Maternal symptoms of anxiety and depression and child nocturnal awakenings at 6 and 18 months

Running title: Anxiety, depression and child sleep

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Abstract

Objective. We aim to estimate the pathways between maternal symptoms of anxiety and depression and child nocturnal awakenings via structural equation modeling using a sibling design.

Methods. Structural equation modeling on data from 14,926 sibling dyads or triads from the Norwegian Mother and Child Cohort Study.

Results. At 6 months, we estimated the association between maternal symptoms of anxiety and child nocturnal awakenings to be due to several non-significant pathways. Child nocturnal awakenings at 18 months, however, were influenced by concurrent maternal symptoms of anxiety ($\beta = 0.10$) and depression ($\beta = 0.12$). Neither maternal symptoms of anxiety ($\beta = 0.04$) nor depression ($\beta = -0.00$) were influenced by concurrent child nocturnal awakenings.

Conclusions. Our findings suggest that maternal mental health influence child sleep behavior at 18 months after birth, and not vice versa. This is in support of hypotheses on maternal mental health influencing child sleep during toddlerhood.

Child nocturnal awakenings are frequent in infants and toddlers, and are moderately stable throughout this developmental period (Byars, Yolton, Rausch, Lanphear, & Beebe, 2012; Hysing et al., 2014). Maternal mental health, such as symptoms of anxiety and depression, has consistently been associated with child nocturnal awakenings (Bayer, Hiscock, Hampton, & Wake, 2007; Teti & Crosby, 2012; Warren, Howe, Simmens, & Dahl, 2006). The associations can be explained in at least three ways.

First, a "mother-driven" model proposes that maternal symptoms of anxiety and depression have an effect on child nocturnal awakenings (El-Sheikh, Kelly, Bagley, & Wetter, 2012). There are indeed previous studies that have conceptualized the associated between maternal anxiety and depressive symptoms and child general sleep problems to be due to a "mother-driven" mechanism. Such a mechanism is not necessarily directly due to maternal mental health, but could be mediated through several maternal behaviors (Gelman & King, 2001; Teti & Crosby, 2012; Warren et al., 2006). It is conceivable that depressed mothers are less sensitive, engage in fewer positive interactions with their children, and have more negative affect compared to mothers without depression (Cornish et al., 2006; Cox, Puckering, Pound, & Mills, 1987; Tiffany Field, 1995; Webster-Stratton & Hammond, 1988; Weinberg & Tronick, 1998). It is also conceivable that depressed mothers have less joy in parenting, see themselves as more unfit parents, and use more ineffective parental strategies compared to mothers without depression (Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Susman, Trickett, Iannotti, Hollenbeck, & Zahn-Waxler, 1985; Teti & Gelfand, 1991; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990).

Second, a "child-driven" model proposes that child nocturnal awakenings have an effect on maternal symptoms of anxiety and depression (Teti & Crosby, 2012). Such a mechanism could be because parents of children with many nocturnal awakenings have fewer hours of sleep, and as a consequence of this get symptoms of fatigue, distress, and depression

(Lam, Hiscock, & Wake, 2003; Meltzer & Mindell, 2007; Moore, Gordon, & McLean, 2012). Reducing the child's nocturnal awakenings and sleep problems by intervention could hence improve the mental health of the parents. There are indeed intervention studies implicating this (Harriet Hiscock et al., 2014; Harriet Hiscock & Wake, 2002; Lam et al., 2003; Leeson, Barbour, Romaniuk, & Warr, 1994; Mindell, Telofski, Wiegand, & Kurtz, 2009).

Third, a hypothesis of common underlying factors, e.g. genetic factors or contextual stressors, proposes that common factors could provide risk for both maternal symptoms of anxiety and depression and child nocturnal awakenings. One type of common factors could be contextual stressors, such as life events, family conflict, and economic hardship (El-Sheikh et al., 2012; Gelman & King, 2001; Goldberg et al., 2013). A second type of common factor could be genetic risk factors for mental health, something supported by studies on "gene-environment" correlation (McAdams et al., 2014; Scarr & McCartney, 1983). A third type of common factor could be increased levels of stress hormones in the mother during pregnancy, which then could affect the fetus. Higher levels of cortisol has been found in depressed mothers, which could explain the link early in life (Azak, Murison, Wentzel-Larsen, Smith, & Gunnar, 2013; T. Field, 2011).

The three modes of association are non-exclusive, and the child-driven and mother-driven conceptualization can be combined to form a reciprocal model where both maternal symptoms of anxiety and depression and child nocturnal awakenings jointly affect each other.

The gold standard for causal inferences is the classical experiment, in which we obtain contrafactual information through randomization of the participants. That is, we conclude on what would happen if a given subject is exposed and not exposed at the same time. In our case, we would need to randomize mothers into having a depressive episode, something that would not be possible, or ethically allowable, to do. Instead, we have to rely on contrafactual

information from natural experiments. One such natural experiment is the sibling study (Donovan & Susser, 2011). Single birth siblings growing up in the same family can at a given biological age be discordant for exposures in their proximal environment that vary across time. This makes it possible to study to what extent they are discordant on both the exposure (i.e. maternal symptoms of anxiety and depression) and the outcome of interest (i.e. child nocturnal awakenings).

In the present study we used data from The Norwegian mother and child cohort study (MoBa), a longitudinal population-based birth cohort study which includes information from women participating with up to three children, to explore the direction of causation between maternal mental health and child nocturnal awakenings at 6 and 18 months of age. Specifically, we aim to test whether maternal symptoms of anxiety and depression have an effect on child nocturnal awakenings in a "mother-driven" process, child nocturnal awakenings have an effect on symptoms of anxiety and depression in a "child-driven" process, or whether they are associated due to common factors. Understanding etiology is crucial for both prevention and treatment. For example, if the direction of effect is predominantly from maternal mental health problems to sleep problems in the child, it would be important for health personnel to take a broad perspective when addressing sleep problems in this age group. Mental health assessment and interventions to improve mental health could be the first step, before specifically addressing infant/toddlers sleep. Similarly, if the opposite direction is stronger, it might be beneficial to focus on sleep problems in infants/toddlers instead of maternal anxiety and depression. Our rationale for estimating the direction of effect between maternal mental health and child nocturnal awakenings is thus to infer how interventions might be hypothesized to cascade from mother to child or child to mother.

Methods

Sample

The present study draws from the Norwegian Mother and Child Cohort Study (MoBa) (http://www.fhi.no/morogbarn) (Magnus et al., 2016). In brief, MoBa is a prospective, population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health. Participants were recruited from throughout Norway between June 1, 1999, and December 31, 2008, and 38.5% of the invited women consented to participate. The cohort included 108 000 children and 90 700 mothers. Self-report questionnaires were sent to the mothers at 17 weeks of gestation, and when their children were 6 and 18 months old. Response rates during pregnancy were 91-95%, and 84.8% and 72.4% at 6 and 18 months, respectively. Since the MoBa is an ongoing study, new data files are delivered regularly. The current study is based on version 6 of these quality-assured data files. For the current study to allow for sibling design, we only used data from those women that had participated with two or three pregnancies and had responded to the 6 and/or the 18 months questionnaire. The eligible sample of mothers participating several times with two or three children in the study was 14 926. Within this sample, number of children from pregnancy 1, 2, and 3 was 14 531, 12 577, and 863, respectively. Compare to the families who had complete data for analyses at 6 and 18 months, the families with data on only 6 or 18 months had no lower age (age in years) (odds ratio (OR) = 1.00; 95%CI 0.98-1.02), no more likely to have a boy (OR=1.02; 95%CI 0.84-1.25) or to be multiparous (OR = 1.21; 95%CI 0.98-1.50). Since age and parity is confounded with birth order, we based these analyses on the first pregnancy of the study.

Written informed consent was obtained from all MoBa participants upon recruitment. The study was approved by the Regional Committee for Medical Research Ethics in South-Eastern Norway.

Measures

Maternal anxiety depression

Maternal symptoms of anxiety and depression were measured at ages 6 and 18 months with eight items, four items for anxiety and four items for depression, from the Hopkins Symptom Checklist (SCL-8) (Tambs & Moum, 1993). We used confirmatory factor analyses (CFA) of the SCL-8 at 6 and 18 months in the structural equation model to reduce measurement error. In separate unidimensional CFAs, the fit to the data was as follows for maternal symptoms of for maternal symptoms of anxiety 6 months (RMSEA = 0.066, 95%CI 0.059-0.073; CFI = 0.991), depression 6 months (RMSEA = 0.040, 95%CI 0.033-0.048; CFI = 0.997), anxiety 18 months (RMSEA = 0.057, 95%CI 0.049-0.065; CFI = 0.995), and depression 18 months (RMSEA = 0.036, 95%CI 0.029-0.045; CFI = 0.998). The standardized factor loadings for maternal symptoms of anxiety were 0.87, 0.88, 0.71, and 0.86. The standardized factor loadings for maternal symptoms of depression were 0.85, 0.84, 0.76, and 0.78.

Child nocturnal awakenings

To assess nocturnal awakenings (6 and 18 months), we used a single question "How often does your child wake up nowadays?" Response categories were "3 or more times per night", "1-2 times per night", "Several times a week", and "Seldom or never".

Statistical methods

The variance-covariance matrix of our two variables (i.e. maternal mental health and child nocturnal awakenings) at two time points can be decomposed into variance-covariance within siblings, comprising all factors specific for each child, (C-factors in figure 1) and variance between siblings, comprising factors shared between children of the same mother (M-factors in figure 1). We used structural equation modeling to model these factors and the covariance between them (figure 1). Since siblings share in average only 50% of their genes, individual-specific factors also comprise effects of genes not shared with siblings for any heritable trait.

The mother-driven model, where symptoms of anxiety and depression have an effect on child nocturnal awakenings, is modeled by path b21 and b43 in figure 1. The child-driven model, where child nocturnal awakenings have an effect on symptoms of anxiety and depression is modeled by path b12 and b34. The third mode of association, where symptoms of anxiety and depression and child nocturnal awakenings are associated because of common factors is modeled by COV^C (child-specific factors) and COV^M (familial factors) in figure 1. For maternal mental health, the familial factors are better understood as factors contributing to stability across pregnancies in the mother (i.e. the M-factors are familial for the children, but not for the mother). It is an assumption in direction of causation modeling that the variables in questions are measured with little random measurement error. Therefore we used confirmatory factor analysis on the SCL-8 items to construct latent measures of anxiety and depression. By using a two-level structural equation model for the sibling data across the two time points, we estimated the factor loading for nocturnal awakenings to be 0.76. In further analyses, we modeled latent measures of nocturnal awakenings fixing the reliability to this value.

We ran separate analyses for maternal symptoms of anxiety and maternal symptoms of depression. Analyses were computed in Mplus version 7.0 and estimated using the robust weighted least squares (WLSMV) estimator (Muthén & Muthén, 2012). Both symptoms of

anxiety and depression and nocturnal awakenings were entered as observed ordinal (ordered categorical) variables. We did all analyses on raw data. Because of non-significant negative variances in the emerging familial factors for symptoms of anxiety and depression at 18 months, we had to fix the m_{33} and related covariance $cov^{M}18_{m}$ to zero for model identification.

Results

At the first pregnancy entered into the study, the mothers were 28.6 years old, and 76.8%, 18.5%, 3.8%, 0.7%, and 0.25% of the mothers had zero, one, two, three, or four or more previous childbirths, respectively. The children in the study were 51.5% boys. The correlation between the latent measures of symptoms of anxiety and depression were 0.89 at 6 months and 0.87 at 18 months.

We fitted the structural equation model on maternal symptoms of anxiety (RMSEA = 0.013, 95%CI 0.012-0.013; CFI = 0.984), and child nocturnal awakenings and symptoms of depression and child nocturnal awakenings (RMSEA = 0.017, 95%CI 0.016-0.017; CFI = 0.981) to the data. The factor loadings for the child-specific and familial factors, or variance components, are presented in table 1. We found that nocturnal awakenings were highly familial; factors shared between siblings in the same family (i.e. m22, m42, and m44, in table 1 and figure 1) were more important for nocturnal awakenings than factors specific for a child within a family (i.e. c22, c42, and c44, in table 1 and figure 1).

At 6 months after birth, the association between maternal symptoms of anxiety and child nocturnal awakenings could not be explained by one single causal pathway, but was estimated to be distributed across several non-significant paths (table 2b). At 18 months, however, maternal symptoms of anxiety were estimated to have an effect on child nocturnal

awakenings (b43, table 2b). The pathway from child nocturnal awakenings to maternal symptoms of anxiety was not significant (b34, table 2b). Common child-specific factors contributed negatively to the association between maternal symptoms of anxiety and child nocturnal awakenings (covC18m, table 2b).

The association between maternal symptoms of depression and child nocturnal awakenings at 18 months, was due to maternal symptoms of depression having an effect on child nocturnal awakenings (b43, table 2a). There was evidence of child-specific confounding factors for maternal symptoms of depression and child nocturnal awakenings at 18 months that contributed negatively to the covariance (covC18m). There was indeed no indication of common familial confounding factors at either of the two time points (covM6m and covM18m).

Discussion

We found support for a "mother-driven" hypothesis, i.e. maternally related processes generating associations between maternal symptoms of anxiety and symptoms of depression and child nocturnal awakenings at 18 months after birth, and no indication of "child-driven" processes at that time-point. We found evidence for neither child-driven nor mother-driven processes at 6 months after birth. We did indeed identify one form of common factor: Child-specific factors for maternal anxiety and child nocturnal awakenings were negatively associated at 18 months after birth.

According to our findings, an increase in maternal symptoms of anxiety or depression could lead to an increase in concurrent child nocturnal awakenings, and is in accordance with maternal symptoms of anxiety and depression having effect on child nocturnal awakenings. As an examples of clinical significance of the effect sizes found, a beta of 0.12 corresponds to

an odds ratio of 1.73 and a beta of 1.10 to an odds ratio 1.67 (assuming prevalences of 5%). These findings are consistent with some earlier literature in the field (Gelman & King, 2001; Teti & Crosby, 2012; Warren et al., 2006), but contrary to other studies (Armstrong, Van Haeringen, Dadds, & Cash, 1998; Bayer et al., 2007; Fleming, Ruble, Flett, & Shaul, 1988; H. Hiscock, Bayer, Hampton, Ukoumunne, & Wake, 2008; Harriet Hiscock et al., 2014; Harriet Hiscock & Wake, 2002; Lam et al., 2003; Leeson et al., 1994; Meltzer & Mindell, 2007; Mindell, Meltzer, Carskadon, & Chervin, 2009; Moore et al., 2012). Maternal anxiety and depression could cause interrupted sleep through a range of mechanisms (Teti & Crosby, 2012). Mothers with symptoms of depression could be prone to have negative cognitions with regard to setting limits for their child, have reduced perceived competence in parental skills, and be more worried about the toddler's sleep (Fleming et al., 1988; Tikotzky & Sadeh, 2009; Zahn-Waxler et al., 1990). Consequently, it is hypothesized that these negative cognitions could affect maternal bedtime and nighttime behavior and possibly end in sequence of events such as putting the child to sleep late, using an inconsistent bedtime routine, increase maternal presence at bedtime, or bed sharing during night. It is thinkable that these behaviors could interfere with the development of self-soothing skills in the child, as bedtime interactions with parents are very rewarding and provide positive feedback, which in turn can prolong dependence on parents (Adair, Bauchner, Philipp, Levenson, & Zuckerman, 1991). The association between frequent nocturnal awakening and parental behavior including presence at bed time and high parental nocturnal involvement is indeed a persistent finding in the literature (Adair et al., 1991; Mindell, Meltzer, et al., 2009; Teti, Kim, Mayer, & Countermine, 2010; Tikotzky & Sadeh, 2009), and this association is also highlighted as the most direct and immediate pathway in one of the leading theoretical frameworks (Sadeh, Tikotzky, & Scher, 2010). The significant effect of maternal depression on child sleep problems at 18 months, but not at six months, may suggest developmentally specific

associations. Sensitive periods for environmental influence of childhood sleep problems in these age groups have been demonstrated previously (Touchette et al., 2013). Future studies should explore if putative causal factors are stable from early to late childhood.

In a recent study we found early sleep problems to predict later development of emotional and behavioral problems (Sivertsen et al., 2015). According to children-of-twin studies, there is indeed evidence for concurrent maternal depression being a putative cause for internalizing problems, and possibly for externalizing problems (Gjerde et al., 2017; McAdams et al., 2014), but there is to date a lack of studies on children below school age. Given these prior findings, and that they interpolate to younger children, it could both be the case that nocturnal awakenings is a mediator on the causal pathway between maternal depression and child internalizing and externalizing problems, or that child internalizing and externalizing problems are mediators on the causal pathway between maternal symptoms of depression and disrupted child sleep. Future longitudinal family studies on children below school age should investigate this.

Our findings do not corroborate a child-driven conceptualization of the association between maternal mental health and child nocturnal awakenings. This is inconsistent with former hypotheses stated in the literature (Armstrong et al., 1998; Bayer et al., 2007; H. Hiscock et al., 2008; Harriet Hiscock et al., 2014; Harriet Hiscock & Wake, 2002; Lam et al., 2003; Meltzer & Mindell, 2007; Moore et al., 2012; Warren et al., 2006) but consistent with others (Gelman & King, 2001; Teti & Crosby, 2012; Warren et al., 2006). This could be because these studies did not test both the mother versus child-driven hypotheses with a design where they two hypotheses could be distinguished. One notable exception is Teti and Cosby (2012) who found support for the mother-driven model compared to the child-driven model using a mediation approach. Our null finding needs replication, given that are several intervention studies supporting a child-driven conceptualization (Harriet Hiscock et al., 2014;

Harriet Hiscock & Wake, 2002; Lam et al., 2003; Leeson et al., 1994; Mindell, Telofski, et al., 2009).

We found evidence for common child-specific confounding factors for maternal symptoms of anxiety and child nocturnal awakenings at 18 months after birth, but these factors contributed to a negative association. This is, to the best of our knowledge, the first time the covariances of such factors have been estimated. Given the lack of prior studies, it would be speculative to point out the content of these factors.

The present findings should be interpreted in light of at least seven limitations. First, individuals with low educational level, young age, and smokers are under-represented in the sample (Nilsen et al., 2009), which may limit the generalizability of the current findings. It has, however, previously been shown that even though estimates of frequencies and means may be biased due to selective participation, exposure-outcome-associations do not differ between MoBa-participants and the general population (Nilsen et al., 2009). By using Monte Carlo simulation studies, Gustavson et al. have demonstrated that association estimates are generally quite robust against selective participation when the frequency estimates are biased (Gustavson & Borren, 2014; Gustavson, von Soest, Karevold, & Roysamb, 2012). Second, it was beyond the scope of this article to include any measured common factors (e.g. marital problems) or putative mediators for the association (e.g. child internalizing problems). Third, the assessment of sleep problems was limited to a single item on parental reported nocturnal awakenings, and the results cannot be generalized to other sleep difficulties, e.g. sleep onset problems/difficulty initiating sleep. Fourth, nocturnal awakenings were measured with the time period set to 'nowadays'. Such an aggregate over a short period of time could lead to loss of information on night-to-night variability. Fifth, information on mental health and sleep problems were based on mothers' reports, and mono-informant may thus have affected the results. For example, it may be that mental health problems are related to the *perception* of

infant sleep problems, not only the frequency of awakenings per se. Sixth, by design our study only included women with two or more children. This could reduce the generalizability to mothers who only have one child. Seventh, fixing the emerging familial factors for symptoms of anxiety and depression at 18 months to zero to secure model identification assumes that those factors stable across the longer period of pregnancies for a mother (e.g. genetic factors for depression) are entirely stable across the shorter period of 6 to 18 months within a pregnancy. If such factors exists, we would not be able to capture them in our current models.

Given the exemplified increased odds of 1.73 and 1.67 for anxiety and depression using a 5% prevalence, our findings could have clinical implications. In our view maternal symptoms of anxiety and depression can be screened for in women at risk. Such women could be mothers where a health worker are aware of previous episodes of internalizing disorders. Our results also indicates that such a screening of the mother could be one aspect of intervention selection in children with sleep problems.

Future studies should aim to measure possible effect mediators, assess maternal mental health and child nocturnal awakenings repetitively across shorter time spans using more objective measures of nocturnal awakenings, and use a children and twin design on the parent level to assess modes of intergenerational transmission of risk for indicators of sleep problems.

We found the association between maternal symptoms of anxiety and depression and child nocturnal awakenings at 18 months to be explained by a mother-driven process. Accordingly, interventions targeting symptoms of maternal symptoms of anxiety and depression could be an effective strategy to improve child sleep problems in this age group.

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Table 1.

	Estimate	
Parameter in figure 1	Depression	Anxiety
c11	0.58**	0.53**
m11	0.82**	0.85**
c22	0.52**	0.52**
m22	0.85**	0.85**
c31	-0.01	-0.02
m31	0.84**	0.86**
c33	0.55**	0.51**
m33	0.00a	0.00a
c42	0.35**	0.34**
m42	0.48**	0.48**
c44	0.44**	0.45**
m44	0.67**	0.67**

** p<.01; a fixed parameter.

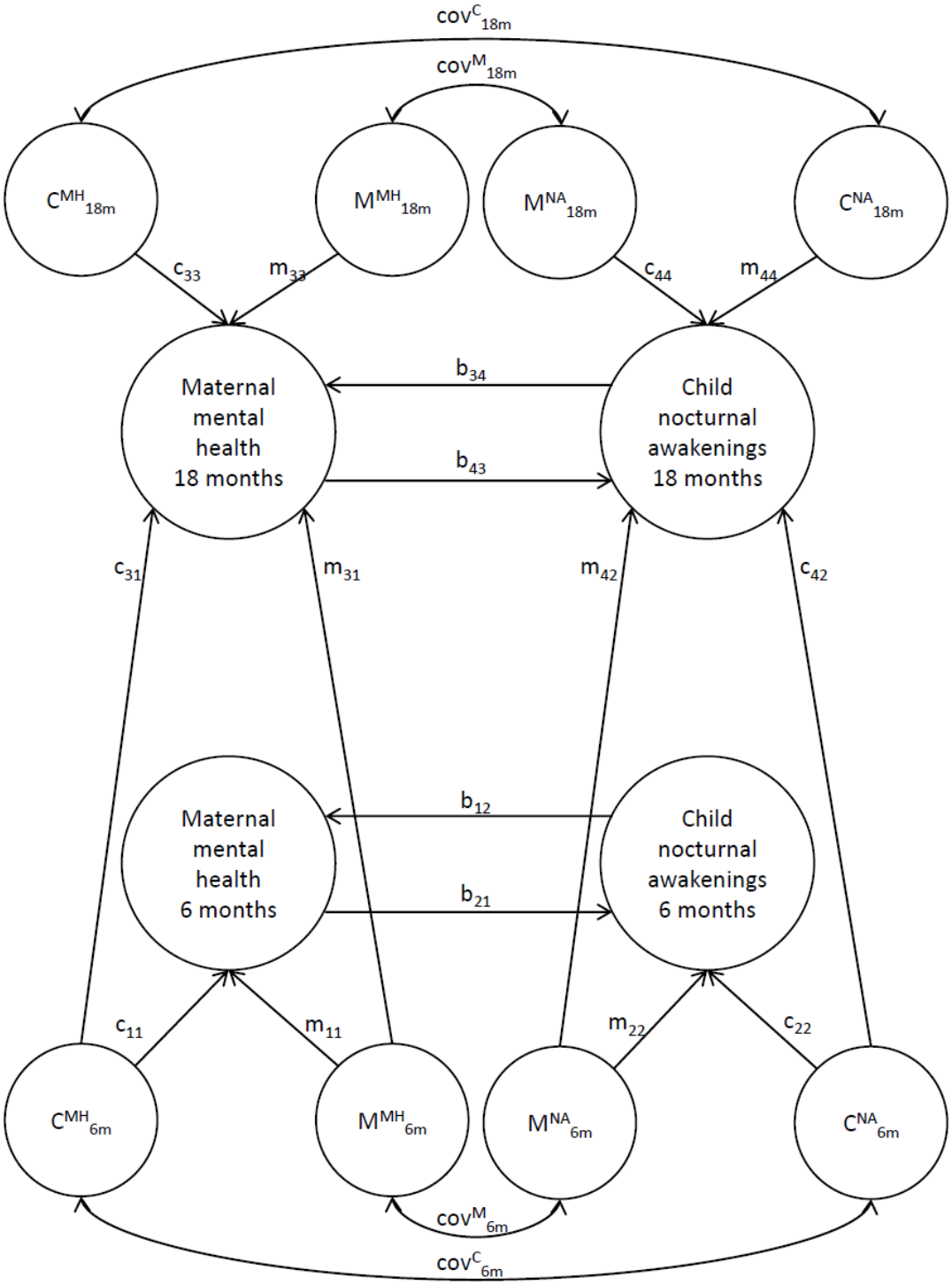
Table 2a. Associations between maternal depression and child nocturnal awakenings.

	Parameter in figure 1		*B (95% CI)*	*p*
Age 6 months	b21	Maternal depression have effect on nocturnal awakenings	0.03 (-0.03; 0.09)	.33
	b12	Nocturnal awakenings have effect on maternal depression	0.00 (-0.05; 0.06)	.89
	covC6m	Covariance child-specific risk factors	0.06 (-0.05; 0.16)	.28
	covM6m	Covariance mother-specific risk factors	0.01 (-0.03; 0.04)	.66
Age 18 months	b43	Maternal depression have effect on nocturnal awakenings	0.12 (0.06; 0.17)	.00
	b34	Nocturnal awakenings have effect on maternal depression	-0.00 (-0.07; 0.05)	.98
	covC18m	Covariance child-specific risk factors	-0.08 (-0.23; 0.07)	.28
	covM18m	Covariance mother-specific risk factors	0.00a	-

Table 2b. Associations between maternal anxiety and child nocturnal awakenings.

	Parameter in figure 1		*B (95% CI)*	*p*
Age 6 months	b21	Maternal anxiety have effect on nocturnal awakenings	-0.03 (-0.09; 0.04)	.40
	b12	Nocturnal awakenings have effect on maternal anxiety	0.03 (-0.04; 0.09)	.37
	covC6m	Covariance child-specific risk factors	-0.01 (-0.14; 0.12)	.90
	covM6m	Covariance mother-specific risk factors	0.00 (-0.04; 0.04)	.94
Age 18 months	b43	Maternal anxiety have effect on nocturnal awakenings	0.10 (0.04; 0.16)	.00
	b34	Nocturnal awakenings have effect on maternal anxiety	0.04 (-0.04; 0.11)	.37
	covC18m	Covariance child-specific risk factors	-0.21 (-0.38; -0.03)	.02
	covM18m	Covariance mother-specific risk factors	0.00a	-

a Fixed parameter.


cov C 18m
cov M 18m
C MH 18m
M MH 18m
M NA 18m
C NA 18m
c33
m33
c44
m44
Maternal mental health 18 months
Child nocturnal awakenings 18 months
b34
b43
c31
m31
m42
c42
Maternal mental health 6 months
Child nocturnal awakenings 6 months
b12
b21
c11
m11
m22
c22
C MH 6m
M MH 6m
M NA 6m
C NA 6m
cov M 6m
cov C 6m