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2022-05

Punamäki , R-L , Tammilehto , J , Flykt , M S , Vänskä , M , Tiitinen , A , Poikkeus , P & Lindblom , J 2022 , ' Determinants of adolescent sleep : Early family environment, obstetric factors, and emotion regulation ' , Journal of Applied Developmental Psychology , vol. 80 , 101420 . https://doi.org/10.1016/j.appdev.2022.101420

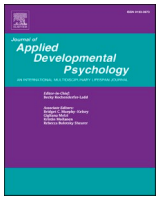
http://hdl.handle.net/10138/348262
https://doi.org/10.1016/j.appdev.2022.101420

Journal of Applied Developmental Psychology 80 (2022) 101420

Contents lists available at ScienceDirect

Journal of Applied Developmental Psychology

journal homepage: www.elsevier.com/locate/jappdp



# Determinants of adolescent sleep: Early family environment, obstetric factors, and emotion regulation

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ARTICLE INFO

*Keywords:*
Adolescent sleep quality
Marital relationship
Parenting
Maternal mental health
Obstetric factors
Emotion regulation
Infancy
Adolescence

ABSTRACT

Optimal sleep quality fosters adolescents' wellbeing and, therefore, learning about its developmental determinants is essential. We examined how early family environment (i.e., parent-reported parenting, marital relationship quality, and mothers' mental health), obstetric factors (i.e., infertility history and assisted reproductive treatments, and pre- and perinatal complications and health risks), and children's emotion regulation in middle childhood predicted adolescents' sleep quality. We also tested the mediating role of emotion regulation in linking early determinants to adolescent sleep. Finnish families ($N = 984$) participated during pregnancy, infancy, middle childhood, and late adolescence. Results showed that only early maternal mental health problems predicted poor adolescent sleep quality. Contrary to hypotheses, emotion regulation did not mediate the effects of early family environment and obstetric factors on later sleep quality. Supporting the early family environment through improving maternal mental health can have long-term positive developmental impacts, including sleep.

## Introduction

Adolescents need optimal sleep for increasingly sophisticated cognitive processes (Crone & Dahl, 2012; Steinberg, 2014) and for managing new kinds of social and emotional challenges (Lovato & Gradisar, 2014). Optimal sleep involves sufficient sleep efficiency (e.g., duration and habits) initiation and maintaining (e.g., appropriate onset and restoration), and lack of sleep disturbances (e.g., night-time or early awakenings), resulting in good daytime functioning (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; Carskadon, 2011). According to the ecological model (Bronfenbrenner, 2005), adolescent sleep is highly intertwined with multi-level psychosocial, contextual, and biological factors that should be considered to understand it comprehensively (Becker, Langberg, & Byars, 2015; El-Sheikh & Sadeh, 2015). Yet, most available research focuses only on single determinants of sleep and has not spanned across different developmental periods, leaving unanswered the question about the importance of early experiences on sleep. The current prospective study aimed to fill these gaps by investigating how both *early family environment* incorporating parenting experiences, marital relationship quality, and mothers' mental health problems, and *obstetrics factors* involving infertility history and assisted reproduction treatment (ART) and obstetric risks (i.e., pre- and perinatal complications and health problems) predict adolescent sleep quality. We conceptualize *adolescent sleep quality* broadly to consist of sleep onset latency, eveningness, sleep duration, perceived sleep quality, sleep disturbances, sleeping medication, and daytime function.

Targeting the earliest predictors of sleep is important because current evolutionary-developmental perspectives posit infancy as a possible sensitive period during which children's emotional and physiological self-regulation development is highly malleable for interpersonal experiences and biological factors (Frankenhuis & Fraley, 2017; Gee, 2020; Perry, Blair, & Sullivan, 2017). These experiences and factors shape infants' abilities to attenuate physiological arousal and to regulate emotions (Crockenberg, Leerkes, & Lekka, 2007; Morris, Silk, Steinberg, Meyers, & Robinson, 2007). These regulatory abilities, in turn, form the basis of circadian rhythm and optimal sleep patterns (Towe-Goodman, Stifter, Mills-Koonce, Granger, and The Family Life Project Key Investigatorsy, 2012). Such developmental effects can be expected to last over

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https://doi.org/10.1016/j.appdev.2022.101420
Received 20 May 2021; Received in revised form 23 April 2022; Accepted 25 April 2022
Available online 2 May 2022

multiple developmental periods from middle childhood to adolescence.

From the developmental perspective, it is also important to identify mechanisms that explain impacts of early family environment on adolescent sleep. Therefore, the current study focused on children's emotion regulation in middle childhood as one potential mediating mechanism for the developmental effects of early determinants on adolescent sleep quality. In line with the broad definition by Eisenberg, Spinrad, and Eggum (2010) and Rydell, Berlin, and Bohlin (2003), we conceptualized *children's emotion regulation* in middle childhood to involve both emotional regulation and emotional reactivity. Understanding of both multiple determinants and mediators help build targeted and more effective interventions to promote adolescent sleep.

## Early family environment and sleep quality

An optimal family environment is characterized by sensitive parenting, high marital relationship quality, and good parental mental health. Such an environment is important for shaping children's optimal sleep patterns and quality, as it creates the safety needed for sleeping (El-Sheikh & Kelly, 2017; Meltzer & Montgomery-Downs, 2011). Ample evidence indicates that secure and emotionally available early parent–child relationship is beneficial for the emergence of balanced infant circadian and homeostatic sleeping patterns (Philbrook & Teti, 2016; Sadeh, Tikotzky, & Scher, 2010), while interparental conflicts and hostility predict sleeping problems in infants and toddlers (Mannering et al., 2011). There is also evidence that maternal pre- and postnatal mental health problems, especially depression, predict children's poorer sleep quality in infancy and toddlerhood (Dias & Figueiredo, 2020; Goldberg et al., 2013). However, research on family determinants of sleep quality concerns mostly infants and toddlers, and less is known about the potential long-term effects of early experiences on later sleep development. Adolescents are at special risk for disturbed sleep (Carskadon, 2011), and thus in need of effective help. Comprehensive knowledge about early predictors of their sleep can contribute to tailoring more erudite sleep-improving intervention elements.

The available adolescent studies on sleep have typically concentrated on single family factors assessed concurrently. Regarding parenting, a study found that supportive and monitoring parenting practices predicted optimal sleep quality in early adolescence, indicated by longer sleep duration, lower eveningness, and less daytime sleepiness (Meijer, Reitz, & Deković, 2016). Restrictive, negative, and inconsistent parenting has, in turn, been associated with negative morning mood and daytime sleepiness in mid-adolescence (Brand, Hatzinger, Beck, & Holsboer-Trachsler, 2009). Regarding marital relationship quality, research shows associations of divorce and family stress (e.g., marital conflicts) with sleeping problems among adolescents and young adults (Bernert, Merrill, Braithwaite, Van Orden, & Joiner Jr, 2007; Turunen, Norell-Clarke, & Hagquist, 2021), although some studies did not find associations between family stress and adolescent sleep (Adam, Snell, & Pendry, 2007). We could find one longitudinal study spanning from infancy to adolescence, considering mother's mental health and children's sleep. Maternal postpartum depression predicted shorter sleep onset latency and frequently interrupted sleep among 16–18-year-old adolescents (Taylor et al., 2017).

Altogether, research indicates that children's sleep is sensitive to family factors, but evidence regarding prospective developmental effects from infancy to adolescence is scarce. Accordingly, the current study addressed this gap by modeling multiple salient family factors involving the quality of parenting and the marital relationship, as well as maternal mental health predicting sleep in late adolescents.

## Obstetric factors and sleep quality

We also considered potential impacts of obstetric factors on adolescent sleep quality, as relatively little is known about these associations over time. In the current study, half of the participating couples had infertility history and became parents through the assisted reproduction treatment (ART). These experiences can interfere with optimal child development through various psychosocial and medical routes. Infertility constitutes a parental crisis, and the treatments can be highly stressful, which may lead to overprotective parenting of the vulnerable child (Ilioi & Golombok, 2015) and heightened parental anxiety (Monti et al., 2015). ART may also impact children's early biological development, involving fetal epigenetic alterations (Choux et al., 2015). In line with this, compared to naturally conceived (NC) children, ART children show more somatic, mental health, and cognitive problems (Bergh & Wennerholm, 2020; Hart & Norman, 2013a, 2013b). As parenting and parents' and children's mental and somatic health problems closely associate with sleep problems (Orchard, Gregory, Gradisar, & Reynolds, 2020), one may expect ART adolescents to show problematic sleep. Yet, research is scarce on the impact of ART on sleep quality in childhood and completely lacking in adolescence. One exception was a polysomnography study among infants (6–11 weeks; $N = 100$), which revealed more periodic breathing episodes in the ART than the NC group, indicating more immature respiratory systems (Audiens et al., 1995). In addition, a registry-based study reported that ART toddlers had an increased risk of difficulties in initiating and maintaining sleep-wake schedule (Lidegaard, Pinborg, & Andersen, 2005). Research shows that sleep problems in toddlerhood predict those in middle childhood (Belmon, van Stralen, Busch, Harmsen, & Chinapaw, 2019), and analogously one may suggest them also to predict sleep problems among ART adolescents.

Pre- and perinatal obstetric risks, such as low birth weight (LBW), preterm birth, smallness for gestational age (SGA), birth complications, and newborn and maternal pre- and perinatal health problems, are important risk factors for early cardiovascular, metabolic, and endocrinological health problems (Mericq et al., 2017). These obstetric risks may also play a central role in the developmental organization of sleep patterns, as preterm birth and SGA are associated with infants' unstable circadian and homeostatic rhythms (Asaka & Takada, 2010; Hysing, Reichborn-Kjennerud, Markestad, Elgen, & Sivertsen, 2019). Indeed, two cohort studies show the predictive effects of obstetric risks on poor sleep quality in later development: A Norwegian study ($N = 216$) found that SGA predicted a two-fold risk for longer sleep onset latency (i.e., the prolonged time of falling asleep) and frequent nighttime awakenings at the children's age of 11 years (Stangenes et al., 2019). In a Finnish study ($N = 159$), preterm birth predicted longer sleep onset latency among young adults of 19–25 years (Strang-Karlsson et al., 2008). Moreover, in this same cohort, very LBW ($< 1500$ g) predicted children's poorer sleep effectiveness, indicated by longer sleep onset latency and shorter sleep duration in middle childhood (Pesonen et al., 2009). Because adolescence is a period of drastic biological, social, and regulatory changes (Crone & Dahl, 2012), we do not know whether pre-and perinatal determinants, important in earlier development, still count in later adolescence.

## Emotion regulation development and adolescent sleep

As discussed above, we also explored emotion regulation as potential mediator, serving to link early family environment and risks to adolescent sleep quality. Emotion regulation refers to a process in which a person modifies the content, intensity, and expression of their emotions in order to them match with environmental demands and serve personal goals (Gross, 2015). As such, emotion regulation is part of the larger construct of self-regulation that involves ability to generating emotional responses, flexibly modulating them, controlling behaviors and attention, and engaging in cognitive planning (Eisenberg et al., 2010; Moilanen, Padilla-Walker, & Blaacker, 2018; Rydell et al., 2003).

Research emphasizes the importance of early interpersonal relationships and family climate on the development of emotion regulation (Morris et al., 2007; Morris, Criss, Silk, & Houltberg, 2017) and its neural underpinnings (Berboth & Morawetz, 2021). Infants are highly

dependent on their caregivers to attenuate stress responses and emotional arousal, and infants develop effective emotion regulation through experiences of sensitive parental co-regulation (Gee, 2020; Tronick, 2005). At the same time, infants adapt their emotion regulation to fit the style of their caregivers (Bowlby, 1969), and the quality of parent–infant interactions are highly vulnerable to detrimental effects of parental mental health problems (Feldman et al., 2009). Moreover, infants tend to develop heightened emotional reactivity and ineffective emotion regulation strategies if the family climate is characterized by tension and interparental conflicts (Lindblom et al., 2017; Schudlich, Jessica, Erwin, & Rishor, 2019).

Regarding obstetric risks, prematurity, and LBW decrease infant soothability and hinder the maturation of early regulatory functions (Feldman, 2009; Montagna & Nosarti, 2016; Voigt et al., 2013; Yaari et al., 2018). For the parents, infants with obstetric risks place higher demands for caregiving that can increase parental stress, further interfering with children's emotion regulation development (Feldman, 2006; Poehlmann et al., 2011).

Research suggests that sleep and emotion regulation are functionally connected (Fairholme & Manber, 2015; Palmer & Alfano, 2017). On one hand, poor sleep quality can lead to emotion regulation problems. This is demonstrated, for example, by studies showing that sleep deprivation leads to altered activation in brain areas related to emotion regulation (Ma, Dinges, Basner, & Rao, 2015). On the other hand, overwhelming negative emotions, often resulting from emotion regulation problems and high emotional reactivity, can lead to poor sleep quality. This is likely caused by physiological hyperarousal involving various dysregulated endocrinological and neural processes (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Espie, 2002; Riemann et al., 2010). Despite the acknowledged reciprocal associations between emotion regulation and sleep quality, research is mainly available on the effects of sleep on emotion regulation in general and also in adolescence (Palmer & Alfano, 2017). Indeed, we could detect only two studies analyzing the impact of emotion regulation on sleep quality among adolescents. A US national cohort study showed that problems with emotion regulation (i.e., suppression and rumination) correlated with poor sleep quality (i.e., long sleep onset latency, short sleep duration, and high morningness (Palmer, Oosterhoff, Bower, Kaplow, & Alfano, 2018). A three-year follow-up study demonstrated bidirectional associations between emotion regulation and sleep quality, as problems with emotion regulation and negative emotional reactivity predicted low sleep duration and high weekend sleepiness, and these sleeping problems in turn predicted ineffective emotion regulation (Tavernier & Willoughby, 2015). In sum, these studies suggest that emotion regulation is linked with sleep quality. Ours is the first study that tests the role of emotion regulation in predicting adolescent sleep in a prospective longitudinal setting and conceptualizes it as a mediational pathway.

### The aims of the study

The primary aim of this prospective study was to test the early developmental determinants of sleep quality in late adolescence and the mediating role of emotion regulation. Fig. 1 summarizes the conceptual model incorporating multiple social, biological, and psychological determinants of sleep quality (Becker et al., 2015; El-Sheikh & Sadeh, 2015). Importantly, considering multiple early family environment determinants (i.e., parenting experiences, marital relationship quality, and maternal mental health) and obstetric factors is critical as it can reveal the relative and unique importance of each determinant on adolescent sleep. The model further suggests that later regulatory development in terms of emotion regulation in middle childhood can be crucial mediating mechanism between early family and obstetric determinants and adolescent sleep quality. It is noteworthy that our unique, multi-reporter design consisted of questionnaires filled by mothers, fathers, and adolescents, thus minimizing biases typical for single-reporter studies (e.g., only mothers) common in this field.

We hypothesized that early negative parenting experiences, low marital relationship quality, maternal mental health problems, infertility history and ART, obstetric risks, as well as children's emotion regulation problems in middle childhood directly predict adolescent poor sleep quality. Moreover, we hypothesized that the obstetric factors and early family environment also have indirect (i.e., mediated) effects on adolescent sleep through children's emotion regulation problems in middle childhood.

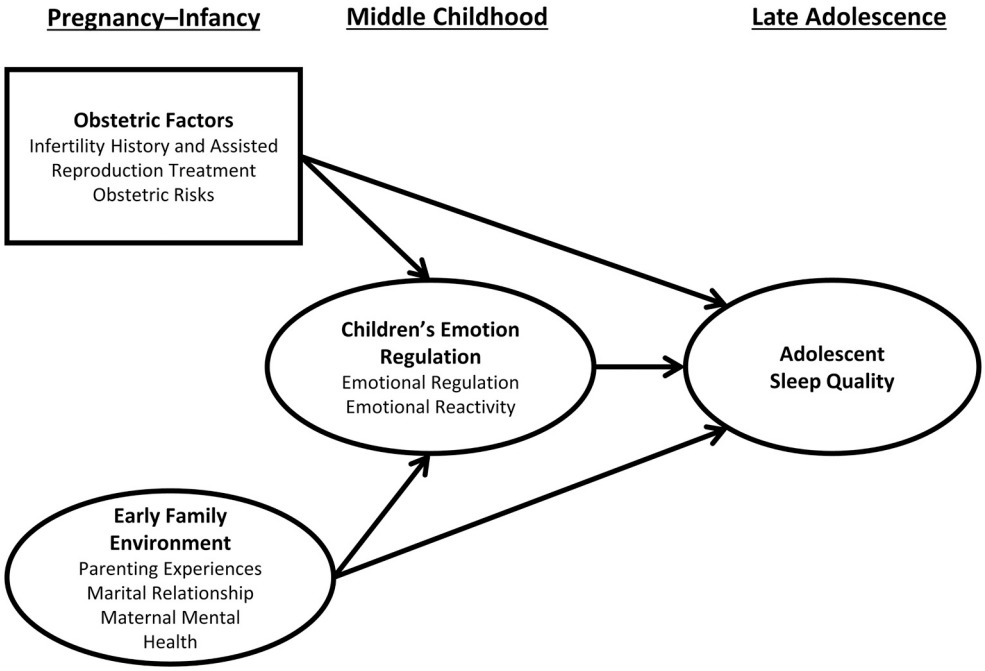


**Fig. 1..** Early Developmental Determinans of Adolescent Sleep Quality.

## Method

### Participants and procedures

The study consists of 984 Finnish families followed from pregnancy to adolescence ($M$ = 18.24, $SD$ = 0.35, range = 17–19 years). This sample involved couples with infertility history who had conceived with ART ($n$ = 484) and naturally conceiving (NC) couples ($n$ = 469), who were recruited at the Helsinki University Central Hospital, or via other infertility clinics for a subset of ART couples (in 31 couples, ART status was missing). The primary inclusion criteria for all couples was fluent Finnish language. For the NC group, additional inclusion criteria were no history of infertility and maternal age ≥ 25 years, in order to correspond with the higher age of mothers in the ART group (ART women $M_{age}$ = 33.2, $SD$ = 4.4; NC women $M_{age}$ = 33.3, $SD$ = 3.0). In the present study, we used the data of mothers' reports ($n$ = 953) during pregnancy (T1), mothers' ($n$ = 663) and fathers' ($n$ = 617) reports at children's age of 2 months (T2), mothers' ($n$ = 546) and fathers' ($n$ = 507) reports at children's age of 12 months (T3), mothers' ($n$ = 535) and fathers' ($n$ = 317) reports at children's age of 7–8 years (T4), and adolescents' reports ($n$ = 449) at age of 17–19 years.

At T1, researchers recruited the couples during their appointment for a routine ultrasonographic scan during gestational weeks 16–18. They informed the couples about the study before the couples signed informed consent documents. After that, mothers and fathers separately filled in the questionnaires. At T2 and T3, a nurse first confirmed parents' willingness to participate in the follow-up and if they agreed, she mailed separate questionnaires to both parents. At T4, researchers approached both parents separately by mail containing informed consent document and questionnaire, and those willing to participate returned both document and questionnaire concerning themselves and the target child by a prepaid envelope. At T5, researchers approached both parents and their adolescent separately by mailed letters informing about the follow-up study. If they were interested, researchers asked them to return their informed consent document in a prepaid envelope. After that, adolescents, mothers, and fathers filled out e-form questionnaires. All the questionnaires were in Finnish, utilizing already existing translations of the questionnaires when possible. A bilingual psychologist translated the questionnaires that were not available in Finnish by using the forward-backward translation method. The researchers piloted all questionnaires before the data collection.

Attrition analysis showed that, compared to T1, fathers with lower SES and higher number of earlier marriages/cohabitation had a lower participation rate at T2 and T3 than those with higher SES and lower number of earlier marriages/cohabitation ($p$ < .05). The ART vs. NC status, parents' age, number of children, duration of the partnership, perinatal factors (i.e., preterm or full-term pregnancy, birth weight or newborn health), or maternal mental health status (depressive and anxiety symptoms or social dysfunction) were not associated with the participation. The participating mothers at T4 were biased towards older age ($M$ = 33.6, $SD$ = 3.8 vs. M = 32.8, $SD$ = 3.6, $t$ = 3.01, $p$ = .010), but the attrition at T4 was not dependent on ART vs. NC, the parents' education, the length of the marriage, number of children, and earlier partnerships.

At T5, the participation rate for adolescents was 49% ($n$ = 449; $n_{NC}$ = 223, $n_{ART}$ = 214, 13 ART status were missing). Girls were less likely to attrit than boys by adolescence, $\chi^2$ (1) = 32.60, $p$ = .001. Of the adolescents 58% ($n$ = 260) reported their gender as female, 40.5% ($n$ = 182) as male, and 2% ($n$ = 7) identified their gender as "other". Almost two-thirds (65%) lived with their both parents, 15% with their mothers, 12% in turns with each parent, and 8% alone. Three quarters (75.5%) studied currently in high school, 14% were in vocational school, and the rest were working or unemployed. At T5, most of the families had a monthly household income between 5000 and 10,000 €. A fourth (24.5%) of the parents had divorced. The families were typically small, with 60% reporting having one or two children.

The project complies with the Code of Ethics of the World Medical Association (WMA-DOH, Declaration of Helsinki 1964–2014), and the Ethical Board of Helsinki University Central Hospital approved the project at early data collections T1–T3 (# HUC050/98 and # HUC 050b/99), T4 (# 220/2006/ 365/E9/06), and T5 (# HUCH/1566/ 2017).

### Measures

#### Obstetric Risks at T1 and T2

Data on obstetric risks of *pregnancy complications*, *birth-related risks, new-born health problems, and maternal perinatal health problems* were from medical records and mothers' own retrospective reports at T2. All obstetric variables were calculated by summing across items and then further standardizing (i.e., subtracting the mean score from a raw score and then dividing this difference by the standard deviation) to form the *Z*-score cumulative obstetric risk index (Ettekal, Eiden, Nickerson, & Schuetze, 2019) that was used in the main analyses (see below). The low value indicated low level and high value indicated high level of obstetric risks.

*Pregnancy complications* included six physician-determined problems: contractions, bleeding, pre-eclampsia (i.e., high blood pressure and protein in blood), high blood glucose, liver problems, or other complications (0 = *no*, 1 = *yes*). *Birth-related risks* involved four risk conditions: length and initiation of labor (1 ≤ *37 h and spontaneous initiation*, 2 ≥ *37 h and assisted initiation,* e.g., *vacuum extraction delivery*, 3 = *operational initiation,* e.g., *Cesarean section*), pain treatment (i.e., use of seven possible analgesia methods, such as epidural anesthetic, scored as 1 = *none*, 2 = *1–2 methods*, 3 = *3–7 methods*), loss of blood (1 = *low < 500 ml,* 2 = *middle 500–1000 ml*, 3 = *high > 1000 ml*), and post-delivery operations (i.e., use of four possible operations, such as placental abruption; scored as 1 = *none*, 2 = *1–2 operations*, 3 = *3–4 operations*). *Newborn health problems* included six indicators: health status (1 = *very good,* 2 = *good,* 3 = *poor*), 1 min Apgar-scores (scored as 1 = *very good, 9–10 points,* 2 = *good, 6–8 points,* 3 = *poor, 1–5 points*), head circumference (scored as 1 = *normative, 32.00–36.99 cm,* 2 = *low, < 32.00 cm,* 3 = *high, ≥ 37 cm*), birth weight (scored as 1 = *normative, ≥ 2.500 g*, 2 = *LBW, < 2.500 g*), gestational maturity (scored as 1 = *normative,* ≥ 37 *weeks*, 2 ≤ 37 *weeks*), and need for treatment (1 = *no,* 2 = *monitoring,* 3 = *need for intensive care*). *Maternal perinatal health problems* involve four gynecological problems: uterus inflammation, breast inflammation, *episiotomy* wound inflammation, and other post-delivery operation (0 = *no,* 1 = *yes).*

After forming the standardized variables for the four obstetric risks, we calculated the average score of these risk variables that was used in the main analyses. This Z-score cumulative risk index is a standard approach for forming cumulative risk scores for continuous scales (Ettekal et al., 2019). It enabled us to use a weighted continuous risk score for our obstetric risks that were assessed with different scales.

#### Negative parenting experiences at T2 and T3

Parenting experiences were assessed by Abidin's (1997) Parenting Stress Index (PSI; short form of 35 items) at T2 and T3. At both times, the parenting experiences consist of parent, interaction, and child domains. The *parent domain* represents the resources and limitations that parents experience (e.g., "I feel in trouble with the parental responsibilities"), the *interaction domain* represents the emotionally laden dyadic relationship (e.g., "My child smiles at me much less than I expected"), and the *child domain* represents characteristics of the child that makes them seem difficult to parent (e.g., "My child cries and gets nervous much easier than most children"). Both mothers and fathers estimated how well the descriptions fit their experiences on a 5-point Likert scale (1 = *completely agree* to 5 = *completely disagree*). Before analyses, we reversed the PSI scales so that high values indicated more negative parenting experiences. At each wave, each parent's mean scores of the parent, interaction, and child domain subscales were aggregated through

averaging, resulting in four scores that were the indicators of the latent negative parenting experiences variable in the analyses. These corresponded to negative mothering experiences at T2 (Cronbach's α = 0.91), negative fathering experiences at T2 (Cronbach's α = 0.92), negative mothering experiences at T3 (Cronbach's α = 0.91), and negative fathering experiences at T3 (Cronbach's α = 0.92).

*Marital relationship quality at T2 and T3*

The Dyadic Adjustment Scale (DAS; Spanier, 1976) has 32 items and covers four domains of the couple relationships. They are dyadic satisfaction (e.g., "Do you ever regret that you married/or lived together?"; "Do you kiss your partner?"), dyadic consensus (e.g., "To what extent do you agree on … handling family finances; philosophy of life; sex relations"), dyadic cohesion (e.g., "Have a stimulating exchange of ideas"; "Laugh together"; "Work together a project"), and dyadic affectional expression (e.g., "Being too tired for sex"; "Not showing love"). Both parents used 6-point Likert scales to evaluate how often the couple engages in different behaviors and interactions, from 0 (*Never*) to 5 (*All the time*), or to what extent they agree on issues, from 0 (*Always disagree)* to 5 (*Always agree)*. Two items had dichotomous scale and two items ranged from 0 (*Nothing to share*) to 4 (*Everything shared*). These items were rescaled to correspond with a majority score calculation. For mothers and fathers at each measurement wave (i.e., T2 and T3), an average score was formed using all 32 items. This resulted in four average scores of mother's marital adjustment at T2 (Cronbach's α = 0.91), father's marital adjustment at T2 (Cronbach's α = 0.91), mother's marital adjustment at T3 (Cronbach's α = 0.89), and father's marital adjustment at T3 (Cronbach's α = 0.88). These four variables were used as the indicators of the latent marital relationship quality variable in the analyses.

*Maternal mental health problems at T2 and T3*

*Depressive symptoms* were assessed at T2 and T3 by the Beck's Depression Inventory BDI-13 (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), assessing low mood, hopelessness, and dissatisfaction. Sample items are, for example, "I am so sad and unhappy that I can't stand it"; "I feel that I am completely failure as person"; "I feel that the future is hopeless and that things cannot improve". Mothers reported on a 4-point Likert scale (0 = *symptom not present* to 3 = *symptom present most of the time*). Average variables for mothers' BDI-13 were constructed at T2 (Cronbach's α = 0.77) and T3 (Cronbach's α = 0.83). *Psychological distress* was measured by the 36-item General Health Questionnaire (GHQ-36; Goldberg & Hillier, 1979), covering depression (e.g., "Been feeling that life is not worth living"; "Been thinking of yourself as a worthless person") anxiety ("Been feeling constantly under strain"; "Been getting scared or panicky for no reason"), social dysfunction (e.g., Reversed "Been feeling capable of making decisions about things"; "Been able to enjoy your normal day-to-day activities"), and somatic symptoms (e.g., Been getting a feeling of tightness or pressure in your head"; Been getting any pains in your head"). Mothers reported how the symptom descriptions matched them over the past few weeks on a 4-point Likert scale (1 = *not at all* to 4 = *much more than usually*). The average total scores of mothers' GHQ-36 were calculated for T2 and T3, and Cronbach's alphas were 0.92 and 0.93, respectively. The four scales (i.e., BDI and GHQ at T2 and T3) were used as the indicators of the latent maternal mental health variable.

*Children's emotion regulation problems at T4*

The Emotion Questionnaire (EQ; Rydell et al., 2003) measures emotional regulation and emotional reactivity with parent-report of 40-items related to child's negative emotions of sadness, anger, and fear, and positive emotions of exuberance. In addition, the questionnaire includes emotions of shame and guilt as they are developmentally relevant in middle childhood (Lindblom et al., 2017). For each emotion, six statements reflected the child's emotional regulation, referring to abilities to regulate emotions with the assistance of others (e.g., "When my child is angry, it is easy for others, for instance, a parent, to calm them down") and by themselves (e.g., "When my child is angry, they have difficulties calming down on their own"). For each emotion, four statements reflected the child's emotional reactivity; that is, the frequency of emotional responses (e.g., "My child often becomes angry and falls in a bad mood") and their intensity (e.g., "When my child is forbidden to do something they want to do, they react strongly and intensely"). Using a 5-point Likert scale, mothers and fathers separately rated how well the statements described their child (0 = *does not fit my child at all* to 4 = *fits my child very well*). Average scores were separately calculated for each parent's reports of the child's emotional regulation and emotional reactivity, with higher values indicating more emotion regulation problems. Thus, mothers' estimation of emotional regulation at T4 (Cronbach's α = 0.88), fathers' estimation of emotional regulation at T4 (Cronbach's α = 0.88), mothers' estimation of emotional reactivity at T4 (Cronbach's α = 0.85), and fathers' estimation of emotional reactivity at T4 (Cronbach's α = 0.84) were used as the indicators of the latent emotion regulation problems variable. These four variables were used as the indicators of the latent child emotion regulation problems variable in the analyses.

*Adolescent sleep quality at T5*

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a self-report questionnaire with seven subscales pertaining sleep features, disturbances, and daytime dysfunction. The reference frame is during the last month. First, sleep onset latency was measured by one open question: "How many minutes does it usually take you to fall to sleep?" Adolescents reported the onset in minutes that is the score used in the analyses. Yet, a linear transformation (dividing by 10) was applied to the sleep onset latency subscale to correspond its variance to other sleep variables. Second, eveningness was measured by one open question: "When have you usually gone to bed?". Adolescents reported the time of going to bed and those time-scores are used in the analyses. Third, sleep duration was indicated by one question: "How many hours of actual sleep did you get?". Adolescents provided the estimated duration in hours that is the score in the analyses. Fourth, perceived sleep quality was asked by one question: "How would you rate your sleep overall?". Adolescents estimated the quality using a 4-point Likert-scale (1 = *very good;* 2 = *pretty good,* 3 = *bad,* 4 = *very bad*), which was the variable used in analysis. Fifth, sleep disturbances were measured via a nine-item questionnaire (i.e., waking up in the middle of the night or early morning, getting up to use the bathroom, difficulties in breathing, coughing and snoring, feeling too cold or hot, having nightmares, having pains, disorientation when waking up, and other sleep disturbances). Adolescents estimated using a 4-point Likert-scale how often they have had each sleep disturbance (0 = *not during the last month,* 1 = *less than once a week*, 2 = *once or twice a week*, 3 = *three or more times a week*). A sum variable was constructed and used in the analysis. Sixth, the use of sleeping medication was inquired by one question ("How often have you taken medicine to help you sleep?"), and adolescents responded by a 4-point Likert-scale (1 = *not during the last month,* 2 = *less than once a week*, 3 = *once or twice a week*, 4 = *three or more times a week*) which variable was used in analysis. Seventh, two questions measured daytime dysfunction: For the first question ("How often have you had trouble staying awake while driving, eating meals, or engaging in social activity?"), adolescents responded using the same 4-point Likert-scale as for sleeping medication. To the second question ("How much of a problem has it been for you to keep up enough enthusiasm to get things done?"), adolescents responded using a 4-point Likert-scale (1 = *no problem at all,* 2 = *some problems,* 3 = *a big problem,* 4 = *a very big problem*). A sum variable was constructed for daytime dysfunction and was used in analysis. Before analyses, all scales except sleep duration were reversed so that high values indicated better sleep quality. The seven PSQI subscales were used as the indicators of the latent adolescent sleep quality variable.

*Analytic strategy*

We conducted the analyses with SPSS 25.0 and the *lavaan* package (Rosseel, 2012) in R 4.0.2. We used confirmatory factor analysis (CFA) to test the measurement model of study variables and structural equation modeling (SEM) to model the hypothesized relationships between early family environment, obstetric factors, and children's emotional regulation in middle childhood, and adolescent sleep quality. In both, we used for estimators the maximum likelihood estimation with robust standard errors and the Yuan-Bentler scaled tests statistic (MLR). The missing completely at random (MCAR) assumption was not met, Little's MCAR $\chi^2$ (1526) = 1718.32, $p < .001$, indicating that the missingness of some variables depended on other variables. Therefore, in CFA and SEM, we handled missing data with Full Information Maximum Likelihood that assumes that the data meet the missing at random (MAR) assumption. The R script is available at https://osf.io/5p3v4/.

The measurement model of study variables involved five latent variables: negative parenting experiences (two indicators from mothers and two from fathers), marital relationship quality (two indicators from mothers and two from fathers), maternal mental health problems (four indicators from mothers), children's emotional regulation problems in middle childhood (two indicators from mothers and two from fathers), and adolescent sleep quality (seven indicators from adolescents). In SEM, we tested the hypothesized direct effects of parenting experiences, marital relationship quality, maternal mental problems, infertility history and ART, and obstetric risks on adolescent sleep quality as well as their indirect effects on sleep quality via children's emotional regulation problems in middle childhood. The infertility history and ART (0 = *no*, 1 = *yes*) and obstetric risks were handled as observed variables, whereas other constructs were handled as latent variables. Infants' sex (1 = *girl*, 0 = *boy*) was a covariate for emotion regulation problems in middle childhood and adolescent sleep quality. The model fit was indicated by the scaled $\chi^2$ test and approximate fit indices of robust comparative fit index (CFI) > 0.90, robust root-mean-square error of approximation (RMSEA) < 0.06, and standardized root mean square residual (SRMR) < 0.08 (Brosseau-Liard & Savalei, 2014).

We made an a priori decision to use the alpha level of 0.050 for unstandardized effects as a statistical criterion of the detected direct effects. We used the 95% confidence interval (CI) that excluded zero as a criterion of detected indirect effects. We computed the standard errors for the MLR-based indirect effects by using the delta method for the Sobel test. To assess the robustness of the results concerning the MLR-based indirect effects, we also tested the indirect effects using bootstrapped 95% CIs with 5000 bootstraps. Finally, we reported the standardized estimates for the direct effects and *R*-squares to indicate effect sizes.

## Results

*Descriptive analyses*

Table 1 presents the descriptive statistics of all variables used in analyses. The correlations between these variables can be found in the Supplementary Table 1 (S1).

*Measurement model of study variables*

Regarding the measurement model, first, we conducted a CFA for the original model in which each indicator loaded on its latent variable, and all latent variables were specified to correlate with each other. This model showed poor model fit, $\chi^2$ (220, $N$ = 795) = 1311.70, $p < .001$, CFI = 0.774, RMSEA = 0.080, 90% CI [0.076 0.085], SRMR = 0.080. As a result, we modified the original measurement model based on theoretical considerations and modification indices. First, we included two orthogonal common method factors in the model for both fathers' and mothers' reports. This allowed us to separate the variance stemming

**Table 1**
Descriptive statistics.

| Variable | *n* | *M* | *SD* | Range | Skewness | Kurtosis |
|---|---|---|---|---|---|---|
| Infertility history and ART | 953 | 0.51 | 0.50 | 0.00–1.00 | −0.03 | −2.00 |
| Pregnancy complications | 653 | 0.90 | 0.94 | 0.00–5.00 | 1.16 | 1.66 |
| Birth-related risks | 649 | 1.76 | 0.42 | 1.00–3.00 | 0.56 | −0.14 |
| Newborn health | 603 | 6.95 | 1.57 | 6.00–14.00 | 2.19 | 5.05 |
| Maternal postnatal health problems | 642 | 1.17 | 0.40 | 1.00–3.00 | 2.15 | 3.75 |
| Average of scaled obstetric risks | 654 | 0.00 | 0.61 | −1.39–2.87 | 1.20 | 2.12 |
| Negative mothering experiences at T2 | 654 | 1.64 | 0.39 | 1.00–3.64 | 0.98 | 1.91 |
| Negative fathering experiences at T2 | 617 | 1.64 | 0.39 | 1.00–2.97 | 0.70 | 0.16 |
| Negative mothering experiences at T3 | 545 | 1.59 | 0.37 | 1.00–3.17 | 1.08 | 1.39 |
| Negative fathering experiences at T3 | 506 | 1.59 | 0.37 | 1.00–3.14 | 0.87 | 0.71 |
| Mothers' marital adjustment at T2 | 655 | 3.75 | 0.45 | 1.71–4.81 | −0.78 | 1.41 |
| Fathers' marital adjustment at T2 | 616 | 3.79 | 0.42 | 1.47–4.84 | −0.72 | 4.10 |
| Mothers' marital adjustment at T3 | 543 | 3.67 | 0.57 | 0.61–4.78 | −1.53 | 4.10 |
| Fathers' marital adjustment at T3 | 504 | 3.77 | 0.46 | 1.69–4.94 | −0.78 | 1.51 |
| BDI at T2 | 657 | 0.53 | 0.30 | 0.00–2.23 | 1.21 | 3.04 |
| GHQ at T2 | 656 | 1.63 | 0.31 | 1.06–3.47 | 1.30 | 2.74 |
| BDI at T3 | 546 | 0.56 | 0.36 | 0.00–2.54 | 1.47 | 3.23 |
| GHQ at T3 | 544 | 1.60 | 0.34 | 1.06–3.56 | 1.77 | 4.35 |
| Mothers' estimation of emotional regulation at T4 | 535 | 2.12 | 0.64 | 1.00–4.44 | 0.35 | 0.03 |
| Fathers' estimation of emotional regulation at T4 | 317 | 2.16 | 0.60 | 1.00–4.44 | 0.23 | 0.18 |
| Mothers' estimation of emotional reactivity at T4 | 535 | 2.83 | 0.65 | 1.00–4.75 | 0.09 | −0.28 |
| Fathers' estimation of emotional reactivity at T4 | 317 | 2.75 | 0.59 | 1.08–4,25 | 0.17 | −0.25 |
| Sleep onset latency at T5 | 449 | 2.12 | 2.04 | 0.00–18.00 | 3.39 | 16.23 |
| Eveningness at T5 | 446 | 23.58 | 1.24 | 21.00–29.50 | 1.32 | 3.17 |
| Sleep duration at T5 | 442 | 7.50 | 1.12 | 4.00–12.00 | 0.24 | 0.75 |
| Perceived sleep quality at T5 | 447 | 1.96 | 0.61 | 1.00–4.00 | 0.20 | 0.32 |
| Sleep disturbances at T5 | 428 | 4.57 | 3.56 | 0.00–22.00 | 1.09 | 1.73 |
| Sleep medication at T5 | 438 | 1.25 | 0.71 | 1.00–4.00 | 2.96 | 7.84 |
| Daytime dysfunction at T5 | 447 | 4.08 | 1.31 | 2.00–8.00 | 0.44 | −0.15 |
| Children's sex | 806 | 0.50 | 0.50 | 0.00–1.00 | −0.02 | −2.00 |

*Note.* For all analyses, Sleep onset latency at T5, Eveningness at T5, Perceived sleep quality at T5, Sleep disturbances at T5, Sleep medication at T5, and Daytime dysfunction at T5 were reversed so that high values indicated better sleep quality. ART = assisted reproduction treatment, BDI = shortened version of Beck Depression Inventory; GHQ = General Health Questionnaire; T2 = children's two months of age; T3 = children's 12 months of age; T4 = middle childhood; T5 = late adolescence.

from the data source (e.g., father's response biases) from the variance of the target construct (e.g., child's emotion regulation problems). The six father-reported indicators were set to load on the fathers' latent method factor, whereas the ten mother-reported indicators were, in turn, set to load on the mothers' latent method factor. Second, on the basis of the modification indices, we estimated one error covariance between sleep duration and sleep disturbances to reflect unique shared variance with extremely high sleep duration and high sleep disturbances. This modified measurement model showed adequate global fit, $\chi^2$ (203, $N = 795$) = 561.66, $p < .001$, CFI = 0.930, RMSEA = 0.047, 90% CI [0.042, 0.051], SRMR = 0.054. Therefore, we decided to use this model in further analyses.

The standardized factor loadings and estimated correlations of the measurement model are shown in Table 2. In general, indicators showed moderate to strong loadings on their expected latent variables. The most notable exception was eveningness, which had a weak loading on the latent adolescent sleep quality factor. The loadings of the fathers' method factor ranged from moderate to strong. By contrast, all but one of the loadings of the mothers' method factor were non-significant and generally showed smaller effect sizes than the loadings of the fathers' method factor, indicating that mothers' method factor operated clearly worse than fathers in capturing the mothers' response style. Nevertheless, the comparison between models with and without the mothers' method factor showed that the inclusion of this factor provided strong improvement in the model fit, $\Delta\chi^2$ (10) = 125.41, $p < .001$. This emphasized the importance of including the distinct method factors for both parents as a parsimonious and theoretically justified solution to improve the global model fit. At the same time, however, the statistical structure of the mothers' method factor was not as clear as the fathers' method factor at the local level (i.e., single factor loadings).

### *Effects on children's emotion regulation problems and adolescent sleep quality*

The SEM model involving both the direct effects of the early family environment, obstetric factors, and children's emotion regulation in middle childhood on adolescent sleep quality and the indirect effects of the early family environment and obstetric factors on adolescent sleep quality through children's emotion regulation in middle childhood, showed adequate model fit, $\chi^2$ (257, $N = 984$) = 648.02, $p < .001$, CFI = 0.925; RMSEA = 0.039, 90% CI [0.035, 0.043], SRMR = 0.053. Table 3 shows the unstandardized parameter estimates for the direct and indirect effects, and Fig. 2 illustrates the standardized estimates.

As hypothesized, early maternal mental health problems predicted poorer adolescent sleep quality. However, contrary to hypotheses, there were no direct effects of parenting experiences, marital relationship quality, infertility history and ART and obstetric risks on adolescent sleep quality (*ps* $\geq$ 0.053). It should be noted, however, that the standardized parameter of the negative parenting experiences effect on adolescent sleep quality was detected with the alpha level of 0.050, $\beta$ = 0.31, *SE* = 0.15, $p = .044$. Yet, as (a) the difference in *p*-values between standardized and unstandardized estimates reflects a statistical artefact and (b) the direction of this effect was unintuitive in light of the current literature, we interpreted this effect as not detected.

The results failed to substantiate the hypotheses about the indirect effects of parenting experiences, marital relationship quality, maternal mental health, infertility history and ART, and obstetric risks on adolescent sleep quality through children's emotion regulation problems in middle childhood. Both the MLR-based (all 95% CIs contained zero, *ps* $\geq$ 0.563) and bootstrapped parameter estimates (all 95% CIs contained zero, *ps* $\geq$ 0.620) were nonsignificant. Finally, negative parenting experiences predicted children's greater emotion regulation problems in middle childhood. However, contrary to hypotheses, marital relationship quality, maternal mental health, infertility history and ART, and obstetric risks were not associated with children's emotion regulation problems in middle childhood. Regarding children's sex, girls

**Table 2**
Standardized loadings and correlations of measurement model.

| Variable | λ | *SE* | *p* |
|---|---|---|---|
| Negative parenting experiences | | | |
| Negative mothering experiences at T2 | 0.80 | 0.06 | < 0.001 |
| Negative fathering experiences at T2 | 0.38 | 0.04 | < 0.001 |
| Negative mothering experiences at T3 | 0.86 | 0.04 | < 0.001 |
| Negative fathering experiences at T3 | 0.38 | 0.05 | < 0.001 |
| Marital relationship quality | | | |
| Mothers' marital adjustment at T2 | 0.82 | 0.02 | < 0.001 |
| Fathers' marital adjustment at T2 | 0.64 | 0.04 | < 0.001 |
| Mothers' marital adjustment at T3 | 0.88 | 0.04 | < 0.001 |
| Fathers' marital adjustment at T3 | 0.72 | 0.03 | < 0.001 |
| Maternal mental health problems | | | |
| BDI at T2 | 0.85 | 0.09 | < 0.001 |
| GHQ at T2 | 0.75 | 0.05 | < 0.001 |
| BDI at T3 | 0.86 | 0.06 | < 0.001 |
| GHQ at T3 | 0.78 | 0.11 | < 0.001 |
| Children's emotion regulation problems | | | |
| Mothers' estimation of emotional regulation at T4 | 0.67 | 0.04 | < 0.001 |
| Fathers' estimation of emotional regulation at T4 | 0.62 | 0.06 | < 0.001 |
| Mothers' estimation of emotional reactivity at T4 | 0.75 | 0.04 | < 0.001 |
| Fathers' estimation of emotional reactivity at T4 | 0.70 | 0.05 | < 0.001 |
| Adolescent sleep quality | | | |
| Sleep onset latency at T5 | 0.44 | 0.05 | < 0.001 |
| Eveningness at T5 | 0.18 | 0.07 | 0.013 |
| Sleep duration at T5 | 0.48 | 0.06 | < 0.001 |
| Perceived sleep quality at T5 | 0.63 | 0.04 | < 0.001 |
| Sleep disturbances at T5 | 0.62 | 0.06 | < 0.001 |
| Sleep medication at T5 | 0.33 | 0.07 | < 0.001 |
| Daytime dysfunction at T5 | 0.53 | 0.04 | < 0.001 |
| Fathers' method factor | | | |
| Negative fathering experiences at T2 | 0.65 | 0.04 | < 0.001 |
| Negative fathering experiences at T3 | 0.76 | 0.04 | < 0.001 |
| Fathers' marital adjustment at T2 | −0.41 | 0.06 | < 0.001 |
| Fathers' marital adjustment at T3 | −0.44 | 0.04 | < 0.001 |
| Fathers' estimation of emotional regulation at T4 | 0.26 | 0.06 | < 0.001 |
| Fathers' estimation of emotional reactivity at T4 | 0.33 | 0.05 | < 0.001 |
| Mothers' method factor | | | |
| Negative mothering experiences at T2 | 0.23 | 0.22 | 0.291 |

(*continued on next page*)

**Table 2** (*continued*)

| Variable | λ | *SE* | *p* |
|---|---|---|---|
| Negative mothering experiences at T3 | −0.11 | 0.20 | 0.560 |
| Mothers' marital adjustment at T2 | 0.00 | 0.09 | 0.970 |
| Mothers' marital adjustment at T3 | 0.22 | 0.11 | 0.037 |
| BDI at T2 | 0.31 | 0.19 | 0.105 |
| GHQ at T2 | 0.19 | 0.24 | 0.437 |
| BDI at T3 | −0.26 | 0.21 | 0.227 |
| GHQ at T3 | −0.45 | 0.24 | 0.063 |
| Mothers' estimation of emotional regulation at T4 | −0.08 | 0.14 | 0.582 |
| Mothers' estimation of emotional reactivity at T4 | 0.01 | 0.09 | 0.895 |
| Correlations | ρ | *SE* | *p* |
| Adolescent Sleep Quality ↔ Negative Parenting Experiences | 0.04 | 0.06 | 0.543 |
| Adolescent Sleep Quality ↔ Marital Relationship Quality | 0.05 | 0.07 | 0.496 |
| Adolescent Sleep Quality ↔ Maternal Mental Health Problems | −0.09 | 0.07 | 0.216 |
| Adolescent Sleep Quality ↔ Children's Emotion Regulation Problems | −0.03 | 0.08 | 0.679 |
| Negative Parenting Experiences ↔ Marital Relationship Quality | −0.56 | 0.05 | < 0.001 |
| Negative Parenting Experiences ↔ Maternal Mental Health Problems | 0.82 | 0.03 | < 0.001 |
| Negative Parenting Experiences ↔ Children's Emotion Regulation Problems | 0.33 | 0.06 | < 0.001 |
| Marital Relationship Quality ↔ Maternal Mental Health Problems | −0.62 | 0.05 | < 0.001 |
| Marital Relationship Quality ↔ Children's Emotion Regulation Problems | −0.18 | 0.08 | 0.014 |
| Maternal Mental Health Problems ↔ Children's Emotion Regulation Problems | 0.26 | 0.06 | < 0.001 |
| Sleep duration at T5 ↔ Sleep disturbances at T5 | −0.45 | 0.07 | < 0.001 |

*Note.* Sleep onset latency, Eveningness, Perceived sleep quality, Sleep disturbances, Sleep medication, and Daytime dysfunction at T5 were reversed. BDI = shortened version of Beck Depression Inventory; GHQ = General Health Questionnaire; T2 = children's two months of age; T3 = children's 12 months of age; T4 = middle childhood; T5 = late adolescence.

had poorer sleep quality than boys. In contrast, children's sex did not predict children's emotion regulation problems in middle childhood. The hypothesized model explained 12% of the variation in adolescent sleep quality and 11% of the variation in children's emotion regulation problems in middle childhood.

*Additional analyses*

Finally, it is notable that, from the theoretical perspective, early determinants could have also shown predictive value on specific sleep indicators over and above the general sleep quality. Therefore, we yet inspected whether the fit of our SEM model would have improved by estimating some effects of early determinants on specific sleep quality indicators. We did this by exploring the modification indices of our SEM model between the early determinants (i.e., infertility history and ART, obstetric risks, and latent parenting experiences, marital relationship quality, maternal mental health problems, and children's emotion regulation problems) and the specific sleep indicators (i.e., sleep onset latency, eveningness, sleep duration, perceived sleep quality, sleep disturbances, sleeping medication, and daytime dysfunction). Using the critical value of $\Delta\chi^2(1) = 3.84$ with the alpha level of 0.050, only two modification indices between (a) maternal mental health problems and daytime dysfunction ($\Delta\chi^2 = 4.67$) and (b) emotion regulation problems and sleep duration ($\Delta\chi^2 = 4.04$) slightly exceeded this value. Yet, when adding these effects into the SEM model, both showed unintuitive directions that contradicted the direction of the effects on latent adolescent sleep quality, indicating statistical artifacts (i.e., mental health problems and emotion regulation problems predicted better daytime function and higher sleep duration, respectively). Overall, as there existed 42 possible associations (six determinants and seven indicators), early determinants showed little evidence of unique associations with specific sleep indicators beyond the latent adolescent sleep quality.

## Discussion

Optimal early family environment creates the safety needed for sleeping and parenting, marital relationship, and mental health influence infant's capacity to soothe and stabilize circadian rhythm. Yet, research is lacking about comprehensive predictive role of these multiple early sleep determinants, and the developmental time frame of available research is limited. Our prospective study incorporated the social, biological, and psychological mechanisms as determinants of adolescent sleep quality (Becker et al., 2015; El-Sheikh & Sadeh, 2015), and extended developmental foci from infancy and toddlerhood into adolescence. In line with our hypothesis, the results show that maternal mental health problems during infancy predicted poor adolescent sleep quality. However, contrary to expectations, early parenting experiences,

**Table 3**
Unstandardized direct and indirect path coefficients for the effects of obstetric factors and early family environment on children's emotion regulation and adolescent sleep quality.

| Path Coefficient | *B* | *SE* | *p* |
|---|---|---|---|
| Direct effects | | | |
| Infertility History and ART → Adolescent Sleep Quality | 0.07 | 0.12 | 0.556 |
| Obstetric Risks → Adolescent Sleep Quality | −0.08 | 0.11 | 0.459 |
| Negative Parenting Experiences → Adolescent Sleep Quality | 0.33 | 0.17 | 0.053 |
| Marital Relationship Quality → Adolescent Sleep Quality | 0.03 | 0.09 | 0.738 |
| Maternal Mental Health Problems → Adolescent Sleep Quality | **−0.37** | **0.18** | **0.036** |
| Children's ER → Adolescent Sleep Quality | −0.05 | 0.08 | 0.546 |
| Children's Sex → Adolescent Sleep Quality | **−0.60** | **0.13** | **< 0.001** |
| Infertility History and ART → Children's ER problems | −0.12 | 0.11 | 0.254 |
| Obstetric Risks → Children's ER problems | −0.01 | 0.10 | 0.962 |
| Negative Parenting Experiences → Children's ER problems | **0.37** | **0.15** | **0.016** |
| Marital Relationship Quality → Children's ER problems | −0.01 | 0.10 | 0.943 |
| Maternal Mental Health Problems → Children's ER problems | −0.03 | 0.14 | 0.823 |
| Children's Sex → Children's ER problems | 0.02 | 0.11 | 0.829 |
| Indirect effects | | | |
| Infertility History and ART → Children's ER problems → Adolescent Sleep Quality | 0.01 | 0.01 | 0.592 |
| Obstetric Risks → Children's ER problems → Adolescent Sleep Quality | 0.00 | 0.01 | 0.961 |
| Negative Parenting Experiences → Children's ER problems → Adolescent Sleep Quality | −0.02 | 0.03 | 0.563 |
| Marital Relationship Quality → Children's ER problems → Adolescent Sleep Quality | 0.00 | 0.01 | 0.944 |
| Maternal Mental Health Problems → Children's ER problems → Adolescent Sleep Quality | 0.00 | 0.01 | 0.838 |

*Note.* ART = Assisted Reproduction Treatment, ER = Emotion Regulation. In bolded values, $p < .050$.

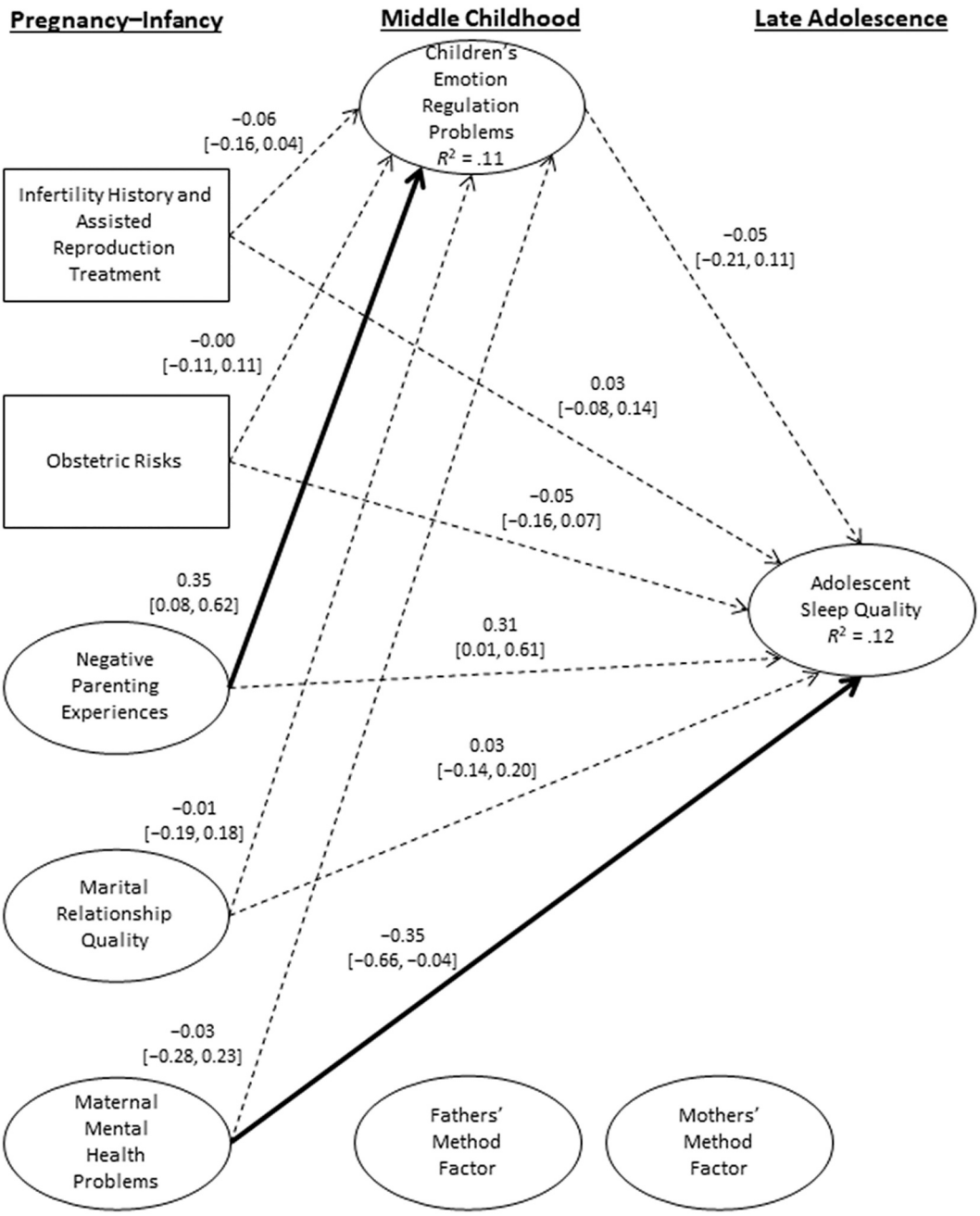


**Fig. 2.** Standardized Estimates and 95% CIs for the Effects of Obstetric Factors and Early Family Environment on Children's Emotion Regulation and Adolescent Sleep Quality.

marital relationship quality, and obstetric factors (i.e., infertility history and ART and pre- and perinatal risks) did not predict adolescent sleep. Furthermore, the results failed to support our hypothesis regarding the mediating role of children's emotion regulation in linking the early environment and later sleep. As a whole, our findings suggest that the direct effects of early life on adolescent sleep are rather modest and are limited to maternal mental health. Yet, as there are no earlier multi-determinant prospective studies on sleep reaching from pregnancy to late adolescence, some of the null findings can be considered important, such as obstetric factors and marriage relationship. Future studies should therefore involve more dynamic models recognizing developmental complexities, such as age-specific vulnerabilities and strengths, malleability of developmental processes, and unique child susceptibilities in explaining adolescent sleep.

*Early maternal mental health predicts adolescent sleep*

Maternal postpartum depression has a well-documented long-term negative impact on child development, including adolescent mental health and social relationships (Hymas & Girard, 2019; Parsons, Young, Rochat, Kringelbach, & Stein, 2012). Yet, only one previous longitudinal study has indicated that maternal postpartum depression impacts the development of offspring sleeping problems in adolescence (Taylor et al., 2017). Thus, our finding contributes to the scarce literature by showing that early maternal mental health problems, including her depression, can have a long-term negative impact on adolescent sleep quality.

There are various plausible explanations for the detrimental effects of maternal mental health on adolescent sleep. First, heritable genetic factors may partially explain the effects. Maternal depression, anxiety, and

sleeping problems are highly comorbid in the pre- and postnatal period (Dørheim, Bjorvatn, & Eberhard-Gran, 2014), suggesting shared genetic mechanisms. Further, studies have demonstrated a genetic pathway that transmits psychopathology from mothers to their offspring over and above early parental stress (Rice et al., 2010), and a genetic link has been identified between psychopathology and circadian rhythms (i.e., eveningness; Toomey, Panizzon, Kremen, Franz, & Lyons, 2015). The second explanation may be related to fetal programming: Mothers with postpartum mental health problems frequently have symptoms already during the pregnancy, and mothers' prenatal stress can negatively affect offspring regulatory development (Glover, 2011), potentially including the development of sleep (Palagini, Drake, Gehrman, Meerlo, & Riemann, 2015).

Third, social factors and parenting practices can be important in explaining the negative impact of maternal mental health on adolescent sleep. Mothers with mental health problems may have poor understanding of infant's sleep-related needs. They have been found to have biased cognitions about the preconditions of optimal infant sleep and unrealistic expectations of age-normative behavior (Tikotzky & Shaashua, 2012). Thus, mothers with mental health problems may have difficulties providing an organized and appropriately scheduled environment that would support their children's sleep development. Fourth, mothers with mental health problems may be less effective in co-regulating the infant's emotional arousal (Feldman et al., 2009; Tronick, 2005). For example, depressive mothers can be intrusive or withdrawn in their bedtime involvement by being insensitive to infant's cues, showing flat vocal expressions, and touching the infant too much or too little (Seymour, Giallo, Cooklin, & Dunning, 2015). Such interactions can heighten the child's stress and leave the infant in a highly aroused state, hindering sleep and its optimal development (El-Sheikh, Kelly, Bagley, & Wetter, 2012).

Regarding these considerations, it is worth noticing that in our study, maternal mental health problems correlated significantly with negative parenting experiences (see Table 2). Despite this, only maternal mental health, and not the parenting experiences, predicted adolescent sleep. While speculative, such specificity suggests that maternal mental health may have some developmental importance on children's sleep that does not occur via the general quality of parent–child interactions. However, more research is needed to scrutinize both the genetic and prenatal biological as well as more specific relational and behavioral mechanisms. It would also be informative to analyze the specific role of parental bedtime practices in addition to more general aspects of parenting.

### *Early family relationships did not predict adolescent sleep*

Contrary to our hypotheses, early negative parenting experiences and low marital satisfaction did not predict adolescent poor sleep quality. This was surprising, as these results stand in contrast to research suggesting that sensitive parenting and a secure family environment are the core preconditions of optimal sleep quality in infancy and toddlerhood (Mannering et al., 2011; Philbrook & Teti, 2016; Sadeh et al., 2010) and in middle childhood (Cimon-Paquet, Tétreault, & Bernier, 2019; El-Sheikh, Buckhalt, Mize, & Acebo, 2006). While definite conclusions necessitate further replications, our findings raise the possibility that the primary early force influencing children's sleep development may be maternal mental health, rather than its manifestations as parental stress or marital relationship problems.

It is also important to note that the previous sleep studies are predominantly cross-sectional, whereas our study focused on the early determinants of adolescent sleep. Thus, the lack of prospective associations for parenting and the marital relationship may indicate that children's sleep development is highly responsive to later experiences and contextual influences. Indeed, for example, the development of biological stress regulation systems (e.g., vagal tone and diurnal HPA-axis), which are important for sleep, continues during toddlerhood and middle childhood (Beijers, Buitelaar, & Weerth, 2014). Relatedly, adolescence is also an important period for experience-related development, characterized especially by the extension of important social relationships and neurocognitive sophistication (Crone & Dahl, 2012; Steinberg, 2014). From this perspective, our model may have been overly simplistic in focusing solely on early determinants of sleep. The future studies should construct a model of developmental determinants of adolescent sleep to include also age salient social, biological and psychological factors in toddlerhood, middle childhood and adolescence. They should include peer and romantic relationships, cognitive and brain development, puberty timing, physical growth, and neuroendocrinological regulation.

The lack of prospective associations of parenting experience and marital relationship with adolescent sleep may also reflect the complex nature of child development. For example, according to differential susceptibility theory, children differ in their sensitivity to developmental experiences (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2011). Highly sensitive infants, indicated, for example, by negative emotionality, tend to be more malleable for both negative and positive family factors (Slagt, Dubas, Deković, & van Aken, 2016). One sleep study has demonstrated this by showing early maternal sensitivity to predict better sleep quality in middle childhood among children with high negative emotionality, while early maternal sensitivity did not play a role among children with low negative emotionality (Conway, Modrek, & Gorroochurn, 2018). Thus, it is possible that considering such moderating factors would have produced a different picture about the role of early family relationships on adolescent sleep.

### *Role of obstetric factors*

Our results suggest that ART children did not differ from NCs in their sleep quality in late adolescence. The finding is important, as our prospective study was the first to focus on the sleep quality of adolescents conceived with ART. Our results do not concur with the vulnerability hypothesis of ART children being at heightened developmental risks, for example, due to medical problems and family dynamics of overprotective parenting (Ilioi & Golombok, 2015; Pinborg et al., 2013). Instead, the results accord with our previous findings based on the same data that revealed protective resources among ART parents, such as mothers showing highly positive parenting experiences and resiliency against the stressful life events and infants' health problems (Repokari et al., 2006). Together with the positive aspects of parental investment, the intactness of ART adolescents' sleep quality speaks for optimal and adaptive development among ART children.

Our cumulative obstetric risks incorporated pregnancy-, birth-, and delivery-related complications and newborn and maternal health problems, in addition to LBW and preterm birth that have been previously identified as potential long-term determinants of sleep patterns and quality (Paavonen et al., 2008; Strang-Karlsson et al., 2008). However, contrary to hypotheses, we did not find evidence that obstetric risks predicted adolescent sleep quality. One reason might be that different obstetric risks can have highly specific roles in shaping sleep development, whereas our study included them all as a single summary variable of various risks. For example, the higher risk of sleeping problems among premature children can reflect an evolutionary mechanism where prematurity provokes calibration in the sleep regulatory system to save more energy (Saper, Scammell, & Lu, 2005). Accordingly, future studies should also consider multiple obstetric factors in predicting sleep, but examine both their combined and interactive impact, as well as their potential unique biological and social mechanisms to explain sleep.

### *Emotion regulation and sleep quality*

Our results failed to confirm the hypothesized role of middle childhood emotion regulation in mediating between early of family environment and adolescent sleep. Adolescence is a period with fundamental

changes in emotional domain (Crone & Dahl, 2012), which may explain why emotion regulation in middle childhood did not predict sleep in late adolescence. Intensive negative emotions and strong fluctuations characterize adolescents' daily life as responses to upheavals in peer, romantic, and parental relationships (Morris et al., 2007). In middle adolescence repertoire of emotion regulation strategies diminishes and dysfunctional regulation of emotions, especially of anger and sadness, increases (Zimmermann & Iwanski, 2014). Yet, with time adolescents also show increasing capacity for long term and purposeful emotion regulation serving their salient goals (Moilanen et al., 2018) and use more mature and sophisticated cognitive regulation strategies, such as reappraisal and non-suppression (McRae et al., 2012). It is plausible that these intensive and contrasting changes in adolescents' emotion regulation are decisive for their sleep, and therefore earlier studies focusing only on adolescence period found dysfunctional emotion regulation associating with sleep problems (Palmer et al., 2018; Tavernier & Willoughby, 2015).

Methodological explanation for the null-finding of the mediating role of emotion regulation may be related to the fact that parents evaluated their children's emotion regulation in middle childhood, whereas adolescents themselves reported their sleep features in late adolescence. It is possible that adolescent self-reported emotion regulation could more highly concur with their sleep quality than parental reports. Yet, there is evidence that children's self-reports tend to concur with the level and structure of parents' corresponding evaluation (Duckworth & Kern, 2011; Goulter et al., 2022).

It is important to consider that negative early parenting experiences did predict children's emotion regulation problems in middle childhood. This finding concurs with previous research indicating infancy to be an important period for emotion regulation development (Feldman et al., 2009; Tronick, 2005) and emphasizing the optimal co-regulating role of the parent–infant relationships to allow infants to develop effective ways of emotion regulation (Philbrook & Teti, 2016; Sadeh et al., 2010). As the age-salient importance of emotion regulation development was demonstrated, it could be fruitful for future studies to investigate more thoroughly the mechanisms of early emotion regulation in predicting long-term development. For instance, children with regulatory problems may evoke more strict and negative parenting practices (Hayden et al., 2013), and thus shaping their own early environment. Thus, future research could also analyze the moderating mechanims of children's emotion regulation in the early family environment predicting adolescent sleep.

### *Strenghts and limitations of the study*

There are three main limitations regarding our study. First, our prospective design had long time intervals between the assessments. It would have been ideal to study the quality of family relationships, emotion regulation, and sleep quality over comparatively shorter durations, including in toddlerhood and middle childhood, in order to chart continuous and ongoing child development. Assessments of sleep determinants would also have been important in early and middle adolescence, where critical changes happen in social, hormonal, and neurocognitive development (Crone & Dahl, 2012; Steinberg, 2014). Second, our analyses were based on adolescent self-reports of sleep and parent-reports about the early family environment and child emotion regulation, which increases risks of subjective biases and social desirability. In particular, objective polysomnographic and actigraphy methods are golden standards for sleep assessment (Buysse et al., 2010). Nevertheless, it is worth noticing that our multi-reported design and statistical modeling of common method factors minimize the potential common method bias due to the same assessor. Third, our psychometric assessment showed some weaker factor loadings, especially in the case of some sleep indicators (e.g., eveningness), suggesting that they captured the overall adolescent sleep quality relatively weakly. Yet, it should be noted that, in several studies, the PSQI has demonstrated a one-dimensional structure (Manzar et al., 2016, 2018), and our additional analyses (i.e., modification indices) revealed little evidence about any effects between the determinants and specific indicators of adolescent sleep. Accordingly, more sophisticated research about the early determinants of adolescent sleep would require a prospective study assessing development in infancy, toddlerhood, middle childhood as well as in early, middle and later adolescence. This knowledge would reveal changes and interactive dynamics in significant age-salient determinants of sleep. Also, future studies should combine both self-reported sleep diaries and objective sleep-graphic methods, as both can provide insightful understanding.

### *Conclusion and implications*

In the current study, we showed that maternal mental health problems in infancy represent an early risk that can have a long-term negative impact on children's sleep quality. Future studies should examine the more complex interaction of early family environment and children's neurobiological susceptibility, indicated, for example, by children's emotion regulation abilities and emotional reactivity. We tested a comprehensive general model of early risks on later sleep, and the next step could be analyzing the specificity of social and biological determinants of adolescent sleep. For instance, genetic factors, fetal programming, parental bedtime behaviors, and specific aspects of parent–infant co-regulatory interactions such as touching may have unique roles in sleep development. It would similarly be intriguing to analyze the timing of problematic family relationships to better understand the roles of different developmental periods on adolescent sleep.

The results have implications for fostering optimal adolescent sleep, understanding the role of early family environment in later development, and enhancing maternal mental health as a core determinant of children's sleep. Clinically, our study provides legitimation for both early psychosocial interventions for mothers with mental health problems and screening of adolescent poor sleep quality as a potential risk for problems in adolescent functioning. Supporting mothers in their transition to parenthood can have positive implications on maternal perinatal health, stress regulation, and mother–infant interaction (Cluxton-Keller & Bruce, 2018; Dol et al., 2020). This may, in turn, enhance fetal and child developmental aspects of self-regulation, with potential long-term significance on the child and adolescent sleep. Importantly, early interventions targeted to mothers with mental health problems are cost-effective (Bauer et al., 2014). Adolescents' sleep development involves drastic changes, increasing the risk of persistent sleeping difficulties and mental health problems (Poitras et al., 2016; Orchard et al., 2020). Yet, there are evidence- and theory-based interventions available to improve adolescent sleep, with potential long-term mental health benefits (Blake Snoep et al., 2017; Hendricks, & C. M., Grodin, L. K., & Slifer, K. J., 2014). General guidelines indicate cognitive–behavioral therapy for insomnia as the first-line treatment (Mitchell, Gehrman, Perlis, & Umscheid, 2012), but interventions for adolescents might also benefit from identifying the manifold developmental and cascading mechanisms that relay the effects of early maternal psychopathology on children's sleep.

## Author note

This study is a part of the Miracles of Development research project (https://projects.tuni.fi/kehi), granted by the Academy of Finland #326641 and #323845 and the Juho Vainio Foundation.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appdev.2022.101420.

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