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| 4 | Sleep problems in preschoolers and maternal depressive symptoms: An evaluation of mother- |
| 5 | and child-driven effects |
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Abstract

35 Child sleep problems are associated with maternal depressive symptoms. It is unclear to what 36 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 37 38 maternal depressive symptoms; or mother-driven processes, where maternal depressive 39 symptoms influence child sleep problems. Common factors could be shared genetic and 40 familial environmental risk. Child- and mother-driven processes are direct in the sense that 41 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 42 43 based longitudinal sample comprising 956 families assessed at 1.5, 2.5, and 4 years of age, 44 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 45 46 sleep problems and maternal depressive symptoms by both child-driven and mother-driven 47 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 48 49 problems will have considerable effect on maternal depressive symptoms. Furthermore, our 50 model supports that treatment of current child sleep problems will have a direct effect on 51 future sleep problems, and also an indirect effect on future maternal depressive symptoms. 52 We recommend that health professionals should assess child sleep problems in mothers at 53 risk for depression.

54

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

57

Introduction

58 Sleep problems are prevalent throughout childhood (Byars, Yolton, Rausch, Lanphear, & 59 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 60 61 experience that their sleep problems, such as frequent nocturnal awakenings and difficulties 62 in settling at night, become chronic and endure well into later childhood (Byars et al., 2012; 63 Hysing et al., 2014; Lam, Hiscock, & Wake, 2003; Wake et al., 2006). 64 The etiology of sleep problems in young children is multifactorial and both genetic and environmental factors are important (Gregory & O'Connor, 2002). Maternal depression 65 66 has repeatedly been shown to be associated with sleep problems in their offspring (Martin, 67 Hiscock, Hardy, Davey, & Wake, 2007; Zuckerman, Stevenson, & Bailey, 1987). From pregnancy and throughout the preschool years, the peak level of maternal depressive 68 69 symptoms is when the child is between 1.5 and 3 years (Ystrom et al., 2014). Maternal 70 depression is related to parental behavior, and research suggests that maternal depression may 71 affect child development on several domains, including cognitive and language development 72 (Grace, Evindar, & Stewart, 2003; Sohr-Preston & Scaramella, 2006), mental health 73 problems (Goodman et al., 2011; Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002), 74 suboptimal diet (Ystrom, 2012; Ystrom, Barker, & Vollrath, 2012), and a number of different 75 social, emotional and behavior problems (Goodman et al., 2011; Grace et al., 2003; Nilsen, 76 Gustavson, Røysamb, Kjeldsen, & Karevold, 2013). There is a limited number of community 77 and population-based studies examining the association between maternal depression and 78 sleep problems in younger children. Significant associations between maternal depression 79 and sleep problems have been found in both infants (Bayer, Hiscock, Hampton, & Wake, 80 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). The direction 81

82 of effect in these studies is less clear, and either child, mother, or common factors may be 83 important. However, using a large sibling study, Ystrom et al. (2017) recently found support 84 for effects going from mother to child, but not from child to mother in 1.5 year old children. 85 One perspective is the "child-driven" model, which suggests that sleep problems in 86 children contribute to maternal depressive symptoms. Most of the literature supporting this 87 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 88 89 (Lam et al., 2003; Meltzer & Mindell, 2007; Moore, Gordon, & McLean, 2012). According 90 to this view, helping parents with their children's sleep problems, e.g. a guided sleep 91 intervention program, should not only lead to improved sleep for children and their parents, 92 but also to an improvement in the parents' psychological well-being (e.g. less maternal 93 depressive symptoms). There are several intervention studies on infants, toddlers and 94 preschoolers supporting this assumption (Hiscock, Bayer, Hampton, Ukoumunne, & Wake, 95 2008; Lam et al., 2003).

96 A second perspective is the "mother-driven" model of children's sleep problems and 97 maternal depressive symptoms suggesting that maternal depressive symptoms contribute to 98 children's sleep problems (Ystrom et al., 2017). This contribution could be mediated through 99 a range of putative maternal behaviors (Gelman & King, 2001; Teti & Crosby, 2012; Warren, 100 Howe, Simmens, & Dahl, 2006). For instance, depressive mothers tend to spend less time in 101 positive interactions with their children, they report more negative perceptions of their 102 children's behaviors, and tend to be more hostile towards their children and make more 103 negative appraisals of their children's behaviors than non-depressive mothers (Cornish et al., 104 2006; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Such interactions and appraisals could 105 influence maternal bedtime and nighttime behavior, leading to poorer self-soothing skills and

106 risk for sleep problems in the child (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 107 1991; Mindell, Telofski, Wiegand, & Kurtz, 2009; Teti, Kim, Mayer, & Countermine, 2010). 108 A third perspective on possible explanatory factors of maternal depressive symptoms 109 and child sleep problems is through common factors. First, biological mechanisms, such as 110 common genetic risk factors for psychopathology and deregulation in both the child and in 111 their mothers, have been supported by several studies (i.e. a gene-environment correlation) 112 (Gjerde et al., 2017; McAdams et al., 2014; Scarr & McCartney, 1983). Another biological 113 mechanism could be elevated hormonal levels in mothers with depressive symptoms, which 114 may affect the fetus and continue to affect the child later on. Studies have found higher levels 115 of pregnancy and perinatal cortisol and norepinephrine in depressed mothers, and suggested 116 this as a possible explanation of the association between pre- and perinatal maternal 117 depression and infant night waking very early in life (Azak, Murison, Wentzel-Larsen, Smith, 118 & Gunnar, 2013; Field, 2011; Field et al., 2007). Second, some studies indicate that social 119 and contextual stressors, including high parenting stress, stressful life events, family conflict 120 and low family income, as well as cultural aspects, could account for the association between 121 sleep problems and maternal mental health (El-Sheikh, Kelly, Bagley, & Wetter, 2012; 122 Gelman & King, 2001; Goldberg et al., 2013). Finally, individual differences in the child 123 (i.e., temperament factors) could also account for the association between children's sleep 124 problems and maternal depressive symptoms (Jimmerson, 1991; Owens-Stively et al., 1997). 125 This would constitute evocative processes also known as active gene-environment 126 correlations (i.e., a heritable phenotype in the child influences the parent) (Narusyte et al., 127 2008; Scarr & McCartney, 1983). 128 To date there are several studies advocating both child-driven and mother-driven 129 processes as the prime mechanism for the association between maternal symptoms of

130 depression and child sleeping problems, and a lack of studies advocating common factors as

131 the prime mechanism. To the best of our knowledge, no studies have integrated all three 132 perspectives in a single model and tested the significance of each mechanism. The rationale 133 of the co-twin control design can be applied to longitudinal data by use of the fixed-effects 134 regression model (Boden, Fergusson, & Horwood, 2010; Hamaker & Wichers, 2017). Such 135 analyses are indicative of the direction of effect, and both child-driven and mother-driven 136 mechanisms could both be active, leading to reciprocal effects in a feedback loop. Structural 137 equation models provide means to address this issue by applying a statistical model to the 138 data that allows reciprocal effects and select the model best fitted to the data. As of today, no 139 studies have formally tested the comparative fit of child-driven and mother-driven processes 140 for child sleep problems and maternal depressive symptoms using longitudinal panel data. 141 By applying an autoregressive fixed effects model to longitudinal data from a 142 population-based study, we aimed to estimate to what extent the association between 143 maternal depressive symptoms and child sleep problems could be attributed to: 1) child-144 driven effects, where child sleep problems causes maternal depression; 2) mother-driven 145 effects, where maternal depression causes child sleep problems; or, 3) common factors to 146 maternal depression and child sleep problems.

- 147
- 148

Method

149 *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

156 questionnaire. Of the 1,081 eligible families, 939 (87%) participated at t1. The parents who 157 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 158 159 had moved to the area and were invited to join the study. The current sample comprised 160 participants having valid data at one or more of the three time points (n=956). The 161 questionnaires were administered by the health care workers (Mathiesen, Tambs, & Dalgard, 162 1999). All participants signed informed consent forms emphasizing the confidentiality of the 163 participants, and the right to withdraw from the study at any point. The Regional Committee 164 for Medical and Health Research Ethics, South East, approved study 2013/863 165 "Intergenerational Risk for Common Mental Disorders". 166 Within the 19 health care areas 28% of the families lived in large cities, 55% lived in 167 densely populated areas, and 17% lived in rural areas. Maternal age ranged from 19 to 46 168 years at t1, with a mean of 30 years (SD=4.7). Data from the child health clinics showed that 169 non-respondents at t1 did not differ from respondents with respect to maternal age, education, 170 employment status, number of children, or marital status (Mathiesen et al., 1999). Additional 171 logistic regression analyses were conducted to examine differences between responders 172 versus non-responders at t3. Mothers responding at t3 were less likely to have boys compared 173 to girls (odds ratio (OR) = 0.63; p < .01) and were to a greater extent employed at t1 (OR = 174 1.58; p < 0.01) as compared to non-responders. There were no significant differences 175 between responders and non-responders in terms of educational level, marital status, number 176 of children, child sleep problems, or symptoms of depression. 177 178 Measures

179 *Indicators of child sleep problems.*

Sleep problems were measured using four items, one item on total sleep time and three items 180 181 from the sleep problems scale in the Behavioral Checklist (BCL) (Mathiesen & Sanson, 182 2000; Richman, 1977). The BCL consists of 19 items covering 12 behavioral categories (i.e. 183 eating, sleeping, soiling, dependency and attention seeking, relationships with siblings and 184 peers, activity, concentration, control problems, tempers, mood, worries, and fears). We 185 present the content and response categories of the BCL sleep items in table 1. Factor analysis 186 of the 19 BCL items completed by 1,047 parents of 3-year-old British children identified 187 sleeping problems as a distinct factor (Sonuga-Barke, Thompson, Stevenson, & Viney, 188 1997). We combined the four items using confirmatory factor analysis (CFA) for ordinal 189 data, also known as a graded response model within the framework of item response theory 190 (Asparouhov & Muthén, 2016; Samejima, 1969).

191 *Maternal symptoms of depression.*

192 Maternal symptoms of depression were measured by the 25-item version of the Hopkins 193 Symptom Check List (Hesbacher, Rickels, Morris, Newman, & Rosenfeld, 1980). The 194 mothers rated how often they had experienced symptoms the last week. The reliability 195 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 196 197 interest or pleasure"-were excluded from the Norwegian questionnaire because some 198 participants in the pilot-project perceived them as offensive (Mathiesen et al., 1999). We used 199 the overall mean of the 23 items, each rated on a 4-point scale ("Not at all", "A little", "Quite 200 a bit", and "Extremely"). Cronbach's alphas for maternal symptoms of depression at t1, t2 201 and t3 were .90, .89, and .90, respectively.

202

203 *Statistics*

204 First, we tested to what extent the cross-time covariance structure of maternal depression and 205 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 206 207 models for maternal depression and child sleep problems, respectively, we included them into 208 the bivariate autoregressive fixed effect model (figure 2). The variance of the time-invariant 209 fixed factors (ψ) , which is equivalent to a random intercept in a mixture model of 210 longitudinal data, is an estimate of the percentage of variance in the observations that is 211 stable. The β matrix denotes the regression paths between study variables. These parameters 212 represent direct effect between study variables. Beta effects across time are autoregressive 213 effects, while effects within time are reciprocal effects between study variables. The θ matrix 214 denotes the residual variance/covariance of the observed variables in question. These 215 represent factors uncorrelated with time-invariant factors and factors present at previous time 216 points (i.e. emerging factors). These variables are allowed to covariate within time to 217 represent common factors for maternal depression and child sleep problems. For sleep 218 problems there is also a measurement model (gray area in figure 2). The squares are observed 219 indicators of sleep problems, the λ denote factor loadings, and the Δ denote scaling factors 220 capturing heterogeneity in variance of the latent response variables for observed indicators of 221 sleep problems across time. The equations and assumptions of this model are also explained 222 in detail elsewhere (Boden et al., 2010). The model partitions the covariance between the two 223 variables of interest into four effects divided into two types. The first type is causal where 224 variable 1 causes variable 2 or vice versa (\beta25 and \beta41, figure 2). The second type is non-225 causal where either time-invariant common factors (Ψ , figure 2) contribute to covariance or 226 time-variant common factors (Θ , figure 2) contribute to covariance. Importantly, only $\Theta 25$ 227 and Θ 36 at T2 and T3, respectively, can be interpreted as measures of time-invariant 228 common factors for maternal depression and child sleep problems. Θ 14 at T1 is modelled as

a baseline total association between maternal depression child sleep problems. All of theseeffects 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).

236 We aimed to reduce the model by setting the causal paths (i.e., β 25 and β 41, figure 2) 237 to zero and compare model fit. Four models were tested: 1) A reciprocal model where 238 maternal depression causes child sleep problems and vice versa (model 0); 2) a child-driven 239 model where only child sleep problems causes maternal depression (model 1); 3) a mother-240 driven model where only maternal depression causes child sleep problems (model 2); and, 241 four, a common factor model where neither variables causes each other, but are associated 242 due to common factors. We estimated the models with only continuous data (i.e., the SCL-243 25) using maximum likelihood (ML) and models including categorical data (i.e. indicators of 244 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 245 246 variance adjusted diagonal weighted least squares (WLSM). We identified the model best 247 fitted to the data by comparing comparative fit index (CFI), the root mean square of 248 approximation (RMSEA), and the Akaike's Information Criterion (AIC). For models 249 estimated by ML, we also used the difference in -2loglikelihood, which has a chi-square (γ^2) 250 distribution and degrees of freedom (df) (not estimateable using WLSMV and linear model 251 constraints, but estimateable using WLSM). A higher CFI and a lower RMSEA indicates a 252 better fit to the data. An AIC increase greater than two indicates a poorer fit relative to the 253 comparison model (Model 0) (Akaike, 1987). By the principle of parsimony, we chose the

- 254 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
- 256 one or more time point. All analyses were done in Mplus, version 7.0.
- 257
- 258

Results

- 259 Child sleep problems
- 260 *Indicators of child sleep problems.*

261 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

263 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 =

268 0.44).

269 Frequencies for the indicators of child sleep problems are shown in table 1. Across 270 time fewer children were scored in the extreme categories of total sleep time (t2 vs t1 p < .01; 271 t3 vs t1 p < .01; t3 vs t2 p = 0.05); leading to an increase in the middle category "sometimes" 272 sleep very little" at 2.5 and 4 years. Ratings of difficulties to settle at nighttime appeared to 273 increase slightly after t2 (t2 vs t1 p < .56; t3 vs t1 p < .01; t3 vs t2 p = 0.02). While there was 274 a slight reduction in nocturnal awakenings after t 1 (t2 vs t1 p = .06; t3 vs t1 p < .01; t3 vs t2 275 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). 276

277 *The longitudinal structure of child sleep problems.*

278 We found child sleep problems to be moderately stable during preschool age (table 3). The 1

- 279 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.
- 281 We estimated models of longitudinal course of child sleep problems (figure 1) by
- 282 WLSMV. Compared to the saturated autoregressive fixed effect model (figure 1c) (CFI =
- 283 0.983; *RMSEA* = 0.035, 95%*CI* 0.026-0.044), the more parsimonious autoregressive model
- 284 (figure 1b) had the best fit to the data (CFI = 0.983; RMSEA = 0.035, 95%CI 0.026-0.043).

285 The more parsimonious fixed effect model (figure 1a) had a lesser fit to the data (CFI =

 $286 \quad 0.978; RMSEA = 0.039, 95\%CI 0.031-0.048).$ The autoregressive model could be further

reduced without a reduction in fit by equalizing the autoregressive paths across time (i.e. β 21

288 = β 32 (figure1b)) (*CFI* = 0.984; *RMSEA* = 0.033, 95%*CI* 0.024-0.042).

289 We estimated the stability of child sleep problems between each interval (β 21 and 290 β 32, figure1b) to 0.59 (p < 0.00). The total stable variance from one time point to the next 291 was 41%. We estimated the relative importance of emerging factors at each time point by 292 tracking prediction from one time point to the next in an autoregressive structural equation 293 model. The relative importance of factors for child sleep problems present at 1.5, 2.5, and 4 294 years for observed child sleep problems at these time points is presented in figure 3. Sleep 295 problems at 1.5 years accounted for 41% of the variation in sleep problems at 2.5 years, 296 which leads us to infer that factors for child sleep problems present at 1.5 years accounted for 297 41% of the variance in child sleep problems at 2.5 years. Likewise, factors for child sleep 298 problems at 1.5, 2.5, and 4 years accounted for 21%, 31%, and 48%, respectively, of the 299 variance in child sleep problems at 4 years.

300

301 Maternal depressive symptoms

302 Indicators of maternal depressive symptoms. Symptoms of maternal depression were

- 303 moderate to highly stable from 1.5 to 4 years after birth (table 3). The 2.5-year stability
- 304 (covariance = 0.63) was approximate to the shorter 1-year stability (covariance = 0.63) and
- 305 1.5 year stability (covariance = 0.65).

306 The longitudinal structure of maternal depressive symptoms.

307 We estimated models of longitudinal course of depressive symptoms (figure 1) by ML.

308 Compared to the saturated autoregressive fixed effect model, the nested fixed effect model

309 (figure 1a) had the best fit to the data ($\chi^2 = 0.51$, df = 2, p = 0.77; CFI = 1.000; RMSEA =

310 0.000, 95%CI 0.000-0.042). The nested autoregressive model (figure 1b) had a poor fit to the

311 depressive symptom data ($\chi^2 = 85.03$, df = 1, p < 0.00; CFI = 0.910; RMSEA = 0.297, 95%CI

312 0.245-0.352).

313 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.

316

317 Child sleep problems and maternal depressive symptoms

318 We present the correlations between all items used in the following structural equation

319 models in appendix 1. Correlations between maternal depression and the sleep problem

320 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).

- 323 The between time covariance for early child sleep problems and later maternal depressive
- 324 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.

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).

331 According to the best-fitting model there is a reciprocal effect where maternal 332 depressive symptoms causes child sleep problems ($\beta = 0.09$; p = 0.03) and child sleep 333 problems causes maternal depressive symptoms ($\beta = 0.34$; p < 0.00). However, the childdriven effect was significantly stronger than the mother-driven effect ($\gamma^2 = 6.45$, df = 1, p =334 335 0.01). Covariance between specific factors for change in child sleep problems and change in 336 maternal symptoms of depression at 2.5 and 4 years (i.e., residual variance; Θ 25 and Θ 36, figure 2) indicated effect of common factors, or third variables, that account for variation in 337 338 both sleep problems and depression. These covariances were negative at 2.5 years (-0.28; p <339 .01) and non-significant at 4 years (-0.09; p = .06).

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Discussion

342 By applying an autoregressive fixed effects model on longitudinal data from a population-343 based study we found the association between maternal depressive symptoms and child sleep 344 problems from infancy to pre-school age could be attributed to three processes: Mother-345 driven mechanisms, child-driven mechanisms, and common factors. Mother-driven 346 mechanisms positively predicted child sleep problems, and child-driven mechanisms 347 positively predicted maternal depressive symptoms. However, the effect of the child-driven 348 mechanisms was significantly stronger than the mother-driven mechanisms. After accounting 349 for mother- and child-driven mechanisms, common factors to maternal depressive symptoms

- and child sleep problems contributed negatively to covariance at 2.5 years and were non-
- 351 significant at 4 years.
- 352
- 353 Indicators of child sleep problems

354 There was a change across time in what was the most prevalent indicator of child sleeping 355 problems, with fewer children scoring in the extreme categories of total sleep time at 2.5 and 356 4 years. While there was a slight reduction in nocturnal awakenings, and an increase in co-357 sleeping in response to nocturnal awakenings, settling difficulties at bedtime was relatively 358 stable from 1.5 to 4 years of age. Both a decline in the total sleep time (Iglowstein, Jenni, 359 Molinari, & Largo, 2003), and stability of difficulties to settle at bedtime (Galland, Taylor, 360 Elder, & Herbison, 2012; Mindell, Meltzer, Carskadon, & Chervin, 2009) is supported in 361 previous findings. However, the literature is inconclusive with regard to the stability of 362 nocturnal awakenings (Hysing et al., 2014; Touchette et al., 2005). We only found a slight 363 reduction in nocturnal awakenings throughout the period, a finding not in discordance with 364 the aforementioned literature. Although there are substantial cultural differences in cosleeping (Blair & Ball, 2004; Hysing et al., 2014; Touchette et al., 2005; Willinger, Ko, 365 366 Hoffman, Kessler, & Corwin, 2003), our finding of an increase in difficulties during 367 preschool age could be due to the child's ability to move from its own bed to the parent's bed 368 during nighttime. Further, the definition of co-sleeping in the present study was that the child 369 slept with the parents due to nocturnal awakenings. This may differ from more descriptive 370 co-sleeping where the child shares a bed with the parents regardless of sleep quality.

371 By CFA for ordinal data we found a difference in the reliability of the indicators of 372 child sleep problems. It appeared that co-sleeping as a response to nocturnal awakenings was 373 the best indicator for general sleeping problems. This may be understood as an indicator of 374 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.

380

381 The structure of child sleep problems

382 We found child sleep problems to be relatively stable across 1 to 1.5 years, but also evidence 383 of lower stability across 2.5 years. This fits well to our finding that child sleep problems 384 follow an autoregressive covariance pattern from 1.5 to 4 years. Our finding that time-385 invariant factors were non-significant suggests that sleeping problems present at 1.5 years 386 could have effect on sleep problems at 2.5 and 4 years, and sleep problems at 2.5 years effect 387 on of sleeping problems at 4 years. We found that an autoregressive model for child sleep 388 problem to be the model best fitted to the data. The implication of an autoregressive 389 hypothesis is that a reduction in early sleeping problems could indeed prevent future sleeping 390 problems. There is some support in the literature that when the children are older they are 391 more prone to prolong the bedtime routine and attract parental attention at night problems 392 such as increase from 1 year to 1.5 years (Beltramini & Hertzig, 1983; Byars et al., 2012). 393 Parent bedtime behaviors (e.g. maternal presence at night, not having a consistent 394 bedtime routine, or letting the child sleep in the parents' bed) is related to persistency in sleep 395 problems (Adair et al., 1991; Burnham, Goodlin-Jones, Gaylor, & Anders, 2002; Hysing et 396 al., 2014; Mindell, Telofski, et al., 2009; Touchette et al., 2005). How the parents handle the 397 child's sleep problems that emerge during preschool age would then be expected to elicit a 398 similar pattern of parental behavior. For example, Burnham et al. (2002) suggested that the 399 parents bedtime behavior contribute negatively to the child's ability to self soothe. In this

400 perspective, we could expect dysfunctional bedtime behavior patterns in both the child and 401 their parents starting at 1.5 years to persist, and in part explain sleep problems at 2.5 and 4 402 years. By helping parents with altering their bedtime behavior early on, it is likely that the 403 child sleep problems will improve rather than persist. 404

405 The structure of maternal depressive symptoms

406 We found that the stability of maternal depressive symptoms was not related to time of 407 measurement and to followed a monotonic covariance pattern (i.e. all time points are equally 408 associated) from 1.5 to 4 years after birth. Hence, we also found autoregressive effects to be 409 non-significant. This conforms to a notion of maternal depression present at 1.5 years not 410 having an effect on maternal depression at 2.5 and 4 years, and maternal depression at 2.5 411 years not having an effect on maternal depression at 4 years. However, we did find time-412 invariant factors for maternal depression to explain 66% of the variance in symptoms of 413 maternal depression. The implication of this notion is that a reduction in time-variant factors 414 for maternal depression will only have a curative effect on that given time-point. Only a 415 reduction in time-invariant factors for maternal depression will have a curative effect on 416 maternal depression across time.

417

418 The association between child sleep problems and maternal depressive symptoms

419 Maternal depressive symptoms were associated with child sleep problems; a finding in line

420 with previous studies (Gelman & King, 2001; Goldberg et al., 2013; Martin et al., 2007;

- 421 Zuckerman et al., 1987). Furthermore, we found early child sleep problems to be more
- 422 strongly associated with later maternal depressive symptoms than early maternal depressive

423 symptoms were associated with later child sleep problems.

424 Using structural models, we estimated that the association between maternal 425 depressive symptoms and child sleep problems could be attributed to mother-driven 426 mechanisms, child-driven mechanisms, and partly to common factors. We also found child-427 driven mechanisms to be significantly stronger than mother-driven mechanisms. 428 Child-driven models of children's sleep problems and maternal depressive symptoms. 429 The findings in this study corroborates the notion that child sleep problems have an effect on 430 maternal depression, and, according to this notion, a reduction in child sleep problems could 431 lead to an improvement in maternal depressive symptoms. This finding is consistent with

432 several former findings (Hiscock et al., 2008; Lam et al., 2003; Meltzer & Mindell, 2007;

433 Moore et al., 2012) and inconsistent with others (Gelman & King, 2001; Teti & Crosby,

434 2012; Warren et al., 2006). Few of these studies did however examine this in a robust

435 manner. Although Teti and Cosby (2012) found support for the mother-driven model

436 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 modelhad the best fit to the data.

439 Child sleep problems could affect maternal depression through different mechanisms. 440 For instance, nightly parental interventions interfering with parental sleep might lead to 441 parental stress, fatigue and symptoms of depression (i.e., child-driven model) (Meltzer & 442 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 443 444 symptoms. An alternative view is that child sleep problems lead to child behavior problems, 445 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 446 447 sleep intervention program, should not only lead to improved sleep for children and their

448 parents, but also to an improvement in the parents psychological well-being (i.e., fewer

449 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.

456 *Mother-driven models of children's sleep problems and maternal depressive*

457 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).

464 Maternal depression could have an effect on child sleep problems through a range of 465 different mechanisms. How negative cognitions and emotions could affect child sleep 466 problems mediated through different parent bedtime and nighttime behaviors are two 467 mechanisms that have been paid close attention to. Mothers with depressive symptoms are 468 more likely to have negative cognitions about setting limits for their children, increased 469 doubts regarding parenting competence and worry more often about infants sleep (Teti & 470 Crosby, 2012). These mothers more easily perceive themselves as insensitive, neglectful or 471 even abusive towards the child if they are not highly involved at bedtime or night time, and 472 even tends to more often interpret their children crying as a sign of anxiety or distress

(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,

480 Telofski, et al., 2009; Teti et al., 2010).

481 In addition, Morrell (1999) claims that these negative cognitions often are related to 482 strong emotions such as guilt, shame, and anger that serve as a negative reinforcement for 483 any change in parenting behavior. Theoretically, emotionally availability measured by 484 sensitivity, structuring, non-intrusiveness and non-hostility will promote feelings of safety 485 and security in children. The ability to feel safe in one's sleep environment is essential to the 486 ability to feel relaxed and achieve deep sleep (Dahl & El-Sheikh, 2007). The emotional 487 availability in the mothers at bedtime has indeed shown to be related to children's sleep 488 problems, even more than the parental bedtime and nighttime behavior (Teti et al., 2010). It 489 could seem less important what mothers do compared to how they do it.

490 Common factors for child sleep problems and maternal depressive symptoms. 491 We found the covariance between common factors for change in child sleep problems and 492 change in maternal depressive symptoms to be negative at 2.5 years and non-significant at 4 493 years. These residual associations are indicative of third variables that account for variation 494 in both maternal depression and child sleep problems. These common factors seem to 495 account for some of the covariation between maternal depression and child sleep. Future 496 studies should aim to replicate these findings using longitudinal extended children of twin 497 and sibling designs estimating shared household factors.

498

499 Strengths and limitations of the study

500 It is important to acknowledge that inferences taken in this article rest on underlying 501 assumptions that are necessary to identify the models presented. The most important 502 assumption is that the association between child sleep problems and maternal depressive 503 symptoms is represented by a process that is qualitatively operative throughout the course of 504 this study (Hamaker & Wichers, 2017). Although this is undoubtedly a strong assumption in 505 a study of child development, this assumption is vital to the model of reciprocal effect. Future 506 studies should aim to have a larger number of measurement points across a shorter period of 507 time. It is assumed in models of reciprocal effect that variables of interest are measured 508 without measurement error. The excellent reliability of the depressive symptom measure and 509 the use of latent child sleep problems variables is therefore a significant strength of the 510 current study. The use of maternal reports of child sleeping problems represents a limitation 511 of the study. Future studies should apply objective measures of child sleep, such as actigrahy. 512 Last, and importantly, the models we have applied are only approximations to a more 513 complex reality. Therefore inferences drawn from this study should be viewed as hypothesis 514 generative rather than absolute.

515

516 *Clinical implications*

517 It is possible to draw clinical implications according to the best fitting model. One, since 518 there is no path going from depressive symptoms at one time point to the next, we would not 519 expect improvement of mood at a single time point to have long term effect. Two, according 520 to the best fitting model, treatment of child sleep problems at a given time point would be 521 expected to improve sleep problems at a later time point. Three, child-driven processes were 522 indeed stronger than mother-driven processes. Hence, we would expect that improvement of

523 child sleep problems to lead to a curative cascade for both later child sleep problems and524 concurrent maternal depressive symptoms.

525 We believe that a reasonable approach would be to assess child sleep problems when 526 meeting mothers with depressive symptoms in the clinic. Such assessments can be done by 527 any health professional. Furthermore, having information about child sleep problems could 528 be indicative for the chronicity of maternal depression. Most important is to find efficient 529 treatments for child sleep problems. Ramchandani, Wiggs, Webb, and Stores (2000) claimed 530 in a systematic review that sedation was the most frequently used treatment for childhood 531 sleep problems. The same authors concluded that although drug treatment seemed to be 532 effective in the short term, the long-term efficacy was more uncertain. They concluded that 533 behavioral treatment was more effective in the short term and also had beneficial effects in 534 the long term. This treatment includes behavioral programs guided by a therapist, parent 535 educational groups, and self-help booklets (Ramchandani et al., 2000; Sadeh, Tikotzky, & 536 Scher, 2010). In a recent randomized controlled trail on infants (6-16 months), Gradisar et al. 537 (2016) found graduated extinction and bedtime fading to provide significant sleep benefits 538 compared to sleep education controls. Although concern has been raised concerning possible 539 stress associated with extinction-based treatments (Blunden, Thompson, & Dawson, 2011), 540 the Gradisar study found neither adverse stress responses in terms of increased cortisol levels, 541 nor any long-term effects on parent-child attachment or child emotions and behavior.

542

543 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

- 548 symptoms will be beneficial for child sleep problems, but a reduction in child sleep problems
- 549 will be even more beneficial for maternal depressive symptoms.
- 550
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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. Ψ_{11} denote time invariant variance. Θ_{11} , Θ_{22} , and Θ_{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). Ψ_{11} denote time invariant variance for depression. Θ_{22} , Θ_{33} , Θ_{55} , and Θ_{66} denote time-variant residual variance. Θ_{11} denote time-variant variance for depression at time 1. β_{54} denote autoregressive paths for sleep problems. β_{25} and β_{41} denote the reciprocal effect of depression on sleep problems and vice versa. λ denote factor loadings for the 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 Θ_{11} , Θ_{22} , and Θ_{33} in figure 1b. The figure illustrates the relative importance of

•

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; Θ_{11} in figure 1b) explain 21% of the variance in child sleep problems at 4 years through the path β_{21} * β_{32} in figure 1b. Emerging factors at 2.5 and 4 years, Θ_{22} , and Θ_{33} in figure 1b, respectively, explain the remaining variance in sleep problems at 4 years.

| Item | [Response category] | Frequency | | | | | |
|-----------------------|--|-----------|---------------|---------|--|--|--|
| | "Content of response category" | 1.5 years | 2.5 years | 4 years | | | |
| | | n = 939 | n = 781 | n = 750 | | | |
| Total sleep time | a) "Usually sleep very little" | 10.6 % | 8.4 % | 6.5 % | | | |
| | b) "Sometimes sleep very little" | 77.2 % | 83.7 % | 86.8 % | | | |
| | c) "Sleeps neither little nor much" | 8.9 % | 5.5 % | 4.6 % | | | |
| | d) "Usually sleep very much" | 3.3 % | 2.5 % | 2.1 % | | | |
| 1: Difficulties to | a) [No difficulties] | 77.0 % | 77.0 % | 72.9 % | | | |
| settle at nighttime | "Easy to get to bed and to sleep" | | | | | | |
| | b) [Moderate difficulties] | 20.8 % | 19.8 % | 22.6 % | | | |
| | "Some difficulties in settling at bedtime" | | | | | | |
| | c) [Definite difficulties] | 2.2 % | 3.2 % | 4.5 % | | | |
| | "Often takes over an hour to settle at bed time" | | | | | | |
| 2: Nocturnal | a) [No difficulties] | 40.9 % | 42.1 % | 45.0 % | | | |
| awakenings | "Hardly ever wakes at night" | | | | | | |
| | b) [Moderate difficulties] | 55.0 % | 56.1 % | 53.7 % | | | |
| | "Sometimes wakes at night" | | | | | | |
| | c) [Definite difficulties] | 4.1 % | 1.8 % | 1.2 % | | | |
| | "Frequently wakes at night and is difficult to | | | | | | |
| | settle" | | | | | | |
| 3: Co-sleeping in | a) [No difficulties] | 70.7 % | 59.8 % | 55.8 % | | | |
| response to nocturnal | "Never sleeps with parent" | | | | | | |
| awakenings | b) [Moderate difficulties] | 23.6 % | 31.5 % | 38.0 % | | | |
| | "Sometimes sleeps with parent because upset or | | | | | | |
| | doesn't want to sleep alone" | | | | | | |
| | c) [Definite difficulties] | 5.7 % | 8.7 % | 6.2 % | | | |
| | "Often sleeps with parent because upset or | | | | | | |
| | doesn't want to sleep alone" | | | | | | |

1 Table 1. Frequency of child sleeping problem indicators.

| | Factor analysis | | | | | | | |
|--|-----------------------------|----------------------------|-----------------|--|--|--|--|--|
| Item | factor loading ^a | scalar factor ^b | | | | | | |
| | | 2.5y | 4y | | | | | |
| Total sleep time | 0.44±0.037° | 1.12±0.041 | 1.22±0.047 | | | | | |
| 1: Difficulties to settle at nighttime | 0.68±0.039 | 0.99 ± 0.054 | 0.87±0.05 | | | | | |
| 2: Nocturnal awakenings | 0.61±0.041 | 1.17±0.069 | 1.21±0.082 | | | | | |
| 3: Co-sleeping in response to nocturnal awakenings | 0.82±0.043 | 0.89±0.052 | 0.98 ± 0.06 | | | | | |

3 Table 2. Confirmatory factor analysis of child sleeping problem indicators.

4 Note. ^aFactor loadings are set to equal for all time points. ^bScalar factors refers to changes in variance of

5 the latent liability response variable for the ordered categorical variable, and are fixed to unity at 1.5

6 years. ^cStandard error (all such values).

| | | Mean (SD) | C | hild sleep prob | olems | Symptoms of depression | | | | |
|-------------|------|--------------------------|-------------------|-----------------|--------|------------------------|--------|--------|--|--|
| | | | 1.5y | 2.5y | 4y | 1.5y | 2.5y | 4y | | |
| | 1.5y | 0.00 ^a (1.00) | 1.00 ^a | | | | | | | |
| Child sleep | 2.5y | 0.08 (0.93) | 0.62** | 0.87** | | | | | | |
| problems | 4y | 0.11 (0.75) | 0.36** | 0.51** | 0.56** | | | | | |
| S | 1.5y | 0.00 (1.00) | 0.24** | 0.07 | 0.24** | 1.00** | | | | |
| Symptoms of | 2.5y | -0.12** (0.96) | 0.21** | 0.06 | 0.13** | 0.63** | 0.93** | | | |
| depression | 4y | -0.18** (0.99) | 0.21** | 0.16* | 0.21** | 0.63** | 0.65** | 0.98** | | |

| 7 Table 3. Means, variances, and covariances for maternal symptoms of depression and child sleeping problems between ages 1.5 and 4 y | years. |
|---|--------|
|---|--------|

8 Note. * p < .05; ** p < .01. ^aFixed parameter. Estimates derived from a saturated model with all variances and covariances estimated. N = 956 (n

9 at t1 = 939, n at t2 = 781, and n at t3 = 750. Variances are on the diagonal, covariances below the diagonal.

| | depression→sleep | depression←sleep | CFI | RMSEA (95%CI) | $\chi^{2 a}$ | df | р | AIC |
|----------|------------------|------------------|-------|---------------------|--------------|----|------|-------|
| Model 0* | 0.09 (0.01-0.17) | 0.34 (0.18-0.49) | 0.975 | 0.033 (0.026-0.040) | | | | |
| Model 1 | 0 | 0.36 (0.21-0.51) | 0.974 | 0.034 (0.027-0.041) | 4.91 | 1 | .02 | 2.91 |
| Model 2 | 0.14 (0.07-0.21) | 0 | 0.962 | 0.041 (0.035-0.048) | 16.95 | 1 | <.01 | 14.95 |
| Model 3 | 0 | 0 | 0.956 | 0.044 (0.038-0.050) | 31.14 | 2 | <.01 | 27.14 |

Table 4. Parameter estimates from bivariate autoregressive fixed effects models on maternal depressive symptoms and child sleeping problems.

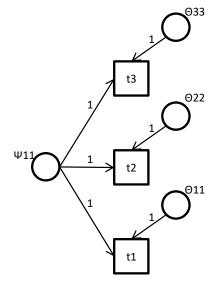
Note. *best fitting model. depression \rightarrow sleep refers to beta coefficient from mother to child (mother driven). Depression \leftarrow sleep refers to beta coefficient from child to mother (child driven). CFI = Confirmatory fit index; RMSEA = Root mean square error of approximation. AIC = Akaike's information criterion. ^aThe 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.

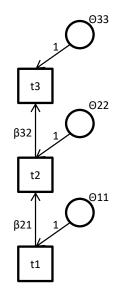
Figure 1

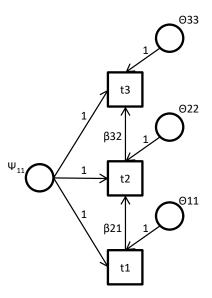
- 1 2
- a) Time-invariant fixed effect model

b) Autoregressive model

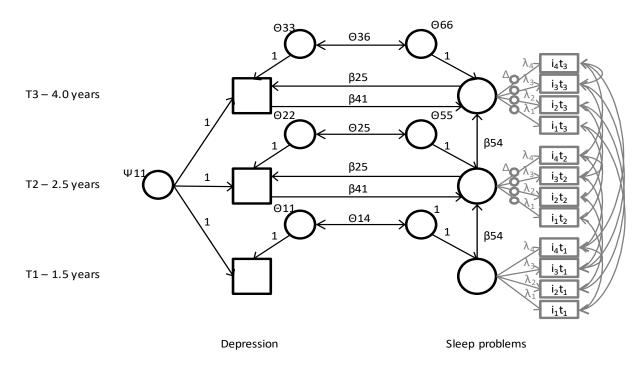
c) Autoregressive fixed effect model



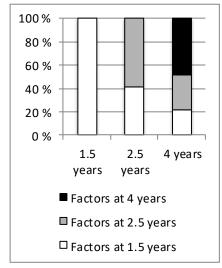




4 Figure 2



- 7 Figure 3. Explained variance in sleep
- 8 problems across time according to the best
- 9 fitting autoregressive model.



11 Appendix 1. Correlations between all items used in analyses.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
|------------------------------------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|
| 1. 1.5y* SCL-25 | 1.00 | | | | | | | | | | | | | | |
| 2. 2.5y SCL-25 | .66 | 1.00 | | | | | | | | | | | | | |
| 3. 4y SCL-25 | .64 | .68 | 1.00 | | | | | | | | | | | | |
| 4. 1.5y Total sleep time | .08 | .09 | .13 | 1.00 | | | | | | | | | | | |
| 5. 1.5y Sleep latency | .20 | .20 | .19 | .35 | 1.00 | | | | | | | | | | |
| 6. 1.5y Night awakenings | .18 | .16 | .12 | .26 | .40 | 1.00 | | | | | | | | | |
| 7. 1.5y Dislike of sleeping alone | .14 | .12 | .13 | .35 | .55 | .73 | 1.00 | | | | | | | | |
| 8. 2.5y Total sleep time | .04 | 02 | .11 | .46 | .23 | .19 | .21 | 1.00 | | | | | | | |
| 9. 2.5y Sleep latency | .10 | .15 | .18 | .20 | .47 | .25 | .34 | .31 | 1.00 | | | | | | |
| 10. 2.5y Night awakenings | .06 | .04 | .08 | .15 | .22 | .48 | .37 | .31 | .38 | 1.00 | | | | | |
| 11. 2.5y Dislike of sleeping alone | .01 | .00 | .08 | .20 | .25 | .38 | .48 | .28 | .45 | .72 | 1.00 | | | | |
| 12. 4y Total sleep time | .15 | .04 | .10 | .34 | .20 | .02 | .15 | .66 | .32 | .22 | .14 | 1.00 | | | |
| 13. 4y Sleep latency | .19 | .14 | .18 | .07 | .26 | .08 | .15 | .08 | .43 | .12 | .14 | .29 | 1.00 | | |
| 14. 4y Night awakenings | .18 | .09 | .14 | .11 | .17 | .24 | .20 | .09 | .18 | .44 | .36 | .18 | .23 | 1.00 | |
| 15. 4y Dislike of sleeping alone | .15 | .10 | .14 | .15 | .22 | .24 | .33 | .14 | .24 | .39 | .50 | .24 | .24 | .79 | 1.00 |

- 12 Note. SCL-25 is the Hopkins Symptom Checklist. *y=years. Correlations between symptoms of depression are product-moment correlations.
- 13 Correlations between symptoms of depression and indicators of child sleep problems are polyserial correlations. Correlations between indicators
- 14 of child sleep problems are polychoric correlations.