These common factors seem to account for some of the covariation between maternal depression and child sleep. Future studies should aim to replicate these findings using longitudinal extended children of twin and sibling designs estimating shared household factors.
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Strengths and limitations of the study

It is important to acknowledge that inferences taken in this article rest on underlying assumptions that are necessary to identify the models presented. The most important assumption is that the association between child sleep problems and maternal depressive symptoms is represented by a process that is qualitatively operative throughout the course of this study (Hamaker & Wichers, 2017). Although this is undoubtedly a strong assumption in a study of child development, this assumption is vital to the model of reciprocal effect. Future studies should aim to have a larger number of measurement points across a shorter period of time. It is assumed in models of reciprocal effect that variables of interest are measured without measurement error. The excellent reliability of the depressive symptom measure and the use of latent child sleep problems variables is therefore a significant strength of the current study. The use of maternal reports of child sleeping problems represents a limitation of the study. Future studies should apply objective measures of child sleep, such as actigraphy.

Last, and importantly, the models we have applied are only approximations to a more complex reality. Therefore inferences drawn from this study should be viewed as hypothesis generative rather than absolute.

Clinical implications

It is possible to draw clinical implications according to the best fitting model. One, since there is no path going from depressive symptoms at one time point to the next, we would not expect improvement of mood at a single time point to have long term effect. Two, according to the best fitting model, treatment of child sleep problems at a given time point would be expected to improve sleep problems at a later time point. Three, child-driven processes were indeed stronger than mother-driven processes. Hence, we would expect that improvement of
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Child sleep problems to lead to a curative cascade for both later child sleep problems and concurrent maternal depressive symptoms.

We believe that a reasonable approach would be to assess child sleep problems when meeting mothers with depressive symptoms in the clinic. Such assessments can be done by any health professional. Furthermore, having information about child sleep problems could be indicative for the chronicity of maternal depression. Most important is to find efficient treatments for child sleep problems. Ramchandani, Wiggs, Webb, and Stores (2000) claimed in a systematic review that sedation was the most frequently used treatment for childhood sleep problems. The same authors concluded that although drug treatment seemed to be effective in the short term, the long-term efficacy was more uncertain. They concluded that behavioral treatment was more effective in the short term and also had beneficial effects in the long term. This treatment includes behavioral programs guided by a therapist, parent educational groups, and self-help booklets (Ramchandani et al., 2000; Sadeh, Tikotzky, & Scher, 2010). In a recent randomized controlled trail on infants (6-16 months), Gradisar et al. (2016) found graduated extinction and bedtime fading to provide significant sleep benefits compared to sleep education controls. Although concern has been raised concerning possible stress associated with extinction-based treatments (Blunden, Thompson, & Dawson, 2011), the Gradisar study found neither adverse stress responses in terms of increased cortisol levels, nor any long-term effects on parent-child attachment or child emotions and behavior.

Conclusion

According to the model best fitted to the data, we found the association between child sleep problems and maternal depressive symptoms to be explained by both child-driven and mother-driven processes, but the effect of child-driven processes was significantly larger than the effect of mother-driven processes. Accordingly, a reduction in maternal depressive symptoms...
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symptoms will be beneficial for child sleep problems, but a reduction in child sleep problems will be even more beneficial for maternal depressive symptoms.

References


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Wake, M., Morton-Allen, E., Poulakis, Z., Hiscock, H., Gallagher, S., & Oberklaid, F.

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Figure Legend

Figure 1. Autoregressive fixed effect path models for longitudinal data. Squares denote observed variables. Circles denote latent variables. One-headed arrows denote regression paths. $\Psi_{11}$ denote time invariant variance. $\Theta_{11}$, $\Theta_{22}$, and $\Theta_{33}$ denote time variant variance. $\beta_{21}$ and $\beta_{32}$ denote autoregressive paths where each time point is regressed on the previous time point. Figure 1a (“Time-invariant fixed effect model”) contains fixed effects, where time invariant variance is modeled, and the observed covariance matrix is here expected to have a monotonic pattern. Figure 1b (“Autoregressive model”) contains only autoregressive paths, and the observed covariance matrix is expected to have an autoregressive pattern. Figure 1c (“Autoregressive fixed effect model”) contains both fixed effects and autoregressive paths, where the observed covariance matrix is expected to be a mixture of monotonic and autoregressive patterns.

Figure 2. Best fitting bivariate autoregressive fixed effect model. Squares denote observed variables. Circles denote latent variables. One-headed arrows denote regression paths. Depression has a monotonic structure across time (see figure 1a) and sleep problems has an autoregressive pattern across time (see figure 1b). $\Psi_{11}$ denote time invariant variance for depression. $\Theta_{22}$, $\Theta_{33}$, $\Theta_{55}$, and $\Theta_{66}$ denote time variant residual variance. $\Theta_{11}$ denote time variant variance for depression at time 1. $\beta_{54}$ denote autoregressive paths for sleep problems. $\beta_{25}$ and $\beta_{41}$ denote the reciprocal effect of depression on sleep problems and vice versa. $\lambda$ denote factor loadings for the sleep problem items (equal across time). $\Delta$ denote scaling factors for sleep problem items at time 2 and 3. Scaling factors estimate changes in variance across time for the latent response variables.

Figure 3. Explained variance in sleep problems across time according to the best fitting autoregressive model. The factors contributing to change and stability in child sleep problems correspond to the $\Theta_{11}$, $\Theta_{22}$, and $\Theta_{33}$ in figure 1b. The figure illustrates the relative importance of
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Factors for child sleep problems present at earlier time points versus factors emerging at later time points. For example, factors for child sleep problems present at 1.5 years (i.e. white area; $\Theta_{11}$ in figure 1b) explain 21% of the variance in child sleep problems at 4 years through the path $\beta_{21} \times \beta_{32}$ in figure 1b. Emerging factors at 2.5 and 4 years, $\Theta_{22}$, and $\Theta_{33}$ in figure 1b, respectively, explain the remaining variance in sleep problems at 4 years.
Table 1. Frequency of child sleeping problem indicators.

<table>
<thead>
<tr>
<th>Item</th>
<th>[Response category]</th>
<th>1.5 years</th>
<th>2.5 years</th>
<th>4 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time</td>
<td>a) “Usually sleep very little”</td>
<td>10.6 %</td>
<td>8.4 %</td>
<td>6.5 %</td>
</tr>
<tr>
<td></td>
<td>b) “Sometimes sleep very little”</td>
<td>77.2 %</td>
<td>83.7 %</td>
<td>86.8 %</td>
</tr>
<tr>
<td></td>
<td>c) “Sleeps neither little nor much”</td>
<td>8.9 %</td>
<td>5.5 %</td>
<td>4.6 %</td>
</tr>
<tr>
<td></td>
<td>d) “Usually sleep very much”</td>
<td>3.3 %</td>
<td>2.5 %</td>
<td>2.1 %</td>
</tr>
<tr>
<td>1: Difficulties to settle at nighttime</td>
<td>a) [No difficulties]</td>
<td>77.0 %</td>
<td>77.0 %</td>
<td>72.9 %</td>
</tr>
<tr>
<td></td>
<td>“Easy to get to bed and to sleep”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) [Moderate difficulties]</td>
<td>20.8 %</td>
<td>19.8 %</td>
<td>22.6 %</td>
</tr>
<tr>
<td></td>
<td>“Some difficulties in settling at bedtime”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>c) [Definite difficulties]</td>
<td>2.2 %</td>
<td>3.2 %</td>
<td>4.5 %</td>
</tr>
<tr>
<td></td>
<td>“Often takes over an hour to settle at bedtime”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2: Nocturnal awakenings</td>
<td>a) [No difficulties]</td>
<td>40.9 %</td>
<td>42.1 %</td>
<td>45.0 %</td>
</tr>
<tr>
<td></td>
<td>“Hardly ever wakes at night”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) [Moderate difficulties]</td>
<td>55.0 %</td>
<td>56.1 %</td>
<td>53.7 %</td>
</tr>
<tr>
<td></td>
<td>“Sometimes wakes at night”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>c) [Definite difficulties]</td>
<td>4.1 %</td>
<td>1.8 %</td>
<td>1.2 %</td>
</tr>
<tr>
<td></td>
<td>“Frequently wakes at night and is difficult to settle”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3: Co-sleeping in response to nocturnal awakenings</td>
<td>a) [No difficulties]</td>
<td>70.7 %</td>
<td>59.8 %</td>
<td>55.8 %</td>
</tr>
<tr>
<td></td>
<td>“Never sleeps with parent”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) [Moderate difficulties]</td>
<td>23.6 %</td>
<td>31.5 %</td>
<td>38.0 %</td>
</tr>
<tr>
<td></td>
<td>“Sometimes sleeps with parent because upset or doesn’t want to sleep alone”</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>c) [Definite difficulties]</td>
<td>5.7 %</td>
<td>8.7 %</td>
<td>6.2 %</td>
</tr>
<tr>
<td></td>
<td>“Often sleeps with parent because upset or doesn’t want to sleep alone”</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2. Confirmatory factor analysis of child sleeping problem indicators.

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor loading&lt;sup&gt;a&lt;/sup&gt;</th>
<th>scalar factor&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.5y</td>
<td>4y</td>
</tr>
<tr>
<td>Total sleep time</td>
<td>0.44±0.037&lt;sup&gt;c&lt;/sup&gt;</td>
<td>1.12±0.041</td>
</tr>
<tr>
<td>1: Difficulties to settle at nighttime</td>
<td>0.68±0.039</td>
<td>0.99±0.054</td>
</tr>
<tr>
<td>2: Nocturnal awakenings</td>
<td>0.61±0.041</td>
<td>1.17±0.069</td>
</tr>
<tr>
<td>3: Co-sleeping in response to nocturnal awakenings</td>
<td>0.82±0.043</td>
<td>0.89±0.052</td>
</tr>
</tbody>
</table>

Note. <sup>a</sup>Factor loadings are set to equal for all time points. <sup>b</sup>Scalar factors refers to changes in variance of the latent liability response variable for the ordered categorical variable, and are fixed to unity at 1.5 years. <sup>c</sup>Standard error (all such values).
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Table 3. Means, variances, and covariances for maternal symptoms of depression and child sleeping problems between ages 1.5 and 4 years.

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Child sleep problems</th>
<th>Symptoms of depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1.5y</td>
<td>2.5y</td>
<td>4y</td>
</tr>
<tr>
<td>Child sleep problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.5y</td>
<td>0.00* (1.00)</td>
<td>1.00*</td>
<td></td>
</tr>
<tr>
<td>2.5y</td>
<td>0.08 (0.93)</td>
<td>0.62**</td>
<td>0.87**</td>
</tr>
<tr>
<td>4y</td>
<td>0.11 (0.75)</td>
<td>0.36**</td>
<td>0.51**</td>
</tr>
<tr>
<td>Symptoms of depression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.5y</td>
<td>0.00 (1.00)</td>
<td>0.24**</td>
<td>0.07</td>
</tr>
<tr>
<td>2.5y</td>
<td>-0.12** (0.96)</td>
<td>0.21**</td>
<td>0.06</td>
</tr>
<tr>
<td>4y</td>
<td>-0.18** (0.99)</td>
<td>0.21**</td>
<td>0.16*</td>
</tr>
</tbody>
</table>

Note. * p < .05; ** p < .01. \*Fixed parameter. Estimates derived from a saturated model with all variances and covariances estimated. N = 956 (n at t1 = 939, n at t2 = 781, and n at t3 = 750. Variances are on the diagonal, covariances below the diagonal.
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Table 4. Parameter estimates from bivariate autoregressive fixed effects models on maternal depressive symptoms and child sleeping problems.

<table>
<thead>
<tr>
<th>Model</th>
<th>depression→sleep</th>
<th>depression←sleep</th>
<th>CFI</th>
<th>RMSEA (95%CI)</th>
<th>( \chi^2 )</th>
<th>df</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 0*</td>
<td>0.09 (0.01-0.17)</td>
<td>0.34 (0.18-0.49)</td>
<td>0.975</td>
<td>0.033 (0.026-0.040)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>0</td>
<td>0.36 (0.21-0.51)</td>
<td>0.974</td>
<td>0.034 (0.027-0.041)</td>
<td>4.91</td>
<td>1</td>
<td>.02</td>
<td>2.91</td>
</tr>
<tr>
<td>Model 2</td>
<td>0.14 (0.07-0.21)</td>
<td>0</td>
<td>0.962</td>
<td>0.041 (0.035-0.048)</td>
<td>16.95</td>
<td>1</td>
<td>&lt;.01</td>
<td>14.95</td>
</tr>
<tr>
<td>Model 3</td>
<td>0</td>
<td>0</td>
<td>0.956</td>
<td>0.044 (0.038-0.050)</td>
<td>31.14</td>
<td>2</td>
<td>&lt;.01</td>
<td>27.14</td>
</tr>
</tbody>
</table>

Note. *best fitting model. depression→sleep refers to beta coefficient from mother to child (mother driven). Depression←sleep refers to beta coefficient from child to mother (child driven). CFI = Confirmatory fit index; RMSEA = Root mean square error of approximation. AIC = Akaike’s information criterion. *The chi-square difference was computed using the mean adjusted diagonal weighted least squares estimator. All other estimations were done using the mean and variance adjusted weighted least squares estimator.
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Figure 1

a) Time-invariant fixed effect model

b) Autoregressive model

c) Autoregressive fixed effect model
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Figure 2

Depression  Sleep problems

T1 – 1.5 years

T2 – 2.5 years

T3 – 4.0 years
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Figure 3. Explained variance in sleep problems across time according to the best fitting autoregressive model.
### Appendix 1. Correlations between all items used in analyses.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5y* SCL-25</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2.5y SCL-25</td>
<td>.66</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>4y SCL-25</td>
<td>.64</td>
<td>.68</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1.5y Total sleep time</td>
<td>.08</td>
<td>.09</td>
<td>.13</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>1.5y Sleep latency</td>
<td>.20</td>
<td>.20</td>
<td>.19</td>
<td>.35</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>1.5y Night awakenings</td>
<td>.18</td>
<td>.16</td>
<td>.12</td>
<td>.26</td>
<td>.40</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>1.5y Dislike of sleeping alone</td>
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MECHANISMS FOR CHILD SLEEP PROBLEMS AND MATERNAL DEPRESSIVE SYMPTOMS

Note. SCL-25 is the Hopkins Symptom Checklist. *y=years. Correlations between symptoms of depression are product-moment correlations.

Correlations between symptoms of depression and indicators of child sleep problems are polyserial correlations. Correlations between indicators of child sleep problems are polychoric correlations.