Depressive symptoms in the transition to parenthood: Patterns, processes and child outcomes

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Summary

Postpartum depression has been recognized as a serious condition by clinicians and researchers for decades, yet our understanding of the complexities of depressive symptomatology during the perinatal period is still in its infancy. It is unlikely that any one factor or perspective can explain the multifaceted presentation and course of depressive symptoms in this period, and a more comprehensive understanding of meaningful distinctions within etiologies, symptom developments and prognosis is needed. Studies have traditionally focused on postpartum women. More recently, the pregnancy phase and fathers' mental health has been included in research efforts, adding new perspectives. Furthermore, we know that depressive states in the transition to parenthood is not only detrimental to the mental health of parents, it can also have profound effects on the caretaking environment for the newborn child. Early parental depression adds to the child's risk of developing a wide range of social, emotional and cognitive problems, however, there is a need for elucidating mechanisms for the transmission of risk, as well as factors that adds or buffers vulnerability for an adverse development. On this backdrop, the main goal of this thesis was to provide a more detailed understanding of how depressive symptoms unfold during the entire perinatal period for mothers and fathers, and how this relates to child outcomes. This included investigating the heterogeneity of perinatal depressive symptom courses among women; the dynamic transmission of depressive symptoms within parental couples throughout the perinatal events of pregnancy, childbirth and early parenthood; and how child outcomes are related to parental depressive symptoms while in the womb and the first 18 months of their lives.

The data for this thesis are from the prospective, multisite study *Little in Norway* (LiN) and comprise nine data collection waves; four prenatal, birth records, and at six weeks, six months, 12 months and 18 months after the children were born. The 1,036 participating families were recruited at nine different well-baby clinics located at geographically diverse sites across Norway. Collected data includes surveys from both parents, birth records and psychological tests of the children.

In paper I, we explored whether depressive symptoms in the perinatal period could be categorized into distinct trajectories of symptom development among subgroups of women, and further investigated predictors to these trajectory groups. By means of growth mixture modeling, four classes of depressive symptom trajectories were identified, including (a) *pregnancy only* (4.4%), (b) *postpartum only* (2.2%), (c) *moderate-persistent* (10.5%), and (d) *minimum symptoms* (82.9%) classes. Membership in the pregnancy only and postpartum only

classes was primarily associated with pregnancy-related anxiety and previous psychopathology, respectively, whereas the moderate-persistent class was associated with diverse psychosocial adversity factors. These findings suggest heterogeneity in temporal patterns of elevated depressive mood, and relate specific trajectories of time courses with distinct adversity factors.

In paper II, we also included the fathers' depressive symptoms throughout the perinatal period, and investigated reciprocal relations in depressive symptomology between partners, expecting that maternal symptoms had a stronger association with subsequent paternal symptoms than the opposite case. We further hypothesized that couples with insecure adult attachment styles would be more prone to transmission of negative mood states. By utilizing an autoregressive latent trajectory modeling approach, we found that women's depressive symptoms late in pregnancy predicted elevated depressive symptom levels in fathers 6 weeks after birth. Moderation analyses showed that regardless of attachment status, fathers in early parenthood are vulnerable for the development of depressive symptoms in cases of elevated maternal depressed mood. However, for couples characterized by an insecure attachment style, transmissions of depressive symptoms were evident at additional time points during pregnancy and throughout the first year of parenthood, suggesting a heightened susceptibility of prolonged disruptive depressive symptom contagion processes for this subgroup.

Paper III investigated child social, emotional, cognitive and language outcomes of parental depressive symptoms during the perinatal period. The paper explored pre- and postnatal depressive factors, differential effects of mothers' and fathers' symptoms, as well as parenting stress when the children are 12 months old as a mediator of adverse child outcomes at 18 months of age. Results indicated that parental perinatal depressive symptoms predicted child social-emotional functioning, specifically externalizing, internalizing, and dysregulation problems, as well as language developmental delay at 18 months. A differential effect was evident, linking maternal symptoms to social-emotional outcomes, and paternal symptoms to language outcomes. Parenting stress mediated most relations between parental depressive symptoms and child outcomes.

In summary, the papers presented in this thesis takes a detailed approach to depressive symptom development and expression in the transition to parenthood. By investigating heterogeneity of symptom courses, transmission of negative mood states within parental couples, and differential and mediating effects on child outcomes, findings provide important distinctions in our understanding of how depressive symptoms unfold during the specific

events of pregnancy, childbirth and early parenthood. Findings have several clinical implications; the importance of including prevention, assessment and treatment efforts of depressive symptoms as early as the pregnancy phase; bringing the mental health of fathers into perinatal care; and direct resources to identify and accommodate the needs of those with a heightened risk for depressive symptomatology.

List of Papers

Paper I:

Fredriksen, E., von Soest, T., Smith, L., & Moe, V. (2017). Patterns of pregnancy and postpartum depressive symptoms: Latent class trajectories and predictors. *Journal of Abnormal Psychology, 126*, 173-183. doi: 10.1037/abn0000246

Paper II:

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Paper III:

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1 Introduction

To experience depressive symptoms when becoming a parent hits hard. Symptoms of sadness, lack of purpose and meaning, and the loss of interest in everyday activities stands in stark contrast to the joy, meaningfulness and vitality we tend to associate with childbirth. Not only can perinatal depressive problems have a detrimental effect on parental mental health, it can also have a profound impact on the caretaking environment for newborns (Field, 2010), posing a significant threat to their development across a wide range of developmental domains (Stein et al., 2014). On the societal level we see rising numbers of perinatal depression (Pearson et al., 2018), and we are still in need for more efficient and targeted preventive and treatment interventions (Howard et al., 2014). Taken together, this gives a sense of urgency to the work of advancing knowledge in this field.

Numerous studies have been conducted on postpartum depression in women, advancing knowledge about issues such as prevalence, risk factors, prognosis, symptom presentation, potential etiological factors and child outcomes (O'Hara & McCabe, 2013), as well as providing clinicians with useful knowledge for building mental health competence into perinatal care services. However, there are many unresolved issues in the field, including understanding the heterogeneity of perinatal depressive states and delineating meaningful distinctions within risk factors, symptom presentations, and prognosis (PACT Consortium, 2015). Relative to the postpartum depression literature, there have been less concern for depressive symptoms during pregnancy. This is now changing, and issues such as how depressive symptoms may directly affect the fetus through the intrauterine environment (Waters, Hay, Simmonds, & van Goozen, 2014), how depressive symptoms may disrupt the psychological preparation of motherhood (Pearson, Cooper, Penton-Voak, Lightman, & Evans, 2010), and how it might interfere with the partner relationship in this transitional period (Rholes et al., 2011) attracts attention. There are, however, few studies that track the symptom development closely during pregnancy, in order to better understand symptom development and course in this period in particular, as well as associations to the symptom development throughout the entire perinatal period and later child outcomes in general.

Depressive symptoms among expectant and new fathers have traditionally been a relatively neglected field. In recent years, however, more studies have been published on fathers' symptoms in the perinatal period (Cameron, Sedov, & Tomfohr-Madsen, 2016; Massoudi, Hwang, & Wickberg, 2016; Ramchandani, O'Connor, et al., 2008; Skjothaug, Smith, Wentzel-Larsen, & Moe, 2015). Obviously, it is necessary to obtain better knowledge on paternal depressive symptoms in the transition to parenthood in order to develop a better

understanding of paternal depressive states and ultimately to prevent and ameliorate fathers' suffering. Moreover, omitting fathers from studies on perinatal depressive symptoms also precludes a more comprehensive understanding of the remaining family members, including reciprocal relations within the family and child developmental outcomes following parental depressive problems.

A large body of evidence have shown that perinatal depression increases the risk of adverse child development across a broad range of domains and age ranges (Goodman et al., 2011; Stein et al., 2014). However, effect sizes have tended to be small, and more refined studies parsing severity and timing effects are needed, along with an investigation of mediating and moderating factors (Goodman et al., 2011), in order to better understand which children under which conditions are at risk. There are less evidence linking paternal depressive symptoms to child outcomes (Ramchandani, Stein, et al., 2008), not to mention studies that include both parental symptom levels in the perinatal period, investigating differential and moderating effects.

The overall goal of this thesis have been to provide a more detailed understanding of how depressive symptoms unfold during both pregnancy and early parenthood for mothers and fathers, and how parental depressive problems in this period relates to child outcomes. This included investigating the heterogeneity of perinatal depressive symptom courses among women; the dynamic transmission of depressive symptoms within parental couples throughout the perinatal events of pregnancy, childbirth and early parenthood; and how child outcomes are related to parental depressive symptoms while in the womb and the first 18 months of their lives.

2 Depressive Symptoms in the Perinatal Period: Background, Perspectives and Ongoing Debates

Perinatal depression is common, and there are indications of it becoming more common with new generations of today (Pearson et al., 2018). A review of prevalence rates for women indicate that about 13 % of women experience postnatal depression and 11 % during pregnancy (Gavin et al., 2005). For men, prevalence rates are a little lower (8.4 %) (Cameron et al., 2016), and associated with more uncertainty, both due to a relative shortage of studies and because there is a lack of well validated questionnaires developed with typically male symptoms in mind (Madsen & Juhl, 2007; Massoudi, Hwang, & Wickberg, 2013). There are considerable cross-cultural and social variation in prevalence rates, with different national prevalence rates ranging from 0 % to 60 % (Halbreich & Karkun, 2006), and with depression rates typically being significantly higher in low- to middle-income

countries compared with high-income countries (Parsons, Young, Rochat, Kringelbach, & Stein, 2012). The widely diverging prevalence rates might reflect differences in cross-cultural interpretation of mental health and childbirth, socio-economic conditions and reporting style (Halbreich & Karkun, 2006). Regarding symptom onset in cases of postpartum depression in women, a large American study show that only about 40 % of depressive episodes begin postpartum, while 33 % have a pregnancy onset and about 27 % a pre-pregnancy onset (Wisner et al., 2013). For some women, depression develops into a persistent condition, leading to an increased risk of aberrant development for both the women and their children (Vliegen, Casalin, & Luyten, 2014). Reviews of the literature indicate that risk factors for perinatal depression include previous psychopathology, domestic violence, history of abuse, life stress, lack of social or partner support, migration status, and anxiety during pregnancy, while the following risk factors have received slightly less systematic evidence: Pregnancy complications, neuroticism, family history of psychiatric illness, low socioeconomic status, substance misuse, and chronic illness (Biaggi, Conroy, Pawlby, & Pariante, 2016; Howard et al., 2014; O'Hara & McCabe, 2013).

2.1 Perinatal depression: Distinct from depression and distinctions within?

The question of whether perinatal depression in women is a distinct entity from depression in general have been contested for a long time (i.e. Bell, Land, Milne, and Hassanyeh (1995); Whiffen and Gotlib (1993); Pitt (1968)). Certainly, the circumstances of becoming a mother when experiencing depressive symptoms shape the experience of the symptoms; with negative thoughts and feelings perhaps revolving around being a bad mother, the loss of self-worth and vitality related to changing roles and identities, and the struggles with daily functioning being extended to not being able to cope with caregiving tasks. In and of itself, however, that does not imply a differential etiology, presentation of symptoms or prognosis from a depressive condition at other time periods. Bernstein et al. (2008) found differences in the symptom presentation among groups of women with postpartum and nonperinatal depression. However, findings were inconclusive in relating the differential symptom features to either distinct phenotypes of depression or merely a consequence of the external circumstances of childbirth and caring for a newborn. Studies have shown a similarity of risk factors for both perinatal and nonperinatal depression (O'Hara & McCabe, 2013). In fact, one of the most robust risk factors of perinatal depression is having a prior history of depression (Biaggi et al., 2016; Howard et al., 2014), providing at a minimum a strong link between these conditions. Further, longitudinal studies have prospectively shown that struggling with mood and anxiety problems in one’s teens adds to the risk of developing

postpartum depression, and findings have been interpreted as support for the notion of postpartum depression as a continuation of mental health problem beginning pre-pregnancy (Patton et al., 2015).

In contrast, other lines of evidence points to the distinctiveness of perinatal depression. In a study on the specificity of postpartum depression, Cooper and Murray (1995) found that first onset depression in the postpartum period predicted subsequent postpartum depressions, but not depression in other life periods. Further, the relative risk of depression in the postpartum period has been found to be higher compared with non-perinatal periods (Munk-Olsen, Laursen, Pedersen, Mors, & Mortensen, 2006; Vesga-Lopez et al., 2008).

Parallel with these developments there have been investigations into the possible heterogeneity of perinatal depression, in terms of etiologies, symptom presentation and prognosis. Investigating differences among pregnancy and postpartum onset of depression, Altemus et al. (2012) found that women with a pregnancy-onset depression were more likely to have a history of depressive episodes (both postpartum and non-perinatal) and were more commonly associated with psychosocial stressors compared to postpartum-onset depression. The PACT Consortium investigated the heterogeneity of symptoms in postpartum depression by modeling latent classes in a large, combined dataset, and found that subtypes of postpartum depression could be distinguished based on severity, timing of onset, comorbid anxiety and suicidal ideation (PACT Consortium, 2015). Relatedly, Forty et al. (2006) employed a detailed examination of how the timing of postpartum depression onset was associated with the familial aggregation (among pairs of sisters) of postpartum depression. Clustering within families were evident with a narrow time definition of the postpartum period (within 4 weeks of delivery), however, with a temporal definition of 6 month postpartum, this clustering were no longer evident. This suggests that familial factors add to the vulnerability to early onset postpartum depression only, suggesting heterogeneity in etiologies of subtypes of postpartum depression. Findings in this field thus seem to be related to the temporal definition of the postpartum period. With no consensus of a defined time frame for postpartum depression, a pragmatic approach has been advised, with a tendency in the field to deploy narrow time frames when investigating biological determinants, whereas wider time frames have been considered more useful when including social determinants and treatment efforts (O'Hara & McCabe, 2013). A prime hypothesis in explaining childbirth as a specific trigger of depression onset with a narrowly defined postpartum depression for a subgroup of women, has been to consider a heightened vulnerability to the hormonal

fluctuations, specifically an abrupt and steep drop of gonadal steroids following childbirth (Bloch et al., 2011; Bloch, Daly, & Rubinow, 2003).

The emergent evidence of the heterogeneity of perinatal depression have important implications for whether to perceive perinatal depression as a distinct phenomenon from depression at other time periods. A tenable interpretation of findings indicates that some mechanisms could be common for perinatal and non-perinatal depression, thus essentially reflecting similar conditions, and in this perspective pregnancy, childbirth and infant care considered as generalized stressors. Whereas in other cases of postpartum depression, the depressive condition seem to be uniquely connected with pregnancy, childbirth and the immediate postpartum period, thus pointing at the specificity of the condition.

2.2 Depressive symptoms when men become fathers

There have been two meta-analyzes of prevalence rates concerning men's pre- and postnatal depression (Cameron et al., 2016; Paulson & Bazemore, 2010), the first reported a 10% prevalence rates, while the more recent study reported 8.4% accounting for about twice as many studies (n = 74) as the preceding meta-analyses. Both reported considerable positive association with maternal depression. Cameron et al. (2016) found that there were no differences in prevalence rates during the pre- and postnatal periods, suggesting relatively stable rates of depression during the transition to parenthood. There were some regional differences, with rates being somewhat higher in North America, and somewhat lower in Europe and Australia. There have been concerns about the measurements used to estimate paternal depressive symptoms, with checklists such as the EPDS receiving criticism for not taking into account the typical male symptoms of depression, thus providing an underestimation of paternal depressive problems (Madsen & Juhl, 2007; Massoudi et al., 2013), which should be noted when considering the studies in which this applies.

Predictors of paternal postnatal depressive symptoms do to some extent mirror that of maternal postnatal symptoms as previous psychopathology, prenatal depression and / or anxiety, partner relationship problems, low social support, having a partner with depressive symptoms, young age, and low socioeconomic status have been found to be predictors of increased risk of depressive symptoms for men (Bergström, 2013; Deater-Deckard et al., 1998; Massoudi et al., 2016; Ramchandani, Stein, et al., 2008; Wee, Skouteris, Pier, Richardson, & Milgrom, 2011). A study investigating prenatal depressive symptoms also found that having experienced adverse childhood experiences increased the risk of depressive and anxious feelings during the pregnancy phase (Skjothaug et al., 2015).

Studies have found that depression in new fathers is negatively associated with father-child engagement, quality of the relationship with the mother and co-parental relationship supportiveness, and positively associated with parenting stress (Bronte-Tinkew, Moore, Matthews, & Carrano, 2007). In a large prospective study Ramchandani, Stein, et al. (2008) found that paternal depression predicted child psychopathology at 7 years of age, relatedly it was also found that a combination of pre- and postnatal depressive symptoms had a greater impact on adverse child outcomes at 3.5 and 7 years compared with either period or no symptoms (Ramchandani, O'Connor, et al., 2008). In a more recent study of mediating factors, it was found that the association between paternal depression and child outcomes was mediated through family environmental factors such as interparental conflict and maternal depression. However, the reverse was not the case, maternal depression evinced a direct association with child behavioral and emotional problems, without mediating factors being identified (Gutierrez-Galve, Stein, Hanington, Heron, & Ramchandani, 2015).

2.3 Transmission of depressive symptoms in a couples context

To experience depressive symptoms in the perinatal period is moderately associated between partners in the parental couple (Cameron et al., 2016; Paulson & Bazemore, 2010), mirroring the positive association of depressive symptoms between spouses in general (Meyler, Stimpson, & Peek, 2007). Several mechanisms have been suggested to play a part in explaining this link, including assortative mating, common contextual stressors / factors, shared health beliefs and habits, and the transmission of depressive symptoms between partners (Meyler et al., 2007). Whereas the former are largely time invariant factors, the transmission of depressive symptoms between partners describe a process of symptom development unfolding over time. Highlighting the interpersonal determinants of symptom development, Joiner and Katz (1999) has termed the transmission of negative affect or depressive symptoms between partners over time for depression contagion, and found support for contagion processes in various types of close relationships in a meta-analysis. The effects were stronger for depressive symptoms than negative affect. Conceptually, they build on the interactional theory of depression (Coyne, 1976) that posits that when depressed we tend to seek excessive reassurance, but also negative feedback from those around us, resulting in a communication pattern of asking for support yet doubting the sincerity of the support and rejecting it when it is offered (Coyne, 1976; Kouros & Cummings, 2010). This interactional style may be burdensome to close partners, to the point that, over time, it may lead to an elevation of depressive symptoms in the partner, constituting a depression contagion process.

Studies have linked marital satisfaction with depressive symptom concordance (Tower & Kasl, 1996; Yorgason, Almeida, Neupert, Spiro III, & Hoffman, 2006), yet very few have investigated transmission of symptoms with an appropriate longitudinal design allowing for reciprocal relations unfolding over time. However, Kouros and Cummings (2010) found that depressive symptoms in husbands' predicted a subsequent rise in depressive symptom levels for the wives over time, by examining dynamic longitudinal pathways. Studies investigating this with respect to the specific events of pregnancy, childbirth and early parenthood are scarce. Further, there is a need for studies identifying which couples that are at risk for prolonged depressive symptom contagion processes.

The perinatal period brings about considerable changes in personal identity, partner relationship and family dynamics, as new identities as mothers and fathers are being shaped. These constitute personal changes and new experiences relevant for the attachment system. Attachment theory have been closely linked with psychopathology in general, and depression in particular since its origin in Bowlby's writings (Bowlby, 1969, 1973, 1980). There is also ample empirical evidence linking insecurities in attachment orientations to depressive problems, including perinatal depression (McMahon, Barnett, Kowalenko, & Tennant, 2005; Mickelson, Kessler, & Shaver, 1997; Rholes et al., 2011; Stern et al., 2018). Attachment theory posits that all humans are born with an inclination to create affectionate bonds with selected others (usually parents or other care givers), and to stay close to and approach attachment figures when distressed. These bonds or relationships become internalized as generalized models self and others over time, termed internal working models (Collins, Guichard, Ford, & Feeney, 2004; Rholes et al., 2011). Adult attachment styles are often categorized along two dimensions, anxious and avoidant attachment. It is typically assessed relating to romantic partners in general, rather than being a measure of one particular relationship (Brennan, Clark, & Shaver, 1998; Fraley, Waller, & Brennan, 2000). Anxious attachment style is characterized by fear of abandonment, difficulty in trusting one's partner and a hypervigilance to signs of their partner leaving them. Individuals scoring high on avoidant attachment orientations, on the other hand, find it difficult to be psychologically close to their partner, are afraid of depending on their partners, and try to maintain psychological independence. Although different in interpersonal strategies, both anxious and avoidant attachment styles are characterized by a distrust in their partners, compared to secure attachment orientations.

There are several potential processes that might link adult attachment styles with depressive symptomatology, such as less efficient coping strategies with anxious attachment

styles relying on emotion-focused coping, and highly avoidant individuals rely on avoidance and distraction. Individuals with more avoidant styles are also less likely to seek out social support. (Rholes et al., 2011). Although there are evidence linking depression and perinatal depression to insecure attachment orientations (McMahon et al., 2005; Mickelson et al., 1997; Rholes et al., 2011), there is a lack of studies with a rigorous design suited to test reciprocal relations within the couple over time, and specifically testing whether depressive symptom contagion processes are moderated by insecure attachment orientations.

2.4 Child outcomes and the transmission of risk

As depressive symptoms are common in the population, a substantial number of children are reared by depressed parents. Studies investigating child outcomes of perinatal depression have found that it predicts a higher risk of mental health problems, adverse social-emotional, language and cognitive functioning in children across various age ranges (Stein et al., 2014). This have been shown in the prenatal period (Koutra et al., 2013; Madigan et al., 2018; Pearson et al., 2013), the postnatal period (Koutra et al., 2013; Liu et al., 2017; Stein et al., 2014), and among fathers (Ramchandani & Psychogiou, 2009). Effect sizes tend to be small, however, stronger effects have been shown in cases of persistent depression (Goodman et al., 2011; Vliegen et al., 2014).

There are a number of ways the transmission of risk might operate to explain such outcomes. In this context it is useful to separate between prenatal and postnatal periods as the fetus and child inevitably experience maternal depression differently in utero and as a newborn, consequently some of the potential mechanisms only pertains to either period. Few studies have disentangled pre- and postnatal symptoms, however, it has been found that prenatal depression uniquely accounts for variation in mental health when the children are 18 years old (Pearson et al., 2013), and cognitive functioning as toddlers (Koutra et al., 2013). Prenatal mechanisms have been termed fetal programming effects, and encompass several hypothesized mechanisms in which the maternal depressive states affects the intrauterine environment to the extent that this impacts the growing fetus with lasting effects (Talge, Neal, Glover, & ESTRPSN, 2007). Broadly speaking this includes fetal malnutrition and fetal overexposure to glucocorticoids (Harris & Seckl, 2011). There have been some support for fetal programming effects by studies using animal models (Golub et al., 2016).

In the postnatal period the child is directly exposed to the depressed parent's affect, cognitions and behaviors. Parenting is thus a potential mechanism through which parental depression is associated with adverse child outcomes, as depression may interfere with the quality of the parent's interaction with children (Dix & Meunier, 2009; Lovejoy, Graczyk,

O'Hare, & Neuman, 2000; Stein et al., 2014). Several processes may link depression with deficiencies in parenting. The emotional availability of the parent may be compromised by symptoms such as a preoccupation with negative thoughts, fatigue, and parental withdrawal, resulting in reduced sensitivity and responsivity to infant cues, which may have an adverse effect on the development of infant regulatory capacities (Field, 2010; Tronick & Reck, 2009). Further, depressive symptoms predict negative or flat affect, as well as reduced positive affect in interaction with their children (Feng, Shaw, Skuban, & Lane, 2007). Depressed parents have also been found to be more intrusive, more hostile and less patient in interactions with their children (Lovejoy et al., 2000), further to show higher rates of conflict and are more likely to respond destructively to oppositional behavior (Caughy, Huang, & Lima, 2009). Social withdrawal might also interfere with the parent's effort to seek out and provide stimulating learning environments, and as the social network and support of the depressed parent may have decreased (Surkan, Peterson, Hughes, & Gottlieb, 2006), the infant is provided with fewer opportunities of social encounters with other adults.

Parental depression is often associated with other contextual factors that might have a negative impact on child development, such as relationship dissatisfaction or conflict among parents, low socioeconomic status, stressful life events that may confound the association between parental depression and child development (Stein et al., 2014). Finally, common genetic risk is an important mechanism that has been shown to account for some of the shared risk among perinatal depression and their children (Hannigan et al., 2018).

3 Aims of the Thesis

The overall aim of this thesis is to investigate the nature of depressive symptoms in the transition to parenthood for women and men, focusing on the heterogeneity of depressive symptom time courses of women, the reciprocal relationship of depressive symptoms between parents, and child outcomes of pre-and postnatal depressive symptoms in mothers and fathers.

3.1 Paper I

In this study, we wanted to explore whether maternal depressive symptoms during pregnancy and the first postpartum year could be categorized into several distinct, empirically defined trajectories of time courses. We expected to find (a) one trajectory characterizing women with elevated symptoms limited to the pregnancy period; (b) one trajectory of early postpartum onset and a gradual recovery; (c) a stable trajectory at a moderate level with pregnancy onset in which symptoms continue into the postpartum period; (d) a small group of women with a very high symptom level throughout the period of study; and (e) a majority of

women presenting minimum symptoms. Second, we aimed to investigate whether potential psychosocial adversity factors, such as sociodemographic factors, previous psychopathology, stress, partner-related attachment patterns, pregnancy-related anxiety, and childhood trauma were differentially associated with the hypothesized trajectories. More specifically, we expected that higher levels of adversity predicted membership in trajectory classes with elevated symptom burden, relative to trajectories with low symptoms. Further, we expected stable courses with elevated symptoms to be predicted by more adversity factors than transient courses.

3.2 Paper II

The main aim of this paper was to investigate how depressive symptoms unfold within the parental couple during pregnancy and early parenthood. In our theoretical model of perinatal depressive symptoms, we propose that symptom development is governed by both a stable tendency of affective mood over time and time-specific changes unfolding in the perinatal period. Our primary goal was to investigate whether either parent's depressive symptoms at one time point predicted the depressive symptom level of the other parent at the subsequent time point, thus delineating time-specific changes in symptom development. We expected stronger effects around and after birth, as the emotional interdependence and reciprocity in the relationship are likely to increase in this period. Further, we expected mothers' symptom levels to have a greater effect on fathers than the opposite case, as maternal and fetal well-being are of central concern in this specific period. Moreover, we aimed to investigate whether adult attachment styles moderated these relations, and expected stronger effects with insecure partner-related attachment styles. Disentangling within-person and between-person effects by statistical design, we also aim to provide a basis for evaluating alternative interpretations of depressive symptom associations within the parental couple

3.3 Paper III

The purpose of this paper was to examine the associations between parental depressive symptoms in the perinatal period (from pregnancy throughout the first postpartum year) and child social-emotional, cognitive, and language skills at 18 months of age. Specifically, we examined (a) whether exposure to parental pre- and postnatal depressive symptoms is associated with children's developmental outcomes, and if there is an independent effect of prenatal symptoms after postnatal symptoms are accounted for; (b) whether any prospective associations between parental depressive symptoms and children's developmental outcomes are different for mothers and fathers; (c) whether associations between parental depressive symptoms and child outcomes are particularly strong when both

parents suffered from high symptom loads; and (d) whether parenting stress measured when children are 12 months old mediates the relations between parental depressive symptoms and the children's social-emotional, cognitive and language functioning at 18 months of age.

4 Method

4.1 The Little in Norway study

The papers in this thesis all used data from the Little in Norway-study (LiN-study), a longitudinal population study of infant vulnerability and plasticity from pregnancy to age 18 months. From September 2011 until October 2012 pregnant women receiving routine prenatal care at nine public well-baby clinics in Norway were invited to participate in the LiN study. The data collection sites were located at geographically diverse sites across Norway; Tromsø in the north, Namsos, Strindheim (Trondheim) and Høylandet in the middle of Norway, Ulset (Bergen) and Straume on the west coast and Lørenskog, Frogner (Oslo) and Østensjø (Oslo) in the east, to ensure a wide variety of demographic conditions. In this thesis we have used data from nine data collection waves: At average gestational week 21 (range: weeks 8-34) (T1), week 28 (T2), week 32 (T3) and week 36 (T4), at birth / birth records (T5), 6 weeks postpartum (T6), 6 months postpartum (T7), 12 months postpartum (T8), and 18 months postpartum (T9). All questionnaire data were collected digitally, primarily by designated computers / tablets at the well-baby clinics, however, T3 and T4 were completed by participants from their home using private computers / tablets. Birth records (T5) were brought in by the participants themselves (birth records are routinely given to women when they are discharged from hospital after delivery), and test data (T9) were collected at the well-baby clinics.

4.2 Ethical considerations

All procedures performed in the study were in accordance with the 1964 Helsinki declaration and its later amendments, and the study was approved by the Regional Committees for Medical and Health Research Ethics in Norway. The expectant parents were given written and oral information before giving their consent to their own and their children's participation. Further, they have been informed about their right not to respond to any parts of the study, and the right to withdraw from the study without giving an explicit reason for doing so. Information about study findings are being communicated to participants in regular newsletters. Established routines in handling potential health and social problems discovered during the course of participation were followed, ensuring that participants were referred to the relevant health or child care services when needed.

Special consideration have to be taken when including infants and toddlers as research participants, as they are too young to give an informed consent or to even be asked for their acceptance of participation as is a recommended supplement to parental consent with older children (Backe-Hansen, 2002). There seem to be a tension between two ethical imperatives in research with infants, namely non-exclusion and informed consent. Infants and toddlers should not be excluded from research and its' benefits, as research is essential for their health and wellbeing, at the same time, all research participation should be free and participants should be able to give an informed consent. This dilemma is commonly solved by providing parental informed consent, but as the parents are not the ones facing the potentially damaging consequences themselves, and further, as there could be conflicting interests between parents and children, infants and toddlers should be protected from any risk that research participation might entail (Diekema, 2009). Guidelines drawn up by The Norwegian National Research Ethics in the Social Sciences and the Humanities (2016) states that "*A prerequisite for including individuals who cannot give a free and informed consent is that any risk and strain associated with the study are negligible for the individuals included.*"

In this study we tried to keep study procedures close to everyday experiences for the infants in order to avoid risk and strain to the infants; the data collection has taken place at local well-baby clinics that the family visits for child care check-up regardless of study participation. Further, the same health nurse met the family during the various data collection waves providing familiarity for the participants. Most importantly, the procedures and questions regarding the infants carries very low risk of pain and strain. However, it has been argued that extensive questioning of parents may, for vulnerable individuals, carry the risk of altering their moods, behaviors or parenting, i.e. by inducing guilt or confusion, that ultimately may affect the infants in a negative manner (Diekema, 2009). In this study, the low likelihood and low risk severity of that scenario combined with the importance of advancing knowledge about infant health and development, have led to the conclusion that the study protocol is ethically justifiable, also when taking the perspective of the infant.

4.3 Recruitment, participation and attrition

Women were recruited by midwifes at their local well-baby clinic at their first prenatal appointment, there were no exclusion criteria. Initially 1,041 gave their informed consent, 5 later withdrew all contributed data leaving 1,036 women as participants in the study. The partners of the pregnant women were also invited, and 884 partners (878 men and 6 women) gave their informed consent. During the course of pregnancy 29 women withdrew from the study, and eventually there were 1,017 children born by participating women,

including 10 pairs of twins. The response rate is estimated to be 50.7%. At five of the clinics, the staff did not establish reliable routines to monitor rates of participation, thus the response rate is calculated based on records from the remaining four clinics. However, participation rates are likely similar at the other five sites as recruitment strategies and resources allocated to the data collection were similar at all well-baby clinics.

There are three sources of missingness in these data; late recruitment, intermittent missingness and study drop-out. There was a wide time frame for enrollment in the LiN-study (weeks 8-34; 50% enrolled in weeks 20-28) despite all women being invited at their first prenatal visit, causing late recruitment to be a considerable source of missing data. Although there are established nationwide schedules of prenatal care in Norway, women may receive these check-ups at either their general practitioner (GP) or by a midwife at their local well-baby clinic. There is considerable variation in local practices and how women choose; many receive prenatal care at their GP in the initial part of pregnancy, while switching to the well-baby clinics as the due date approaches. As the recruitment to the study happened at the well-baby clinics only and many women had their first appointment at their local well-baby clinic late in pregnancy, a significant proportion of participants were recruited too late to participate in the early data collection waves. Intermittent missingness comprise missing data by participants who missed one or more data collection points, but contributed with new data at later data collection waves. Finally, study drop-out comprise participants who either explicitly withdrew from the study, or were lost to follow-up.

Selective attrition has been investigated by means of logistic regression analyses in all three papers. Due to variations as to when the last data collection point was, and whether individual time points or averages across time points were used in the respective analytic design, numbers vary slightly in the papers. Overall, however, depressive symptoms were negatively associated with participation at the last data collection point for women and men. Further, lower maternal education and reporting adverse childhood experiences decreased the odds for participation at the last data collection point. Maternal parity was related to attrition at T8, but not at T9, while the number of fathers' previous children did not show any significant associations with missing status. Both parents' age and partner-related attachment were unrelated to attrition, along with maternal previous psychopathology, life stress, and pregnancy-related anxiety.

Three different sub-samples were included in the respective three papers in this thesis; all three papers used data from all pregnant / postpartum women at T1-T8 (excluding T5/ birth records). Papers II and III further included data from all participating fathers at the same

time points. Finally, in Paper III data from participating children collected at T5 and T9 were added.

4.4 Participant characteristics

At enrollment the mean age of the pregnant women was 30.3 years (range: 17-43, *SD* = 4.8) and 32.8 years (range: 16–57, *SD* = 5.9) for expectant fathers. Of the women, 54.9 % were nulliparous, while 56.2% of the male participants were first-time fathers. Most women were married (36.2 %) or cohabitating (59.7 %) with their partners, with only a small fraction being single/divorced/separated (2.7 %) or not specifying their marital status (1.4 %). A majority of participants was educated at university level (77.1 % and 67.1 % for women and men, respectively). At enrollment, 77.3 % of the women and 91.0 % of the men were full-time employed, 5.8 % of women and 2.5 % of men full-time students. Further, 13.6 % of women and 5.4 % of men reported being part-time students and/or part-time employed, while 3.0 % and 1.0 % of men reported being unemployed/on benefits/at home. Median annual personal income for women ranged from the equivalent of $37.000 to $55.000 (44.4 %) for women, while 31.1 % had lower and 24.3 % higher income. For men median personal income ranged from and $55.000 to $74.000 (27.5%), with 48.9 % earning less and 23.6 % having a larger income. The ethnic majority was Norwegian (93.9 % and 95.4% for women and men respectively), with a few reporting a diversity of other ethnic backgrounds on a measure of self-defined ethnic identity (6.1 % of women and 4.6 % of men).

More boys (52.4%) than girls were born in this sample. The mean gestational period was 39.5 weeks (*SD* = 1.9), and mean birth weight was 3,535 grams (*SD* = 542).

4.5 Measures

4.5.1 Depressive symptoms

Depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS) for men and women at T1 to T8 (except T5). The EPDS is a 10-item self-report questionnaire asking respondents to consider various depressive symptoms during the last seven days on a four-point scale (range: 0–30). Examples include “I have been so unhappy that I have had difficulty sleeping” and “I have felt sad or miserable”. The EPDS was originally developed to screen for depressive symptoms in women during the postpartum period (Cox, Holden, & Sagovsky, 1987). It was later validated for prenatal use by Murray and Cox (1990), and in a Norwegian community sample (Eberhard-Gran, Eskild, Tambs, Schel, & Opjordsmoen, 2001). It has also been validated on male populations in the postnatal period (Matthey, Barnett, Kavanagh, & Howie, 2001). Although developed with cut-off scores indicating probable depression, the EPDS composite score has also been used as a

continuous variable for research purposes (Matijasevich et al., 2015), with the benefit of yielding a more detailed range of depressive symptomatology at both clinical and subclinical levels. In this study, the EPDS composite score was used as a continuous variable. Cronbach's alphas were high at each assessment (ranging from .80 to .85 for women and from .76 to .84 for men), indicating good internal consistency.

4.5.2 Previous psychopathology

Participants were asked the following question: "Have you ever experienced mental health problems earlier in life?" (yes/no). Similar single question measures have been shown to serve as acceptable screeners for mental health problems (Veldhuizen, Rush, & Urbanoski, 2014), and have previously been used extensively in research (van der Waerden et al., 2015).

4.5.3 Adult attachment styles

Characteristics of adult attachment styles were assessed by the Experiences in Close Relationships Scale (ECR), which is a 36-item self-report measure of adult romantic attachment styles rated on a 7-point scale. The ECR yields two subscales of underlying attachment orientations: anxiety (fear of interpersonal rejection or abandonment, an excessive need for approval from others, and distress when one's partner is unavailable or unresponsive), and avoidance (fear of dependence and interpersonal intimacy, an excessive need for self-reliance, and reluctance to self-disclose) (Brennan et al., 1998). Higher scores reflect greater levels of insecure attachment within each attachment orientation (range 18-126 on each subscale). In this study Cronbach's alphas were .88 and .89 for anxiety and avoidance subscales, respectively, in accordance with the high level of internal consistency reported in other studies (Brennan et al., 1998; Fraley et al., 2000).

When creating grouping variables necessary for the moderator analyses in Paper II, we wanted to utilize a categorization that ensured that the secure/insecure attachment status was emphasized, rather than the types of insecure patterns. Thus, participants scoring in the lower range on both subscales (cut-off on the 75th percentile) were categorized as securely attached, while participants scoring above cut-off on either subscale were categorized as insecure. Couples in which both partners were categorized as insecurely attached comprise the insecure partner-related attachment group, while the secure partner-related attachment group includes couples with one or both partners being securely attached.

4.5.4 Stressful life events

Stress was measured by the life stress subscale, which is part of The Parenting Stress Index (PSI) (Abidin, 1995). The Norwegian version of the subscale lists 22 major life events (Kaaresen, Ronning, Ulvund, & Dahl, 2006), such as serious illness in the family, changing

school or work place (range 0-91). The respondents are asked to indicate whether the family had experienced each of the life events during the last 12 months. Items were weighted according to the Professional Manual of the Parenting Stress Index (Abidin, 1995), and the composite score was used in this study.

4.5.5 Anxiety during pregnancy

Anxiety related to pregnancy and birth was assessed by the 10-item Pregnancy Related Anxiety Questionnaire-Revised (PRAQ-R) (Huizink, Mulder, Robles de Medina, Visser, & Buitelaar, 2004). Each item is measured on a 5-point scale. PRAQ-R yields three subscales (fear of giving birth, fear of bearing a physically or mentally handicapped child and concerns about one's own appearance). In this study, mean scores across all 10 items were computed to obtain an indication of overall level of anxiety related to pregnancy and birth (Cronbach's alpha = .84, range: 10-50).

4.5.6 Childhood trauma

Childhood traumas were assessed retrospectively by the Adverse Childhood Experiences Scale (ACE), a self-report measure of childhood abuse, neglect, and household dysfunction (Dong et al., 2004). It lists ten types of adverse childhood experiences and asks whether they have been experienced during their childhood. Dong et al. (2004) showed that experiencing one type of adverse childhood event increased the odds of having additional adverse childhood experiences, and highlighted the importance of looking at the extent of such experiences rather than effects of a specific type. In this study we used the sum of reported types, ranging from 0 to 10.

4.5.7 Parenting stress

Parenting stress was measured with the Parenting Stress Index (PSI; Abidin, 1995) when children were 12 months old. The PSI examines the level of stress within the parent-child system, and consists of factors reflecting parental coping and their perceptions of the child. It is measured on a five-point scale, consists of 101 items, and has two domains: the parent domain assesses the personal characteristics and social support of the parent, whereas the child domain reflects the degree to which various child characteristics causes stress to the parents. Total scores are the sum of both domains, with a range from 101 to 505. In this study, we used total scores, as we were primarily interested in the level of parental stress, rather than the source of that stress. The PSI has shown good internal consistency, and has been validated in both normal and diverse clinical populations (Abidin, 1995). In this study, Cronbach's alphas for total scores were .94 for both mothers and fathers.

4.5.8 Cognitive and language functioning

Cognitive and language development was measured by the Bayley Scales of Infant and Toddler Development, Third Edition (Bayley-III; Bayley, 2006) when the children were 18 months. The Bayley-III is an individually administered instrument designed to measure the developmental functioning of infants and toddlers, especially suited to detect developmental delays. In this study, the cognitive and language scales were used (omitting the motor scales). The cognitive scale evaluates abilities, such as sensorimotor development, exploration and manipulation, object relatedness, concept formation, memory, and simple problem solving. There are two language scales: receptive communication, which measures verbal comprehension, and expressive communication such as babbling, utterances, and gestures. Health nurses, who were trained in the method, administered the test at the well-baby clinics under supervision of a clinical psychologist specialized in infant and child development. The administration and scoring rules were digitally implemented, so the sequencing and number of tasks were automatically computed based on the performance of the toddler. The parents were present in the room but instructed not to interfere with the testing.

4.5.9 Social and emotional functioning

The Infant-Toddler Social and Emotional Assessment (ITSEA; Carter and Briggs-Gowan, 2006) was used to assess social and emotional functioning when the children were 18 months old. The parent or parents accompanying the child to the well-baby clinic answered the questionnaire, resulting in 387 mothers, 32 fathers and 162 jointly answering the questionnaire. The ITSEA is a 168-item instrument measuring areas of social-emotional problems and competencies in 12-to 36-month-olds, with each item rated on a 3-point scale. It yields four domains: an internalizing domain, consisting of depression/withdrawal, general anxiety, separation distress, and inhibition to novelty subscales; an externalizing domain, consisting of activity/impulsivity, aggression/defiance, and peer aggression subscales; a dysregulation domain, consisting of negative emotionality, sleep, eating, and sensory sensitivity; and a competence domain, consisting of compliance, attention, mastery motivation, imitation/play, empathy, and prosocial peer relations. The ITSEA is widely used, and has shown acceptable reliability and validity (Carter, Briggs-Gowan, Jones, & Little, 2003). In this study, Cronbach's alphas were .65, .76, .78, and .82 for the internalizing, externalizing, dysregulation, and competence domains, respectively.

4.6 Statistical analyses: A longitudinal structural equation approach

The analyses in this thesis are conducted within a structural equation modelling (SEM) framework. SEM is a multivariate statistical approach that is used to analyze the

structural relationship between measured variables and latent constructs, combining features of factor analysis and multiple regression, and permits the use of various types of analyses combined in one model. Consequently, it has the advantage of estimating all elements in a model simultaneously, making it possible to evaluate the contribution of each parameter in the context of all parameters in the model.

All measurements are prone to error, and it is unlikely that our measurements in this longitudinal design was error-free. An important benefit of the SEM-approach has thus been the explicit modeling of measurement error, in order to reduce such bias in the estimates. Parameter estimation has been done by fitting means and covariances of the actual data with the best fitting model obtained through maximum likelihood estimation with robust standard errors to account for non-normality in the data. Model fit has been evaluated by inspecting χ^2-square statistics, Confirmatory Fit Index (CFI), Tucker–Lewis Index (TLI), and the root mean square error of approximation (RMSEA). Following the recommendations of Hu and Bentler (1999) CFI and TLI values of .95 or greater and RMSEA values of .06 or lower have been considered to indicate good fit. All data analyses were performed in Mplus 7.3 and 7.4 (Muthén & Muthén, 2015).

The longitudinal design in the LiN-study have yielded the opportunity of using 7- and 8-waves of depressive symptom data to address research questions regarding change and temporal associations in the perinatal period. Examining change necessitates (a) an explicit theory of how we expect change to unfold over the specified time period (i.e. rate and shape of change), and (b) a statistical approach that is suited to detect whether this articulated change hypothesis is supported or not supported in the data (Ram & Grimm, 2007).

4.6.1 Growth mixture Modeling (GMM)

Growth mixture modeling is an extension of latent growth curve (LGC) modeling in which subgroups with distinct trajectories can be identified in cases of unobserved heterogeneity in the population, and was used to examine study aims regarding the heterogeneity of perinatal depressive symptom courses, described in Paper I. When conducting GMM it is recommended to initially formulate hypotheses that includes expected change rate and shape of all the groups that are expected to be found (Ram & Grimm, 2009), in order to model appropriate growth factors. In the present case these included: (a) one trajectory characterizing women with elevated symptoms limited to the pregnancy period; (b) one trajectory of early postpartum onset and a gradual recovery; (c) a stable trajectory at a moderate level with pregnancy onset in which symptoms continue into the postpartum period; (d) a small group of women with a very high symptom level throughout the period of study;

(e) a majority of women presenting minimum symptoms. The next step in the data analyses process is to obtain a single-class baseline growth model (Bollen & Curran, 2006). To evaluate how the respective basic LGC models fit the data, fit indices of the various models should be inspected and compared, as well as considering whether the individual model allows for the change processes described in the hypotheses set forth (Ram & Grimm, 2007). As shown in Table 1, we began by fitting a basic linear model, however, the fit indices showed a poor fit, moreover, it does not accommodate any deviations from a purely linear symptom development across the perinatal period and are thus not in accordance with hypothesis. In the next step a quadratic growth curve model was fitted, this model was also rejected as it did not show good enough fit indices. The linear spline model (Ram & Grimm, 2007), which included a linear growth curve during pregnancy and freely estimated postpartum measurement points showed a reasonable fit, and was deemed a viable choice for further analyses. However, as this was a rather complex single-class model, more parsimonious models were also tested. In the next step, we tested two piecewise models, with transition points either at T4 (last pregnancy measurement point) or T6 (first postpartum measurement point). Although an improvement on the linear and quadratic models, the piecewise model with transition point at T6 had only nearly an acceptable fit. Further, by modeling the transition point in either pregnancy or postpartum, one of the curves would be anchored outside the period to be modelled (i.e. the postpartum curve would have its' initial measurement point during pregnancy). Both fit indices and theoretical consideration thus indicated that adjustments should be made, and we fitted a three-piece piecewise model (Flora, 2008). In this model, we could include two transition points, thus effectively being able to model the pregnancy, the perinatal and postnatal periods as three separate linear curves. This model showed excellent fit. In comparing this model with the linear spline, the three-piece model had the advantage of being easier to interpret as well as superior fit indices. By allowing for sharp transitions at the last measurement point during pregnancy and the first time point postpartum, the statistical model was able to represent the theoretical expectation of differential change rates during these three periods (Ram & Grimm, 2007), i.e. a pattern of rapid change in symptom levels during the peripartum phase and relatively slower change during the pregnancy and postpartum phases.

Table 1

Fit Indices of Conventional Latent Growth Curve Models

Model	χ^2[df]	CFI	TLI	RMSEA [90% CI]
Linear	188.04 [23]	0.860	0.872	0.083 [0.072, 0.094]
Quadratic	165.85 [19]	0.875	0.862	0.086 [0.075, 0.099]
Linear spline	74.95 [20]	0.953	0.951	0.051 [0.039, 0.064]
Two-piece T4	173.23 [19]	0.869	0.855	0.089 [0.077, 0.101]
Two piece T6	157.35 [19]	0.882	0.870	0.084 [0.072, 0.096]
Three-piece	27.00 [14]	0.989	0.983	0.030 [0.012, 0.047]

Note. df = degrees of freedom; CFI = Confirmatory Fit Index; TLI = Tucker–Lewis Index; RMSEA = root mean square error of approximation.

After establishing an appropriate single class latent growth curve model, a series of GMM models should be estimated. According to recommendation in the methodological literature (Nylund, Asparoutiov, & Muthen, 2007), GMM analyses were conducted in a stepwise fashion beginning with a single class model and increase number of classes by one in each subsequent step. To decide on number of classes, the bootstrapped likelihood ratio test (BLRT), the Lo–Mendell–Rubin adjusted likelihood ratio test (LMR-LRT), and the Bayesian information criteria (BIC) / sample size adjusted BIC (SABIC) were evaluated (Nylund et al., 2007). Further, entropy values, which represent the quality of classification of individuals into latent classes were also inspected (Celeux & Soromenho, 1996) along with overall interpretability (i.e. excluding classes with less than 20 participants). Further, we constrained variances and the shape of change to be equal across classes (Ram & Grimm, 2009).

There has been a debate on when to include predictor variables, either in the same step as the estimation of classes, or in a separate step after the class structure has been established. A recent simulation study concluded that it is preferable to include predictors in a separate step, as predictors otherwise may unduly affect class structure (Nylund-Gibson & Masyn, 2016). Class membership was thus regressed on predictors in a separate step after the class structure had been established. This was done by estimating multinomial logistic regression models using the three-step modal ML approach as this approach further accounts for class assignment uncertainties (Asparouhov & Muthen, 2014; Vermunt, 2010). When estimating class structures, the fit of each individual trajectory to all of the class trajectories are estimated. This implies that although all women are assigned to one class, the fit to the assigned class as well as the remaining classes are estimated for all individuals, thus providing a measure of class uncertainties. These estimates are taken into account when

estimating predictors of the latent classes by means of multinomial logistic regression models.

4.6.2 Autoregressive latent trajectory (ALT) modeling

To examine the interparental relations of depressive symptoms unfolding the perinatal period described in Paper II, specifically the potential transmission of depressive symptoms between parents, the typical choice would have been an autoregressive cross-lagged panel model. As we were interested in the cross-lagged estimates, it is important to account for the stability of maternal and paternal depressive symptom levels, respectively, within the model, otherwise we would run the risk of biased cross-lagged estimates. The inclusion of autoregressive paths do this to a certain extent, as it controls the cross-lagged estimates of the prior level within the same process i.e. maternal depressive symptoms. This is especially important if maternal and paternal symptom levels have different stability properties at the various measurement points. However, it is likely that the symptoms levels also comprise trait-like, time-invariant stability, in which case the autoregressive paths do not account for this to a satisfactory degree as it fails to separate the within-person and between-person effects (Berry & Willoughby, 2017; Hamaker, Kuiper, & Grasman, 2015). Omitting this aspect from the model, you run the risk of the between-person variance (i.e. variances in mean levels and slopes of depressive symptoms) being forced to become manifest in the cross-lagged coefficients. This would render these estimates difficult to interpret as the within-person and between-person variance would be lumped together (Berry & Willoughby, 2017). Consequently we chose an autoregressive latent trajectory (ALT) modeling approach. ALT models combines the strengths of growth curve modeling and autoregressive cross-lagged analyses within a structural equation modeling framework (Bollen & Curran, 2004). This approach is particularly suited to examine the hypotheses regarding depressive symptom contagion between parents throughout the perinatal period as it provides an opportunity of examining the time-adjacent effects among parents while at the same time avoiding potential bias that could result if the overall stability factors (i.e. parents' mean level and change over time) had not been simultaneously modelled (Berry & Willoughby, 2017; Hamaker et al., 2015).

Prior to evaluating the hypotheses in the study, univariate ALT models were modeled for each parental process separately and compared with univariate autoregressive models and latent growth curve models, as recommended by Bollen and Curran (2004). Inspecting the fit indices presented in Table 2, it is clear that for both mothers and fathers, ALT models represent a better fit of data to the models. In the next step a bivariate ALT model were

compared with an autoregressive cross-lagged model and a latent growth curve models with two parallel processes. Again, the ALT model showed superior fit. To obtain a more parsimonious model within-time correlations were fixed to equality. This model was compared with the full ALT model by chi-square difference test, and as there were no significant differences between models ($\Delta\chi^2(5) = 6.40$) the more parsimonious model was chosen.

Table 2

Fit Indices of the Univariate and Multivariate Latent Curve Models (LCM), Autoregressive Models and Autoregressive Latent Trajectory (ALT) Models

Models	χ^2 (df)	CFI	TLI	RMSEA[90%CI]
Mothers				
1a. LGC, linear model	195.37 (23)*	.855	.867	.085 [.074, .096]
2a. Autoregressive model	206.99 (15)*	.838	.773	.111 [.098, .125]
3a. ALT, full model [1]	27.95 (14)*	.988	.982	.031 [.013, .048]
Fathers				
1b. LGC, linear model	31.39 (23)	.985	.986	.020 [.000, .036]
2b. Autoregressive model	133.94 (15)*	.786	.700	.094 [.080, 109]
3b. ALT, full model	13.38 (14)	1.000	1.002	.000 [.000, .031]
Both parents				
1c. LGC, linear model	304.09 (91)*	.908	.908	.048 [.042, .053]
2c. Autoregressive cross-lagged model	444.44 (60)*	.833	.749	.079 [.072, .086]
3c. ALT, full model[1]	78.79 (56)	.990	.984	.020 [.008, .029]
4c. ALT fixed within-time correlations	85.19 (61)	.989	.984	.020 [.008, .029]

Note. χ^2 = chi-square test of model fit; df = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval.
*$p \leq 0.01$
[1]Variance of mother's slope was not significant and set to zero to allow for convergence of the model.

In the final multivariate ALT, we modeled maternal depressive symptoms by random intercept and linear slope in addition to the autoregressive effects of prior levels of maternal symptoms, and the cross-lagged effects of prior levels of paternal depressive symptoms. Paternal symptoms were modelled correspondingly within the same model. Following Bollen and Curran (2004), the baseline observed variables were treated as predetermined and not included in the intercept and slope factors. Further, the latent factors and baseline observed

variables were allowed to correlate, along with within-time residuals. The linear slopes were parametrized in weeks.

To explore whether partner-related attachment moderated these relations multi group analyses were performed on the ALT model to compare groups of insecurely and securely attached couples. Following Hussong, Hicks, Levy, and Curran (2001), a fully constrained model was compared with a model in which the cross-lagged parameters were relaxed. Chi-square difference tests evaluated resulting model change. Post hoc tests of each path were performed to identify which of the cross-lagged parameters that were significantly different across groups.

4.6.3 Confirmatory Factor Analysis and Measurement Invariance

Confirmatory factor analysis (CFA) were performed in relation to both measurement invariance analyses and to model separate pre- and postnatal depressive symptom factors for mother and fathers respectively, both are analyses included in Paper III. The idea behind CFA is that your measurements, or indicators as they are called in this context, are conditional on a latent concept that we are not able to measure directly. The indicators thus become an indirect way of measuring the latent concept of interest. In a SEM framework, we are able to separate the covariation between indicators, which are thought to be explained by the latent factor, and the remaining error variance in the model.

When using questionnaire data, it is often the case that a large number of items loads onto the same underlying factor. Reducing indicators through parceling has been shown to provide superior tests of structural model parameters as the constructs are defined more precisely (Little, Rhemtulla, Gibson, & Schoemann, 2013). As EPDS consist of 10 items, we constructed three parcels that functioned as indicators for the latent variables of depressive symptoms. However, the practice of parceling has also been criticized for not taking into account the variability in estimates created by the specific item-to-parcel allocation (Sterba & MacCallum, 2010). To reduce this potential bias, we followed suggestions in the methodological literature by re-estimating all models repeatedly with varying item-to-parcel allocations (Sterba & MacCallum, 2010). More specifically, the average of fit indices across 20 identical models with different randomly selected allocations are reported.

In longitudinal studies using repeated measurements it is important to ascertain whether the repeated measures in fact does measure the same at the different measurement occasions. Measurement invariance analyses were performed to investigate whether EPDS measured depressive symptoms similarly across the seven waves of data collection points, specifically undertaken to examine potential differences between pre- and postnatal periods.

Towards that end, we conducted confirmatory factor analyses on all time points, using the parceling strategy described above, in order to perform tests of measurement invariance. First, we tested for configural invariance by constructing latent factors based on the three parcels for each wave, with correlated factors and correlated error variance of identical parcels at different time points (Widaman, Ferrer, & Conger, 2010). Next, weak measurement invariance was tested by constraining factor loadings to be equal across time points. Strong invariance was then tested by additionally constraining intercepts of the parcels to be equal across time. To compare model fit across the models, chi square differences between models were evaluated, along with Confirmatory Fit Index (CFI), Tucker–Lewis Index (TLI), and the root mean square error of approximation (RMSEA), with CFI and TLI values greater than .95 and RMSEA values lower than .06 indicating good fit (Hu & Bentler, 1999; Widaman et al., 2010).

Depressive symptom factors (four in total) were modeled to investigate potential differences among parents and perinatal periods in associations with child outcomes. Prenatal latent depression factors were constructed for mothers and fathers, separately, loading on the four EPDS composite scores during pregnancy. Likewise, postnatal depression factors were modeled for both mothers and fathers, based on the three EPDS composite scores measured at 6 weeks, 6 months and 12 months after birth. When modeling the pre- and postnatal depressive factors, all factor loadings were constrained to be equal, thereby forcing depressive symptoms at all the time points to be equally important indicators for the latent factor of depressive symptoms. Imposing equality across time thus prevents any bias in how the timing of symptoms is related to the factor.

4.6.4 Cholesky Factorization

Cholesky factorization was used in Paper III to estimate the unique contribution of pre- and postnatal depressive symptom factors, respectively, on child outcomes in the context of potential multicollinearity problems (de Jong, 1999; Lervåg, Bråten, & Hulme, 2009). Multicollinearity can arise when highly correlated variables are included in the same regression model. The potential for any one variable to contribute with unique predictive information decreases as the correlation between included variables increases, to the point that it may cause unstable coefficients that may change in magnitude, rendering them uninterpretable (Langballe, Innstrand, Hagtvet, Falkum, & Gjerløw Aasland, 2009). Cholesky factorization solves potential problems with multicollinearity by decomposing the variance of the latent factors, in an order consistent with the proposed hypothesis (de Jong, 1999). In a first step, the predictor that the remaining variables are adjusted for, is modeled without

decomposing any variance. In the next subsequent steps, each of the remaining variables are modeled in the hypothesized order, by regressing them as phantom factors, without any indicators of their own, and with the sole purpose of decomposing the variance in a sequential order (de Jong, 1999). In this thesis, Cholesky factoring thus provides information about the additional amount of variance accounted for in child outcomes by prenatal depressive symptoms after postnatal depressive symptoms were accounted for, equivalent to hierarchical regression or fixed-order regressions in standard regression analyses.

4.6.5 Mediation and Moderation Analyses

Mediation analyses were performed to investigate whether parenting stress mediated the relation between parental depressive symptoms and child outcomes in Paper III. To test for mediation indirect paths from each predictor factor, through the mediating variable to outcome variables were estimated. As recommended by the methodological literature, standard errors of the indirect paths were estimated based upon bootstrapped bias-corrected 95% confidence intervals with 5,000 draws (Hayes, 2009).

Moderation analyses refers to situations in which the strength of an association between two variables is dependent on another variable, thus differing with various values of that variable, and were performed in Papers II and III. To investigate whether partner-related attachment styles moderated the relation between partners' depressive symptoms in Paper II, multi-group analyses were performed to compare ALT model estimates from insecurely and securely attached couples. Following Hussong et al. (2001), a fully constrained model was compared with a model in which the cross-lagged parameters were relaxed, as indicated by χ^2-differences. Post-hoc tests of each path, using one degree of freedom, tested which paths that were significantly different between groups.

In Paper III, moderation analyses were conducted to investigate whether each parent's postnatal depressive symptoms would moderate the relation between the other parent's depressive symptoms and child outcome. This was done in accordance with the latent moderated structural equations (LMS) approach (Klein & Moosbrugger, 2000), using the XWITH command in Mplus. This approach is an adaptation of moderation analyses to SEM-analyses using latent factors, as the LMS estimation procedure takes the distributional characteristics of the nonnormally distributed joint indicator vector that arises in latent interaction models, explicitly into account (Klein & Moosbrugger, 2000).

4.6.6 Missing data

Missing data were handled by the full information maximum likelihood procedure (FIML) accounting for missing at random (MAR) assumptions. FIML procedures deal with

missing data, do parameter estimation and calculate standard errors in a single step using all available information, and is a recommended strategy for dealing with missing data in SEM analyses (Graham, 2009). MAR assumptions allows for differences among participants with missing and non-missing data on responses at any or all occasions prior to dropout, thus missingness can be controlled for by variables present in the dataset. This is in contrast to missing not at random (MNAR), referring to differences between participants with missing and non-missing data that can not be controlled for by other variables present in the dataset (Graham, 2009), because it depends on unseen responses after participants drops out.

5 Results

5.1 Paper I: Patterns of pregnancy and postpartum depressive symptoms: Latent class trajectories and predictors

In the first paper, a growth mixture model of four distinct latent piecewise trajectory classes accounted for the heterogeneity of depressive symptom course among women during pregnancy and 12 months postpartum. The four classes were labeled according to trajectory characteristics as pregnancy only (4.4%), postpartum only (2.2%), moderate-persistent (10.5%), and minimum symptoms (82.9%). Referring back to our initial hypothesis about trajectory features, we found: (a) One trajectory with elevated symptoms limited to the pregnancy period; (b) one trajectory with stable low symptoms during pregnancy, rapidly increasing after birth with a gradual recovery the first postpartum year, termed postpartum only; (c) a trajectory characterized with moderately elevated symptom levels during pregnancy, with a slight increase in symptom burden postpartum; (d) no class with a high chronic trajectory, contrary to our expectations; and (e) one trajectory including the majority of women without elevated depressive symptoms, as evident in the minimum symptoms class. Regarding our second aim, all psychosocial adversity factors as well as education distinguished the elevated trajectory classes from the minimum symptoms class. Further, in accordance with previous research distinguishing between remitting and chronic courses of postpartum depression (Vliegen et al., 2014), the moderate-persistent class showed the highest number of associated psychosocial adversity factors. Overall, our findings were consistent with our hypothesis of heterogeneity in pathways of elevated depressive mood during pregnancy and the first postpartum year, connecting distinct trajectories of time courses with differential psychosocial adversity factors.

5.2 Paper II: Depressive symptom contagion in the transition to parenthood: Interparental processes and the role of partner-related attachment

In line with our hypothesis, we found that mothers' depressive symptoms at the end of pregnancy predicted fathers' level of depressive symptoms six weeks after the baby was born. The statistical modeling approach used takes time-invariant confounding into account, thereby rendering interpretations such as assortative mating, common time-invariant contextual influences or shared health habits unlikely, as these mechanisms would have been manifest as time-invariant components in the model. Thus, the finding indicates depressive symptom contagion in the perinatal period, in terms of a transmission of negative mood states from mothers to fathers around the time of birth. The effect size of the association was small; however, relations were tested in a conservative modeling context, as the ALT model takes into account both time-invariant confounding influences and effects from the individual's own prior depressive symptoms. Also in accordance with hypothesis, we found that partner-related attachment moderated the interparental relations of depressive symptoms: Maternal symptoms predicted subsequent paternal symptoms at additional time points during the latter part of pregnancy and throughout the first year after the baby was born in the insecurely attached couples group only. And fathers' depressive symptoms predicted mothers' symptom levels in mid-pregnancy also only in the insecurely attached group. It is notable that the association between maternal and paternal depressive symptoms at the time of birth was not moderated. This suggests that new fathers, regardless of attachment status, are vulnerable to depressive symptom contagion during this particular time.

5.3 Paper III: Parenting stress plays a mediating role in the prediction of early child development from both parents' perinatal depressive symptoms

In paper III, we found that parental depressive symptoms, which were highly stable over the perinatal period, predicted children's externalizing, internalizing, and dysregulation problems, as well as language developmental delay at 18 months of age. There was no evidence of independent prenatal effects on children's developmental outcomes, across a broad range of developmental domains. A differential effect was demonstrated, linking fathers' depressive symptoms to language outcomes, and mothers' symptoms to adverse effects in the social-emotional domain. There was no evidence of stronger associations between depressive symptoms and child outcomes when both parents showed high symptom loads. Examining parenting stress at 12 months as a broad mediator between both parents' postnatal depressive symptoms and child outcomes, we found that it mediated the relations between maternal dysphoria and both externalizing and dysregulation problems in the children, as well as paternal postnatal depressive symptoms and receptive communication in the 18-month-old children.

6 Discussion

6.1 General Discussion of Findings

The overall aim of this thesis has been to investigate the nature of depressive symptoms in the transition to parenthood for women and men, focusing on the heterogeneity of depressive symptom time courses of women in Paper I, the reciprocal relationship of depressive symptoms between parents in Paper II, and child outcomes of pre-and postnatal depressive symptoms in mothers and fathers in Paper III. The results provides new knowledge pertaining to three broader developments in the field; (a) the investigation of depressive symptoms and child outcomes during the entire perinatal period; (b) the inclusion of both parents in study of depressive symptoms in the perinatal period; (c) the need for the identification of parents with a heightened risk for prolonged depressive problems. I will discuss the findings in light of these perspectives in the following.

6.1.1 Depressive Symptoms and Child Outcomes during the Entire Perinatal Period

In recent years, studying depressive symptoms in the prenatal period in conjunction with the postnatal period has yielded insights into the onset of depression in this period of life, with findings indicating that for many women symptom onset happen early (Patton et al., 2015; Wisner et al., 2013), underscoring the advantages of studying the entire perinatal period. Our findings in Paper I are in accordance with the literature indicating that for a substantial number of women struggling with postnatal depressive symptoms, the timing of symptom onset happened during pregnancy or before (Wisner et al., 2013), as this is the case with two of the trajectory classes. In the pregnancy only class, symptoms were limited to the pregnancy period, whereas the moderate-persistent trajectory was characterized by elevated symptom levels from the outset and continuing throughout the first postpartum year. Moreover, studies have also indicated that there is considerable heterogeneity in perinatal depression (Cents et al., 2013; Mora et al., 2009; PACT Consortium, 2015; van der Waerden et al., 2015). This is also in accordance with our results, as we found several distinct trajectories of symptom courses suggestive of heterogeneity of symptom courses. Providing evidence of differential predictors to these symptom trajectory courses further supported this interpretation, providing an extension of the current literature. Notably, demonstrating a symptom course with heightened symptoms during pregnancy only was especially important, as there has been a lack of studies on heterogeneity of perinatal depressive symptoms with sufficient measurement points during pregnancy to map out symptom courses in this period. Including several measurement points during both the prenatal and postnatal period was thus

instrumental in order to both obtain knowledge on early symptom onset, as well as to obtain a detailed account of the heterogeneous symptom courses and related predictors.

Including both pregnancy and early parenthood phases is also important when examining potential interpersonal processes of transmission of depressive symptoms between parents, as was the focus of Paper II. The partner relationship is undergoing considerable changes in roles and identities as the couple becomes parents together. To be able to map out any reciprocal relations in depressive symptom development within the parental couple, measurements taken within both pregnancy and the early parenthood period is needed. We found that new fathers were vulnerable for their partner's depressive problems particularly around the time of birth regardless of risk status, while a subgroup of couples struggling with insecure attachment styles showed multiple instances of reciprocal depressive symptom contagion. These pathways and distinctions might not have been sufficiently clear had we focused exclusively on the postnatal period. Few other studies have investigated depressive symptom contagion within couples in the perinatal period, however, there is one study of such processes without reference to this specific period finding that women experience an elevation of symptoms following their husbands' depressive problems (Koutra et al., 2013). In this study cross-lagged relations were constrained to be equal across time points providing an estimate of transmission of negative emotion that is more generalizable across time. Our findings extend those by incorporating the specific events of pregnancy, childbirth and early parenthood, and model the timing of the transmission effects in relation to the perinatal events. Further, our findings also demonstrate a moderation effect, indicating that a subgroup of couples characterized by insecure adult attachment styles are more prone to reciprocal depression symptom contagion in this period.

There have been efforts to disentangle the effects of pre- and postnatal symptom loads in terms of child outcomes, elucidating different potential mechanisms for the transmission of risk, including shared genetic risk, fetal programming effects, direct exposure to parental depressed mood, behavior and parenting, and common contextual factors (Goodman & Gotlib, 1999; Hannigan et al., 2018; Pearson et al., 2013; Stein et al., 2014). When undertaking such endeavors, it is necessary to include the entire perinatal period with separate pre- and postnatal measurement points. In Paper III, we utilized both pre- and postnatal symptom factors to investigate a range of child social, emotional, language and cognitive outcomes. We found that pre- and postnatal symptoms correlated highly, and that depressive symptoms in either period predicted increases in externalizing and dysregulation difficulties, as well as language delays when the children was 18 months old. This is in line with the

literature, as both social-emotional functioning (Goodman et al., 2011; Ramchandani & Psychogiou, 2009; Velders et al., 2011) and language delays (Paulson, Keefe, & Leiferman, 2009; Quevedo et al., 2012) have been associated with perinatal parental depression. We did however, not find an association with cognitive development, in contrast to earlier studies that have demonstrated such a link (Sutter-Dallay et al., 2011). Our findings further indicated that the prenatal symptoms did not explain any unique variance in child outcomes beyond what was explained by postnatal symptoms, and although the study had potential to provide indirect evidence of prenatal effects of maternal symptoms on later child functioning, this was not the case with our study. In studies investigating prenatal associations with child outcomes while controlling for postnatal depressive symptoms, findings have been mixed (Hannigan et al., 2018; Pearson et al., 2013; Stein et al., 2014). Investigating parenting stress as a potential mediating factor between parental depressive symptoms and child social-emotional and language outcomes, we found that there was an indirect effect through parenting stress in most of the relations. This is in accordance with studies showing associations between parental depression and parenting (Field, 2010; Lovejoy et al., 2000), and studies demonstrating the impact of parenting on child outcomes (Barroso, Mendez, Graziano, & Bagner, 2017; Wang & Dix, 2013). However, our study were not designed to conclude among several potential mechanisms of risk transmission, i.e. shared genetic risk, common contextual factors are not included in analyses, and should be interpreted with that in mind.

Taken together, the findings in this thesis thus adds to the theoretical and empirical development in the field as conceptualizing the perinatal period as a continuous period in terms of important interpersonal and psychological processes, and underlines the advantages of including both pregnancy and early parenthood when investigating depressive symptoms in this period of life. However, this does not imply that specific perinatal events may not trigger or be exclusively associated with depressive symptoms under certain circumstances (i.e. depressive symptoms exclusively expressed during one perinatal period, paternal symptom rise following their partner's depressive problems towards the end of pregnancy). Rather, it implies that in order to delineate such specific associations as well as a more continuous symptom development including both pregnancy and postpartum periods is necessary.

6.1.2 Both Parents' Depressive Symptoms in the Transition to Parenthood

There are several advantages of including fathers in the study of perinatal depressive symptoms, as evident in Papers II and III in this thesis. First, to gain more knowledge on

paternal perinatal depressive symptoms is warranted, as this is understudied (Stein et al., 2014). Second, including paternal depressive symptoms further enables investigations into depressive symptom contagion processes or other partner dynamics pertaining to depressive symptom development (Kouros & Cummings, 2010; Rholes et al., 2011). Third, to continue to investigate child outcomes following paternal depressive states is needed (Ramchandani, Stein, et al., 2008), not to mention differential and combined effects of maternal and paternal symptoms (Paulson, Dauber, & Leiferman, 2006).

Depressive symptoms in the perinatal period is rarely experienced living alone, rather a majority of expectant and new parents live in a partner or family setting. Getting a more detailed understanding of the reciprocal development of depressive symptoms in the partner context may advance the knowledge of interpersonal determinants of depressive problems. Our findings in Paper II confirmed earlier knowledge on the concordance of depressive symptom levels among partners in the perinatal period (Cameron et al., 2016), and extended these to demonstrate an increased vulnerability of developing depressive symptoms for new fathers following their partners' heightened symptom levels towards the end of pregnancy.

Not only did both parents' depressive symptoms predict child outcomes in Paper III, there was a differential effect linking maternal symptoms to social-emotional outcomes and paternal symptoms to language outcomes. Connell and Goodman (2002) proposed that depressive symptoms may not affect parenting behavior in a uniform manner, providing a potential explanation of differential effects. Paulson et al. (2009), finding a differential pattern regarding child language outcomes, also measured the extent to which the depressed parent continued to read books for their children, and discovered that depressed mothers continued performing this parental task to a larger extent than did depressed fathers. This was thus offered as an explanation for the differential finding of language development following maternal and paternal depressive problems. Although, we have not measured time spent on parental tasks in our study, the notion of depressed mothers spending more time on parental tasks relative to depressed fathers, could provide potential interpretations of the association between maternal depressive symptoms and child social-emotional functioning. There is an established association between prolonged caretaking by a depressed parent and adverse child social-emotional development (Dix & Yan, 2014; Tronick & Reck, 2009). Given that children are exposed to depressed mothers for longer periods of time than depressed fathers, it could explain the comparably larger negative impact of maternal depressive symptoms on child social-emotional functioning.

Summing up, including both parents in the study of perinatal depression is not only important to gain more knowledge of paternal perinatal depressive problems and related child outcomes, although that is highly needed in its own right, but also to make more complex family dynamics pertaining to parental depressive symptoms more accessible for study.

6.1.3 Identifying Parents with at Risk for Prolonged Depressive Problems

Investigating vulnerability factors or groups beyond that of main effects is a necessary precondition to the development of more tailored or personalized approaches to prevention and treatment of depressive problems in the perinatal period. A more nuanced parsing of processes and factors underpinning depressive symptom development and maintenance is needed to identify those at risk for prolonged depressive problems. This is especially important to prevent aberrant child development, as infants are dependent on emotional availability in its caretakers (Dix & Meunier, 2009), which may be compromised by parental depression. Further, studies show that persistent depressive problems in parents predicts greater adverse child outcomes compared with more transient problems (Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007; Campbell, Morgan-Lopez, Cox, McLoyd, & NICHHDE, 2009; Vliegen et al., 2014).

This thesis, especially findings in Papers I and II details knowledge on risk factors and groups, and as these are measured early in pregnancy they provide an early identification opportunity. In Paper I we found that higher levels of pregnancy-related anxiety predicted a trajectory of elevated depressive symptoms during pregnancy. When the trajectory shape and predictors are interpreted together, this trajectory class characterized women with anxieties, worries and negative feelings revolved around being pregnant and anticipating childbirth and parenting. We also identified a small trajectory group with symptoms primarily postpartum, only differentiated from the baseline group by having a history of mental health issues. The moderate-persistent trajectory, however, was associated with a range of psychosocial risk factors. This symptom course was characterized by a rise in symptom levels throughout the first year after birth. The persistent and increasing nature of this trajectory suggests that this group could be more vulnerable for the development of persistent depressive problems (Vliegen et al., 2014). Prenatally there seem to be an accumulation of psychosocial risk factors, and with a persistent and rising trajectory shape a tenable interpretation may be that the tasks and strain accompanying early parenthood adds to the total burden and rather than recover from their depressive problems the symptom level deteriorate. Although symptom levels on average lies around cut-off for a clinical diagnosis of a depressive episode (using Norwegian cut-off criteria (Eberhard-Gran et al., 2001)), the symptom development course

warrants clinical and research attention. Persistent postnatal depression have been shown to have greater adverse effect on developmental outcomes for the children, as well as long-term effects on the mothers' own mental health (Racine et al., 2018; Vliegen et al., 2014), underlining the importance of identifying such groups.

Looking at depressive symptom contagion processes in Paper II, there was a clear difference of the magnitude of reciprocal depressive symptom transmissions between groups with couples characterized by secure and insecure adult attachment styles. The couples with insecure attachment styles showed transmission of depressive symptoms throughout the period and included both directions, suggesting that contagion effects were present at most time points. This suggests that insecure attachment style is a vulnerability factor for experiencing a rise in depressive symptom following depressive symptom problems in your partner. As insecure attachment is thought to reflect inner working models of the self in relation to others, it is reasonable to assume that being characterized by insecure adult attachment styles entails a susceptibility to the mood and behavior of intimate others, thus a heightened vulnerability in terms of developing symptoms when exposed to depressed mood and behavior in intimate relations. This might also have implications for the persistence of depressive states, as such negative feedback processes might play a part in the maintenance of symptoms when both partners in a couple is characterized by insecure adult attachment styles. The difference among attachment groups as shown by the moderation effect thus paints a picture in which all new fathers seem to be vulnerable around the time of childbirth, whereas a subgroup of couples remain vulnerable throughout the period.

6.2 Methodological Considerations

The findings in this thesis must be considered in the context of several methodological strengths and limitations. First, the study includes a population-based, multisite sample, strengthening the generalizability of findings. Further, it has a longitudinal design enabling us to study change over time, as well as longitudinal associations. Second, there are several measurement points during pregnancy, allowing for the study of the development and course of symptoms during pregnancy, which is rare in this field. Third, the inclusion of paternal data in parallel with maternal data broadens the scope of the thesis, as well as making potential family dynamics more accessible for study. Fourth, this is a multimethod, multi-informant design, including both surveys and psychological test data.

Obviously, there are also methodological limitations with implications for how we should interpret the findings of this thesis. These should be discussed and are presented under

subtopics of (a) measurement issues, (b) inner validity, (c) statistical conclusion validity, and (d) representativeness and generalizability.

6.2.1 Measurement Issues

The EPDS was used to measure depressive symptoms in both women and men. It is a self-report questionnaire, and there is no available information on clinical diagnosis. It was originally developed for the use among postpartum women (Cox et al., 1987), but have been validated for use among men, and has been widely used among male populations (Matthey et al., 2001). It has been criticized for not taking typically male symptoms of depression into account, as well as measuring worry, anxiety and unhappiness rather than pure depression (Madsen & Juhl, 2007; Massoudi et al., 2013). Cronbach's alpha in this study was found to be at an acceptable level, however, lower than for the maternal variables. Administrating EPDS to the male participants might thus have led to an underestimation of depressive symptoms among male participants, and caution is advised in directly comparing male and female levels of depressive symptoms.

Partner-related attachment or adult attachment style was measured by the ECR. Adult attachment may be measured by interviews such as the Adult Attachment Interview (AAI), or self-report measures, representing to different traditions within the attachment literature, with the self-report measures pertaining to the social and personality psychological tradition, whereas interviews have more often been used in the developmental field (Roisman et al., 2007). While the attachment concepts measured in either tradition is related, they do not completely overlap, and should not be considered to measure the exact same phenomenon within the person, thus some caution should be taken in comparing results with studies using differing measurement approaches to adult attachment. Within the self-report tradition, the ECR have shown excellent psychometric properties (Fraley et al., 2000).

Parenting stress was measured by Parenting Stress Index, total stress score, which is a combination of the parent and child domains. By using the total stress score, the specificity of using the parent and child composite scores are lost. The PSI was used as a mediating variable, and it could have been interesting to look further into the details of parenting stress as the mediator in an indirect pathway between parental depressive symptoms and child outcomes. However, as the model was already complex, we decided to include the total scores.

Several of our measurements are self-reports, this includes the depressive symptoms measurements, the predictor variables in Paper I, parenting stress measurements in Paper II and measurements of adult attachment styles in Paper III. Relations between these may thus

be inflated due to shared method variance. Likewise, social-emotional functioning in children when they were 18 months old were measured by questionnaires (ITSEA) to their parents, and may also be subject to shared method variance. However, the Bayley-III were measured by objective observers, and exhibits relations to depressive symptoms comparable to those of the ITSEA.

6.2.2 Internal validity

Internal validity refers to the extent to which a study provides support to make claims of causal relationships between variables in the study. It is necessary to fulfill three criteria to make valid causal inferences: (a) temporal precedence, (b) covariation, and (c) there being no other plausible alternative explanations for the observed covariation (Shadish, Cook, & Campbell, 2002). The gold standard for making causal inferences is a true experimental design. Studying elevated trajectories of perinatal depressive symptoms, depressive symptom contagion processes and outcomes of parental depressive symptoms on children an experimental design would not be ethically justifiable. With experimental design not being an option, it can be argued that prospective, longitudinal design might be the best alternative to approximate a high internal validity. The longitudinal design of this thesis permits analyses in which the temporal order of variables are accounted for. Further, the criteria of covariation of variables have been demonstrated in each of the papers. The third criteria of ruling out alternative explanations, however, has not been fulfilled. We can not know whether there are variables, other than those included in the study that can account for the covariation we have demonstrated. In Paper I, there might be other predictors than those we have measured that predict elevated trajectory memberships. In Paper III, there might be other factors that account for the relation between parental depressive symptoms and child outcomes, i.e. shared genetic risk or common contextual factors. The ALT-modeling approach used in Paper II, however, have been proposed to approximate causal relations by disentangling within-person and between-person design, as within-person relations implies that measured and unmeasured time-invariant confounds are controlled for by statistical design (Berry & Willoughby, 2017). In this case, relevant time-invariant confounds that are controlled for, include factors such as assortative mating, common contextual factors or shared habits and beliefs. This leaves the possibility of other time-varying external factors explaining the time-specific relations in the model. Although this is not sufficient to claim a causal relationship, the model is deemed to have strong internal validity, and superior to the more traditional autoregressive cross-lagged panel model (Berry & Willoughby, 2017).

6.2.3 Statistical conclusion validity

Statistical conclusion validity refers to the extent to which the conclusions about the relationships between the variables in the study are reasonable (Cozby, 2009). Common threats to the statistical conclusion validity are low statistical power and violations of underlying assumptions of selected modeling approaches.

Although analyses in this thesis are based on a relatively large sample size (N = 1,036), there are instances in which we have to consider the statistical power. In particular, this is relevant to the growth mixture modeling analyses conducted in Paper I. The resulting class solution included two small classes of 2.2 % and 4.4 % of the total sample. Relating these classes to predictors by means of multinomial logistic regression analyses, low statistical power should be considered. This may, to a lesser extent, also be the case with the moderation analyses conducted in Paper II, in which groups of securely and insecurely attached couples were compared, as the insecure attachment style group was rather small (n = 158). Statistically significant associations and group differences are more difficult to detect in smaller samples, this increases the likelihood of not rejecting a false null hypothesis (committing a Type II error). However, research on clinical or at risk groups often implies small samples. Thus it is important to be careful not to conclude or generalize from statistically insignificant findings particularly with small sample sizes.

Another important methodological discussion relevant for the statistical conclusion validity is the controversy regarding growth mixture modeling as a method of identifying unobserved trajectory groups. Two main lines of criticism have been (a) the potential violations of underlying assumptions and (b) the interpretation of the trajectory groups. Bauer (2007) and Bauer and Curran (2003) have extensively discussed the limitations of GMM, and possible consequences of violating the underlying assumptions. One of the most important assumptions states that there should be within-class normality. A combination of several classes could, however, result in a non-normal distribution of the combined classes. If the non-normality have other origins than being a composite of several (normally distributed) classes, GMM approaches run the risk of extracting too many classes, including a multiclass model when in fact a one class model is more appropriate. In light of this, it has been warned against equating the process of choosing the number of classes with a validation of the resulting class structure, as such validation must be found elsewhere (Sher, Jackson, & Steinley, 2011; Steinley & Brusco, 2011). Responding to the criticism, Nagin and Tremblay (2005) underlined the approximating role of trajectory groups, and point out that although the trajectory groups may be literally distinct groups, more often they are not, and should then be seen as heuristic approximations. Even if it is the case that the population heterogeneity of

individual-level trajectories is a continuous distribution (i.e. not containing distinct groups), they argue that using group trajectories can have advantages to basic growth curve models. One of these advantages is that groups can be used as a method to approximate a continuous distribution of developmental trajectories when this distribution is unknown, including identifying small and unusual growth trajectories that would not have been identified with a LGC approach. Consequently, Nagin and Tremblay (2005) encourage the use of trajectory groups, highlighting the benefits of being able to identify unusual trajectories not found in aggregated analyses and relate these to other variables, however, they do warn against a reification of the groups in the interpretation of results.

Relating this debate to the analyses done in this thesis it becomes clear that the matching of change theories and articulated hypotheses in the perinatal period with latent growth factors capable of detecting the potential symptom developments was an important step in trying to prevent misspecification of the model. Regarding validation of resulting classes, the fit indices alone was not be taken as evidence of the class structure. Rather, it can be argued that the associations with predictor variables supported the interpretation of the shape of the trajectories and overall class structure, by representing differential associations with the classes. Further, findings were interpreted in context of the extant literature, relating classes to external sources of validation.

6.2.4 Representativeness and Generalizability

All three papers in this thesis are based on the *Little in Norway* sample. The response rate was 50.7%, and as in any population-based studies, there might be self-selection bias with an overrepresentation of healthy participants, and in this specific context an underrepresentation of those struggling with depressive problems. This might have led to underestimation of depressive problems as those with elevated levels of depressive symptoms, and might limit generalizability of results. However, it has been shown that self-selection bias affects the prevalence of what is being measured, while relations between measured variables remain largely unaffected (Nilsen et al., 2009). Further, late recruitment may also be a concern, as it led to a sizable proportion of missingness during the initial part of pregnancy. This may cause more uncertainty of estimates from this time period, however, late recruitment was due to choosing to visit a general practitioner for early pregnancy check-ups, rather than the well-baby clinic, and it is unlikely that this choice is related to depressive symptom levels. A related concern is selective drop-out, depressive symptom was related to attrition in this thesis. However, by using FIML in analyses all three papers we tried to reduce the impact of selected attrition. Further, the specific Norwegian context might also give rise

to questions regarding generalizability. Parents receive one year paid parental leave, including a paternal quota of 14 weeks as a statutory right (in the period of this study). Statistics Norway (2017) reports that for children born in 2011 and 2012, about 70% of all fathers in Norway stayed at home the 14 weeks that are reserved for fathers, or longer. This indicates that most fathers spend considerable amount of time with their infants and take part in care giving activities. Our findings might not be generalizable to context with less generous social policies in general nor to contexts lacking facilitating policies for new fathers in particular.

6.3 Implications and Future Directions

There are several clinical and research implications of the findings in this thesis. Demonstrating heterogeneity in depressive symptom courses during the perinatal period for women, the findings provide a basis for researchers and clinicians to inquire about possibly diverse underlying mechanisms, pathogenic pathways and prognosis. Further, as findings indicate that the experience of symptoms during pregnancy is common, the pregnancy period should be treated as a pertinent period for future clinical and research work.

The thesis further underlines the advantages of including fathers in studies on depressive symptoms in the perinatal period. Clinicians need to be aware of new fathers' vulnerabilities and needs in instances of maternal depressive states in the time around birth, and direct preventive and treatment efforts to both parents. Working to prevent symptom development in the initially unaffected parent is especially important considering newborns' need for emotional availability of their caretakers. In future studies, increased efforts in understanding temporal processes of perinatal depressive symptom development within a couples' context are needed, as well as a broader investigation into interpersonal determinants of perinatal depressive problems. Moreover, the link between fathers' depressive symptoms and delayed language development in their toddlers indicate the need for preventive and treatment efforts to affected families, as well as research efforts to understand the transmission of risk among both parental depressive problems and their children.

Specific attention should be directed to identify parents susceptible for the development of persisting depressive problems. In this thesis this has been indicated by an aggregation of psycho-social risk factors early in pregnancy, as indicated by the predictors of membership in the moderate-persistent trajectory group. Further, parental couples characterized by insecure partner-related attachment styles may also have a heightened risk for prolonged depressive problems as indicated by ongoing depressive symptom contagion

processes throughout the perinatal period. In future studies there is a need for a refinement of indicators of heightened risk for prolonged depressive problems, as this may inform clinical efforts to alleviate suffering for parents and children.

The findings of adverse child outcomes following parental depressive symptoms suggests areas of child and family functioning that may be affected in context of perinatal parental depressive states, and thus warrants clinical attention. As most outcomes were mediated by parenting stress, the investigation of parenting factors continues to be an important avenue for research in this field, as well as both preventive and treatment aspects of clinical efforts. Research efforts aimed at investigating early interventions to prevent and treat prenatal depressive problems are needed, as many show a high stability of symptoms throughout the perinatal period, it may reduce suffering for parents and children to shorten the period of elevated symptoms. In future studies, advancing knowledge of longer term outcomes for the children is recommended, as well as investigating the effect of chronic exposure to parental depression. Moreover, a continued focus on understanding the underlying mechanisms of risk transmission is central to advancing knowledge in this area of research. From a clinical perspective, a more detailed understanding of perinatal depression is warranted to be able to tailor targeted preventive and treatment efforts in order to reduce depression during this defining period in life for new mothers, fathers and babies.

7 Conclusions

A detailed investigation into patterns, processes and child outcomes related to depressive symptoms in the transition to parenthood have advanced the knowledge in the field on several accounts. Our findings support the conceptualization of heterogeneity of perinatal depression for women, by identifying four distinct classes of depressive symptom courses and relating these to specific predictors. Two classes, pregnancy only and postpartum only, showed rapid changes in symptom levels in close proximity to childbirth, while a third class, moderate-persistent, evinced a more gradual symptom development with an increase of symptoms postpartum. This latter trajectory course was linked with a range of psycho-social risk factors, while the former were associated with fewer and more specific predictors. Further, we have demonstrated depressive symptom contagion processes among partners in the perinatal period. Fathers are susceptible for heightened depressive symptoms in the time after birth in instances of elevated maternal symptoms towards the end of pregnancy. For couples characterized by insecure adult attachment styles, reciprocal transmission of depressive symptoms are evident throughout the perinatal period, rendering these families more prone to the development of longer term depressive problems. Finally, we have

demonstrated adverse child outcomes following perinatal depressive symptoms in language and social-emotional domains at child age 18 months, mostly mediated by parenting stress. A differential effect was found, linking maternal symptoms to social-emotional outcomes, while paternal symptoms were linked with language outcomes. Taken together, these findings provide new insights into the conceptualization of the heterogeneity of perinatal depression for women, interpersonal determinants of perinatal depressive symptoms, and mediated and differential associations with adverse child outcomes following depressive symptoms in the transition to parenthood for both parents.

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Depressive Symptom Contagion in the Transition to Parenthood: Interparental Processes and the Role of Partner-Related Attachment

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Abstract

How depressive symptoms unfold within a couple during the perinatal events of pregnancy, childbirth, and early parenthood is poorly understood. In this prospective study, we aim to investigate the reciprocal relation between maternal and paternal depressive symptomatology, specifically how symptoms in one partner relate to subsequent symptom level changes in the other partner throughout the perinatal period. Further, we aim to identify couples that are particularly vulnerable to the development of disruptive processes of negative mood states. Data were collected from 1,036 mothers and 878 fathers participating in the *Little in Norway* study from mid-pregnancy until 12 months postpartum. Depressive symptoms were assessed at seven time points (four prenatally) in both parents. Partner-related attachment was measured early in pregnancy. By utilizing an autoregressive latent trajectory modeling approach, accounting for time invariant confounding, we found mothers' depressive symptoms late in pregnancy to predict elevated symptom levels in fathers 6 weeks after birth. No other time-adjacent effects were observed among partners at other time points or with the opposite directionality. However, moderation analyses revealed that in couples characterized by insecure partner-attachment styles, additional cross-lagged pathways were evident during pregnancy and throughout the first year of parenthood. Clinicians need to be aware of fathers' vulnerability to symptom development in instances of maternal perinatal depressive states at the time around childbirth, and tailor preventive and treatment efforts to address both parents' needs. Further, particular attention should be directed to couples with heightened susceptibility to prolonged depression contagion processes.

Keywords: Perinatal depression, postnatal depression, partner-related attachment, autoregressive latent trajectory (ALT) models

General Scientific Summary: We found that heightened maternal depressive symptoms towards the end of pregnancy predicted elevated depressive symptoms in fathers after the baby was born. For couples with an insecure attachment style, such processes were also evident earlier in pregnancy and throughout the first year after birth. These findings underscore the importance of taking the partner of the affected parent into account when working to prevent and treat depressive states in the perinatal period.

Depressive Symptom Contagion in the Transition to Parenthood: Interparental Processes and the Role of Partner-Related Attachment

To experience perinatal depressive symptoms is not only disruptive for new parents, it also places children at risk of a variety of social, emotional, and developmental problems (Stein et al., 2014). While researchers and clinicians have recognized postpartum depression in women as a serious condition for decades, they have, until recently, given little attention to understanding and treating depressive symptoms during pregnancy, not to mention among new fathers (Howard et al., 2014; Underwood et al., 2017). Consequently, the dynamics of how depressive symptoms unfold over time within a couple during the transition to parenthood are poorly understood; however, advances in this field have the potential to yield clinical information that might translate into interventions suited to addressing both parents' symptom courses.

Meta-analyses show that maternal and paternal perinatal depressive symptoms are moderately associated (Cameron, Sedov, & Tomfohr-Madsen, 2016; Paulson & Bazemore, 2010). Attempts to explain depression concordance within couples include assortative mating, shared health beliefs and habits, common contextual influences, and depression contagion (Joiner & Katz, 1999; Meyler, Stimpson, & Peek, 2007). The latter refers to the transmission of negative mood states in the context of interpersonal relationships and as such highlights the interpersonal determinants of depression. It has been shown that increases in husbands' depressive mood predict subsequent elevations in their wives' symptom levels (Kouros & Cummings, 2010). However, there is a lack of studies examining this partner dynamic with respect to perinatal depressive symptoms and the specific events of pregnancy, childbirth, and early parenthood.

Becoming a parent involves considerable changes in personal identity, partner relationships, and family dynamics, creating new relational experiences relevant to the

attachment system. Attachment theory posits that the combination of insecure attachment and interpersonal stress may render individuals more prone to developing depressive symptoms (Bowlby, 1980). Following this line of reasoning, one would expect that spousal depression would influence depressive symptomatology predominantly in insecurely attached partners; however, empirical studies on this issue are scarce. It has been shown that insecure attachment predicts persistent postnatal depression (McMahon, Barnett, Kowalenko, & Tennant, 2005). Further, Rholes et al. (2011) showed how anxious and avoidant attachment orientations interact with interpersonal factors among spouses in predicting depressive symptoms during the first two years of parenthood.

With perinatal depression on the rise (Pearson et al., 2018), and studies documenting that partners rarely are being included in perinatal depression treatment efforts (Alves, Martins, Fonseca, Canavarro, & Pereira, 2018), more comprehensive knowledge of how depressive symptoms unfold within the parental couple during pregnancy and early parenthood are called for. Key aspects include understanding the roles and needs of fathers and identifying couples who are vulnerable to prolonged disruptive processes when struggling with depressive symptoms. In our theoretical model of perinatal depressive symptoms, we propose that symptom development is governed by both a stable tendency of affective mood over time and time-specific changes unfolding in the perinatal period. Our primary goal was to investigate whether either parent's depressive symptoms at one time point predicted the depressive symptom level of the other parent at the subsequent time point, thus delineating time-specific changes in symptom development. We expected stronger effects around and after birth, as the emotional interdependence and reciprocity in the relationship are likely to increase in this period. Further, we expected mothers' symptom levels to have a greater effect on fathers than the opposite case, as maternal and fetal well-being are of central concern in this specific period. Moreover, we aimed to investigate

whether partner-related attachment moderated these relations, and expected stronger effects with insecure partner-related attachment styles. Disentangling within-person and between-person effects by statistical design, we also aim to provide a basis for evaluating alternative interpretations of depressive symptom associations within the parental couple.

Method

Procedure and Participants

This study is based on data from 1,036 families participating in the prospective, multisite *Little in Norway* project. Pregnant women (N = 1,036) were recruited together with their partners (N = 878) at nine well-baby clinics during pregnancy (estimated response rate 50.7%), and were followed until the end of the first postpartum year, with four prenatal (mean gestational weeks 21, 28, 32 and 36) and three postnatal (6 weeks, 6 months and 12 months after birth) data collection points. Participant characteristics are presented in Table 1. There were three categories of missingness: late recruitment, as the sample was not full until T3; study withdrawal or participants lost to follow-up (n = 116); and intermittent missingness, as some participants missed one or more data collection points but participated at later data collection waves. Number of participants at each data wave is presented in Table 2. For a detailed account of data collection procedures, see Fredriksen, von Soest, Smith, and Moe (2017). Bivariate logistic regression analyses showed that high levels of mothers' depressive symptoms at T1 predicted attrition at T7 (OR = 1.09, 95% CI [1.05, 1.14], p < .001), whereas fathers' depressive symptoms at T1 and attachment levels were not related to dropout (p > .05). Written informed consent was obtained from all participants. The study was approved by the Regional Committees for Medical and Health Research Ethics in Norway (#2011/560).

Measures

Depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS) for men and women at T1 to T7. The EPDS is a 10-item self-report questionnaire measuring depressive symptoms during the previous seven days on a 4-point scale (range: 0–30). It was originally developed for mothers in the postnatal period (Cox, Holden, & Sagovsky, 1987) and has since been validated for prenatal use (Murray & Cox, 1990) and for men (Matthey, Barnett, Kavanagh, & Howie, 2001). Cronbach's alphas at the various assessments indicated adequate internal consistency in this study (range: .80 – .85 for women; .76 – .84 for men).

Partner-related attachment style was assessed by the Experiences in Close Relationships Scale (ECR) at T1. The ECR is a 36-item self-report measure of adult romantic attachment styles rated on a 7-point scale, yielding two subscales of underlying attachment: anxiety and avoidance (Brennan, Clark, & Shaver, 1998). When creating grouping variables for the moderator analyses, we used a categorization that ensured that the secure/insecure attachment status was emphasized, rather than the types of insecure patterns. Thus, participants scoring in the lower range on both subscales (cutoff on the 75^{th} percentile) were categorized as securely attached, while participants scoring above the cutoff on either one or both of the subscales were categorized as insecure. Couples in which both partners were categorized as insecurely attached comprised the insecure partner-related attachment group. The secure partner-related attachment group included couples with one or both partners being securely attached.

Statistical Analyses

To examine the relation between maternal and paternal depressive symptoms we used an autoregressive latent trajectory (ALT) model, which combines the strengths of growth curve modeling and autoregressive cross-lagged analyses within a structural equation modeling framework (Bollen & Curran, 2004). This model is particularly suited to examining

our hypotheses as it provides an opportunity to investigate the time-adjacent effects among parents while at the same time avoiding potential bias that could result if the parents' mean level and change over time had not been simultaneously modeled (Hamaker, Kuiper, & Grasman, 2015). More specifically, we modeled initial level and linear change in maternal depressive symptoms (MDS) by estimating random intercepts and slopes in a growth curve framework in addition to the autoregressive effects of prior levels of MDS and the cross-lagged effects of prior levels of paternal depressive symptoms (PDS). PDS were modeled correspondingly within the same model. The time-adjacent effects are thus based upon within-person variance only and the model does take into account confounding due to time-invariant covariates. Following Bollen and Curran (2004), the baseline observed variables were treated as predetermined and not included in the intercept and slope. Further, the latent factors and baseline observed variables were allowed to correlate, along with within-time residuals. The linear slopes were parametrized in weeks, providing an estimate of change in depressive symptoms within one week. Within-time residual correlations were constrained to equality. To explore whether partner-related attachment styles moderated the relation between partners' depressive symptoms, multi-group analyses were performed to compare ALT model estimates from insecurely and securely attached couples.

Missing data were handled using the full information maximum likelihood procedure (FIML) accounting for missing at random (MAR) assumptions. All data analyses were performed in Mplus version 7.3 using robust maximum likelihood estimation.

Results

Means, standard deviations, and correlations between study variables are presented in Table 2. The level of MDS increased somewhat during pregnancy and then declined slightly postpartum. The mean level of PDS remained relatively stable. At all time points, parental

depressive scores were positively correlated (range: r = .19 to .27) and partner-related attachment showed small to moderate correlations with depressive symptom scores.

Results from the multivariate ALT model are presented in Figure 1. Our theoretical model of symptom development proposed that the repeated measures of maternal and paternal depressive symptoms were governed by both a stable component across the perinatal period, as represented by the slope and intercept factors, and by time-specific changes represented by autoregressive and cross-lagged relations, with our primary interest being the cross-lagged relations. Maternal and paternal symptom trajectories had a declining (slope mean = -0.01, $p < .001$) and flat trend (slope mean = 0.00, $p = .717$), respectively. For both parents, initial status of depressive symptoms differed significantly between individuals, but not development over time. The maternal autoregressive estimates showed significant associations of prior depressive states with current depressive symptom levels across the entire perinatal period (β ranging from 0.17 to 0.39, $p < .01$, see Figure 1), while paternal autoregressive estimates were only significant in the initial part of pregnancy (β = 0.18 and 0.23, $p < .05$, respectively). However, data points were unevenly distributed across time; thus, the relative strength of coefficients at different time points cannot be compared directly. When examining the cross-lagged relations, maternal symptom levels towards the end of pregnancy predicted paternal depressive symptom levels in the early postpartum period (β = 0.11, 95% CI [0.03, 0.18], $p = .006$), while the remaining cross-lagged relations were not significant ($p > .05$).

Moderation hypotheses were tested using multi-group analyses in which couples characterized by secure partner-related attachment (n = 710) and by insecure partner-related attachment (n = 158) were compared. Following Hussong, Hicks, Levy, and Curran (2001), a fully constrained model was compared with a model in which the cross-lagged parameters were relaxed, and a significant moderation effect $\Delta\chi^2$ (12) = 38.07, $p < .01$ was found. Post-

hoc tests revealed that several paths were significantly different between groups, with χ^2-differences ($\Delta\chi^2$) with one degree of freedom ranging from 4.45 to 11.00 ($p < .05$). More specifically, maternal symptoms predicted paternal symptoms in the insecurely attached group: from MDS3 to FDS4 ($\beta = .17$, 95% CI [0.06, 0.28], $p = .002$), from MDS5 to FDS6 ($\beta = .16$, 95% CI [0.04, 0.27], $p = .006$), and from MDS6 to FDS7 ($\beta = .20$, 95% CI [0.02, 0.38], $p = .032$). None of these had significant relations in the securely attached group. Notably, the path from MDS4 to FDS5 that was significant in the single group model was not significantly different among groups. Further, two paths from paternal to maternal depressive symptoms were also moderated ($\Delta\chi^2$ [1] = 4.47 and 12.17) with significant associations in the insecurely attached group only: from FDS2 to MDS3 ($\beta = .15$, 95% CI [0.01, 0.29], $p = .038$) and from FDS3 to MDS4 ($\beta = .17$, 95% CI [0.05, 0.28], $p = .003$).

Discussion

In line with our hypothesis, we found that mothers' depressive symptoms at the end of pregnancy predicted fathers' level of depressive symptoms six weeks after the baby was born. This supports previous studies of perinatal depression concordance between spouses (Cameron et al., 2016) and provides new knowledge on the temporal pattern of the partner dynamics of symptom development. The statistical modeling approach used takes time-invariant confounding into account, thereby rendering interpretations such as assortative mating, common time-invariant contextual influences or shared health habits unlikely, as these mechanisms would have been manifest as time-invariant components in the model. Thus, the finding indicates depressive symptom contagion in the perinatal period, in terms of a transmission of negative mood states from mothers to fathers around the time of birth. This specific timing suggests that the period around birth is a particularly vulnerable time for fathers in terms of being influenced by depressive symptoms in their partners. The finding that women's depressive symptoms are not predicted by men's symptoms is in accordance

with the notion that the focus at this time is primarily on the mother's and the newborn's well-being. The effect size of the association was small; however, relations were tested in a conservative modeling context, as the ALT takes into account both time-invariant confounding influences and effects from the individual's own prior depressive symptoms.

Also in accordance with hypothesis, we found that partner-related attachment moderated the interparental relations of depressive symptoms: Maternal symptoms predicted subsequent paternal symptoms at additional time points during the latter part of pregnancy and throughout the first year after the baby was born in the insecurely attached couples group only. Further, only in the insecure group did fathers' depressive symptoms predict mothers' symptom levels in mid-pregnancy. Among insecurely attached couples, a partners' depressed mood may evoke negative working models of the self and others, thereby increasing the risk of the expression of depressive symptoms. It is notable that the association between maternal and paternal depressive symptoms at the time of birth was not moderated. This suggests that new fathers, regardless of attachment status, are vulnerable to depressive symptom contagion during this particular time, a time characterized by considerable change, emotional interdependence, and a heightened focus on the mother's well-being.

The present study has several limitations. First, perinatal depression is complex, and it is unlikely that interpersonal perspectives alone can sufficiently explain the heterogeneous presentation and symptom course. Second, depressive symptoms were assessed by self-report; no information on clinical diagnosis was available. Third, partner-related attachment was also measured by self-report; thus, associations might be inflated due to shared methods variance. Fourth, data points were unevenly distributed, making direct comparisons of effect sizes among the autoregressive and cross-lagged coefficients difficult. Fifth, selective attrition might be a concern, as dropout was related to depressive symptoms in this study. However, by using FIML to handle missing data we reduced the impact of such selective

attrition. Sixth, the study was conducted in Norway and may not be generalizable to other contexts. Finally, as in any community-based study, there is a possibility of self-selection bias with an overrepresentation of healthy participants.

Clinicians need to be aware of new fathers' vulnerabilities and needs in cases where mothers show depressive symptoms in the period around birth and direct preventive and treatment efforts at both parents. Notably, as there were relatively low levels of depressive symptoms in this community sample, findings underscore that symptoms do not have to reach clinical levels before one's partner's mental health is affected. Working to prevent symptom development in the initially unaffected parent is especially important considering newborns' need for emotional availability in their caretakers. Specific attention should be directed to identifying susceptible parents as indicated by insecure partner-related attachment styles. In future studies, increased efforts to understand temporal processes of perinatal depressive symptom development within the family context are needed.

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Figure 1

Standardized Parameter Estimates from the Single Group ALT.

Note. **p* < .05; ***p* < .01. ALT = autoregressive latent trajectory model; MDS = mothers' depressive symptoms; FDS = fathers' depressive symptoms; M = means; Var = variances. The variance of MDS slope factor has been fixed to zero due to convergence issues. Model fit ($\chi^2(61) = 85.19$, CFI = 0.989, TLI = 0.984, RMSEA = 0.020, 90% CI [0.008, 0.029]).

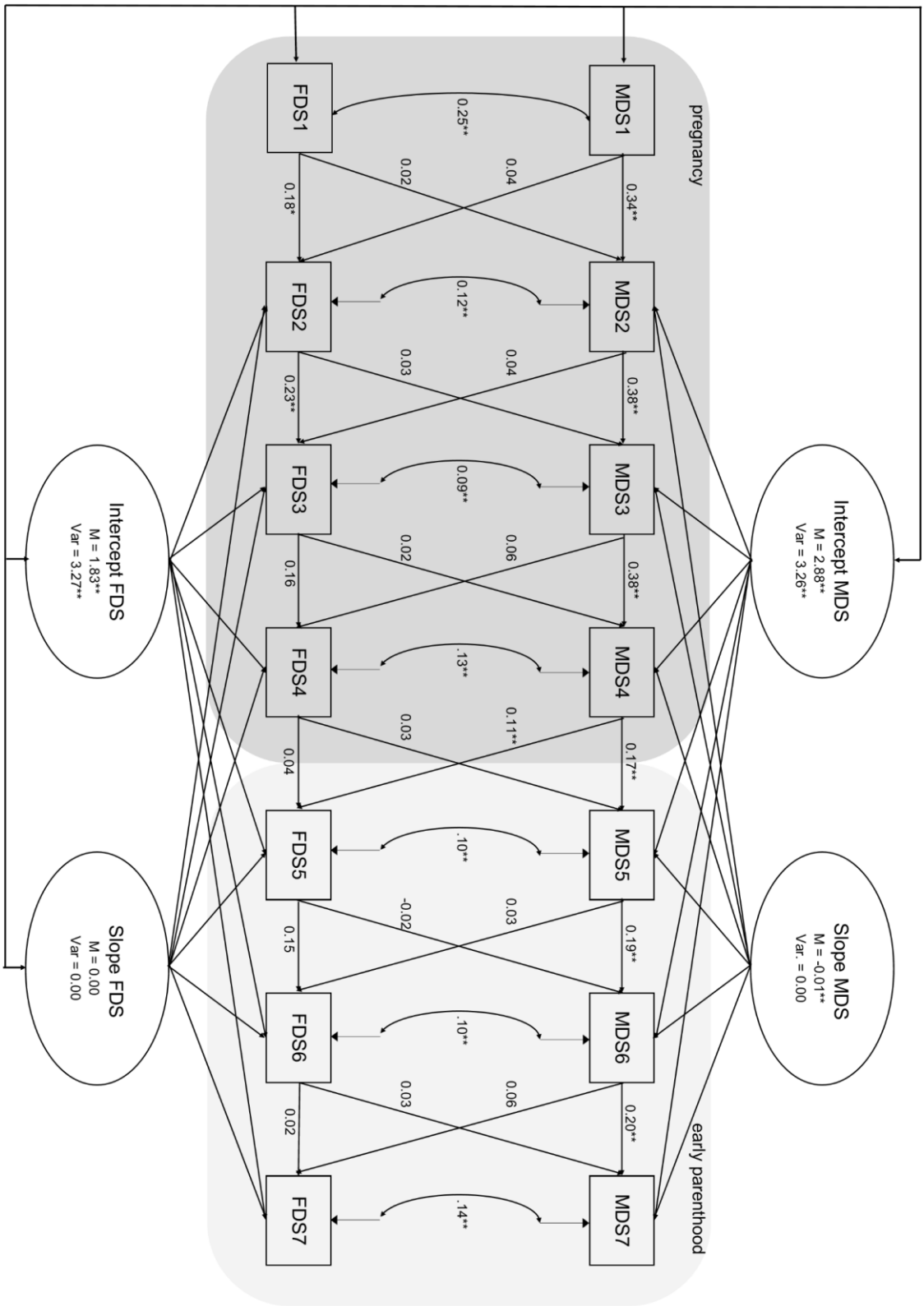
pregnancy
early parenthood
MDS1
MDS2
MDS3
MDS4
MDS5
MDS6
MDS7
FDS1
FDS2
FDS3
FDS4
FDS5
FDS6
FDS7
Intercept MDS
M = 2.88**
Var = 3.26**
Slope MDS
M = -0.01**
Var. = 0.00
Intercept FDS
M = 1.83**
Var = 3.27**
Slope FDS
M = 0.00
Var = 0.00
0.25**
0.12**
0.09**
.13**
.10**
.10**
.14**
0.34**
0.38**
0.38**
0.17**
0.19**
0.20**
0.18*
0.23**
0.16
0.04
0.15
0.02
0.02
0.04
0.03
0.04
0.02
0.06
0.03
0.11**
0.03
-0.02
0.03
0.06

Table 1

Demographic Information for Mothers and Fathers Participating in the Study

	Mothers Mean (SD) / Proportion	Fathers Mean (SD) / Proportion
Age	30.26 (4.78)	32.76 (5.90)
Education (in years)	16.05 (2.13)	15.59 (2.37)
Ethnic minority	6.1%	4.6%
First-time parent	54.9%	56.2%
Work status:		
Full-time job	77.3%	91.0%
Part-time job	7.4%	1.7%
Student	11.6%	6.2%
Disability/Unemployed/At home	3.8%	1.0%
Relationship status:		
Married	36.2%	35.2%
Living together	59.7%	62.4%
Single	2.5%	0.9%
Divorced	0.2%	0.2%
Other	1.4%	1.2%

Note. SD = Standard Deviation.

Table 2

Intercorrelations and Descriptive Statistics of all Study Variables

	M	SD	N	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Mothers' Depressive Symptoms																	
(1) at T1	4.41	3.73	659														
(2) at T2	4.41	3.76	579	.69**													
(3) at T3	4.68	4.07	906	.60**	.72**												
(4) at T4	4.67	4.00	913	.56**	.63**	.73**											
(5) at T5	3.75	3.39	930	.41**	.46**	.48**	.49**										
(6) at T6	3.08	3.41	860	.42**	.47**	.47**	.44**	.56**									
(7) at T7	3.05	3.17	762	.49**	.51**	.55**	.55**	.47**	.56**								
Fathers' Depressive Symptoms																	
(8) at T1	2.44	2.81	487	.24**	.23**	.16**	.21**	.11*	.20**	.14**							
(9) at T2	2.36	2.80	590	.16**	.19**	.19**	.16**	.09*	.10**	.13**	.66**						
(10) at T3	2.71	3.34	487	.12*	.12*	.19**	.16**	.10*	.13*	.11*	.51**	.63**					
(11) at T4	2.39	2.74	486	.15*	.16**	.21**	.19**	.07	.08	.18**	.56**	.65**	.67**				
(12) at T5	2.41	2.71	677	.14**	.17**	.18**	.21**	.21**	.17**	.20**	.52**	.50**	.54**	.60**			
(13) at T6	2.57	2.99	575	.14*	.14**	.22**	.20**	.12**	.21**	.19**	.50**	.52**	.61**	.62**	.57**		
(14) at T7	2.38	2.82	540	.23**	.17**	.21**	.24**	.17**	.24**	.27**	.41**	.51**	.55**	.56**	.50**	.61**	
Partner-related attachment																	
(15) Insecure attachment style[1]	0.19	0.40	878	.33**	.30**	.29**	.33**	.22*	.22**	.23**	.27**	.17**	.22**	.23**	.22**	.22**	.14**

Note. M = means, *SD* = Standard Deviation, *N* = Number of participants.

[1]Insecure attachment style was coded: 0 = securely attached couples and 1 = insecurely attached couples. Positive correlations thus reflect higher depressive symptom level among insecurely attached couples.

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

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Parenting Stress Plays a Mediating Role in the Prediction of Early Child Development from Both Parents' Perinatal Depressive Symptoms

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Abstract

Maternal postnatal depression has been associated with a broad range of developmental risk among children. However, there has been less focus on disentangling the effects of pre- and postnatal depressive symptoms, as well as examining the symptoms of both parents. This study aims to investigate the separate effects of pre- and postnatal depressive symptoms in mothers and fathers, and parents' differential effects on child social-emotional, cognitive, and language development at 18 months of age. Further, we investigate whether effects of depressive symptomatology on child outcomes are particularly strong when both parents evinced high symptom loads and whether parenting stress mediates associations between perinatal depressive symptoms and child developmental outcomes. The study used data from 1036 families participating in a community-based study from mid-pregnancy until 18 months postpartum. Depressive symptoms were assessed at seven time points (four prenatally). Within a structural equation framework, we found that parental perinatal depressive symptoms predicted child social-emotional functioning, specifically externalizing, internalizing, and dysregulation problems, as well as language developmental delay at 18 months. Controlling for postnatal symptoms we found no independent effect of prenatal depressive symptoms on any child outcomes. A differential effect was evident, linking maternal symptoms to social-emotional outcomes, and paternal symptoms to language outcomes. There was no evidence of stronger associations between depressive symptoms and child outcomes when both parents showed high symptom loads. However, parenting stress mediated most relations between parental depressive symptoms and child outcomes. Findings demonstrate the importance of including paternal depressive symptoms in both clinical and research contexts.

Keywords Perinatal depressive symptoms · Social-emotional development · Language development · Parenting stress

The issue of perinatal depression and the ways in which depressive symptoms in this period may affect child development has been of longstanding interest for both clinicians and researchers. Traditionally, there has been an emphasis on the mothers' symptom load in the postnatal period, with findings demonstrating adverse effects of maternal depression in diverse domains of child development from infancy through adolescence. Such adverse effects include impaired infant regulatory functioning (Feldman et al. 2009), early difficulties in regulating negative affective states (Tronick and Reck 2009), disturbances in children's emotional, behavioral, and social functioning (Stein et al. 2014), altered brain structure (Lebel et al. 2016), adolescent mental health diagnoses (Pearson et al. 2013), and delayed cognitive and language development (Barker et al. 2011). Less attention has been given to investigating the relations between prenatal depression and child development, as well as child developmental outcomes following fathers' depression (Ramchandani and Psychogiou 2009). Of note is the lack of studies combining these aspects to examine differential and joint effects of mothers' and fathers' depression in the entire perinatal period. Further, there is an ongoing effort to elucidate the mechanisms of how parental depression translates into aberrant child development, as such mechanisms are only partially understood (Goodman and Gotlib 1999; Stein et al. 2014).

This study combines repeated pre- and postnatal measurements of depressive symptoms in both mothers and fathers, examining the effects of their pre- and postnatal symptom load on a broad spectrum of child developmental outcomes at

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18 months of age, including cognitive, language, and social-emotional domains. Further, we explore whether these effects are mediated by each parent's experience of stress and coping in the parenting role, as parenting stress and coping are proposed to be important mechanisms in the relation between perinatal depressive symptoms and child developmental outcomes.

Pre- and Postnatal Mechanisms of Transmission of Risk

There is accumulating evidence that perinatal depressive symptoms have a wide impact on infant and child developmental outcomes (Stein et al. 2014). Rather than pointing at specific and delimited effects of parental depressive symptoms, this is suggestive of a more extensive influence of parental depression, operating through a complex interplay of parental, child, and contextual factors over time. In a seminal review Goodman and Gotlib (1999) described a model of possible pathways from maternal postpartum depression to adverse child outcomes, highlighting genetic transmission, the development of dysfunctional neuroregulatory mechanisms, exposure to the mother's maladaptive affect, behavior and cognitions, as well as contextual stressors associated with maternal depression. Maternal prenatal depression has been shown to influence child outcomes irrespective of postnatal depression (Pearson et al. 2013), although findings have been mixed (Bekkhus et al. 2011; Van Batenburg-Eddes et al. 2013). The mechanisms of risk transmission in pre- and postnatal depression may differ. Prenatal mechanisms include fetal exposure to maternal depressive symptoms while in utero, generally referred to as fetal programming and may partially account for variance in later child outcomes. Prime hypotheses of such intrauterine mechanisms state that maternal depressive symptoms influence the intrauterine environment through (a) heightened maternal hypothalamic–pituitary–adrenal (HPA) activity leading to lasting alterations in the fetal HPA axis, (b) reduced blood flow through the placenta potentially leading to impaired fetal development, or (c) epigenetic dysregulation within HPA pathways (Waters et al. 2014). In contrast, an exclusively postnatal mechanism would involve the child being directly exposed to parental emotions, cognitions, and behaviors affected by a depressed mood.

The effects of prenatal depression on child outcomes might also be explained by the continuation of prenatal symptoms into the postnatal period, during which a direct exposure would be likely to occur. Such a mechanism may be likely, as studies investigating the stability of depression from pregnancy through the postpartum period have found that for a majority of affected women, symptoms are elevated throughout the whole perinatal period, or even originated before pregnancy (Wisner et al. 2013). Further, prenatal symptom onset is more frequent for depressed women affected by the most severe symptom levels, compared to subgroups of women with less severe symptom loads (PACT Consortium 2015). The high stability of depressive symptoms during the perinatal period renders it useful with study designs measuring both pre- and postnatal symptoms, to disentangle the associations.

Alternative explanations of either an intrauterine effect or a direct exposure-mechanism would be genetically transmitted risk (Kendler et al. 2006), gene-environment interactions including epigenetic mechanisms (Rice et al. 2013), or contextual correlates of depression posing risks to the developing fetus/child (i.e., poor nutrition and reduced compliance with perinatal health guidelines). These alternative conjectures do not assume a differential impact of pre- and postnatal symptoms on child developmental outcomes.

Maternal and Paternal Depressive Symptoms: Differential and Moderating Effects

Most findings associating depressive symptoms in the perinatal period with adverse developmental outcomes in children have been in relation to maternal depression, while the effects of paternal depression are understudied. However, there is an increasing interest in paternal psychopathology in the perinatal period (Skjøthaug et al. 2015), and paternal depressive symptoms have been associated with non-optimal social-emotional functioning and language development in offspring (Paulson et al. 2009; Ramchandani and Psychogiou 2009). Depressive problems in the perinatal period may not affect parenting behaviors in the same manner for men as they do for women (Connell and Goodman 2002), which brings us to the question of whether paternal depressive symptoms may differ from maternal depressive symptoms in their effect on children. In a meta-analysis on the differential impact of maternal and paternal mental health issues on internalizing and externalizing problems in children, both parents' psychological problems predicted symptoms in both domains across the entire age range from toddlerhood to adolescence. For children in the youngest age group (below 6 years of age), maternal mental health had a larger impact on both internalizing and externalizing problems, relative to paternal psychological problems (Connell and Goodman 2002), although this review did not apply a fine-grained parsing of age groups, and there was little data on any differential effects on toddlers. Research on language development, however, has especially pointed to fathers' depression and the comparatively larger influence of their depressive symptoms on children's language development (Paulson et al. 2009), although maternal depression has also shown adverse effects in this domain (Quevedo et al. 2012).

The degree to which parental symptoms affect child development may differ according to other strains that the child and

family are exposed to. Recent research efforts have increasingly focused on identifying factors that may accentuate or ameliorate the risk of aberrant child development following parental depression (Stein et al. 2014). One important factor in this respect may be that both parents have high depressive symptom loads. More specifically, the association between depressive symptoms in one parent and aberrant child development may be particularly strong when the other parent as well shows high symptoms loads, as this could cause additional burdens and remove the potential buffering role that the non-depressive parent could have played. There are very few studies examining such moderation effects of both parents' mental health problems, and findings have been mixed (Paulson et al. 2009).

Parenting Stress as a Mediator of Heightened Risk Following Parental Depression

Parenting stress refers to the difficulties of adjusting to the parenting role, pertaining to both perceived shortcomings as a parent and the parent's perceptions of the child, and has an inverse relation to the broader concept of parenting quality (Cappa et al. 2011). Research has supported the mediating role of parenting quality in the relation between maternal depressive symptoms and adverse child outcomes, and has indicated that the direct exposure to depressed mothers' affect, behavior, and cognition plays a part in risk transmission (Lovejoy et al. 2000; Wang and Dix 2013). Less focus has been given to the specific role of parenting stress in these associations.

Depressive symptoms in the postnatal period have been associated with increased levels of parenting stress and dysfunction in parent-child interactions (Leigh and Milgrom 2008). Parents with more stress display more authoritarian and less responsive parenting styles (Belsky et al. 1996), which in turn has been associated with behavioral, social-emotional, and cognitive problems throughout the child's development (Deater-Deckard et al. 1994). Parenting stress has also been shown to be directly related to negative child outcomes such as separation anxiety (Deater-Deckard et al. 1994), and child oppositional behavior (Podolski and Nigg 2001). Research suggests that parenting stress and child coping strategies are closely related through a bidirectional influence over time (Cappa et al. 2011).

Study Aims

Overall, a large body of research suggests that there is an association between parental perinatal depressive symptoms and child adverse social-emotional, language, and cognitive outcomes. However, while informative, these findings are limited in several ways. First, while the effects of maternal postnatal symptoms have received the lion's share of attention, the impact of paternal depressive symptoms is understudied. Moreover, an investigation of differential associations with child outcomes is warranted. Second, there is a lack of studies with a thorough examination of prenatal symptoms together with postnatal symptoms, with a design capable of disentangling the associations of pre- versus postnatal symptoms with child outcomes. Third, the mechanisms of risk transmission are only partially understood, and research efforts are needed to further elucidate mediating and moderating factors in these relations. Finally, few studies have combined these features by obtaining repeated measurements of both maternal and paternal symptoms throughout the perinatal period.

The purpose of this study was to examine the associations between parental depressive symptoms in the perinatal period and child social-emotional, cognitive, and language skills at 18 months of age. We used a population-based, multisite sample and measured maternal and paternal depressive symptoms seven times, beginning in mid-pregnancy throughout the first postpartum year. By using a dimensional approach, we could capture subclinical depressed mood as well as severe depressive problems and were thus able to examine the associations with child outcomes on a detailed range of depressive problems. Specifically, we examined (a) whether exposure to parental pre- and postnatal depressive symptoms is associated with children's developmental outcomes, and if there is an independent effect of prenatal symptoms after postnatal symptoms are accounted for; (b) whether any prospective associations between parental depressive symptoms and children's developmental outcomes are different for mothers and fathers; (c) whether associations between parental depressive symptoms and child outcomes are particularly strong when both parents suffered from high symptom loads; and (d) whether parenting stress measured when children are 12 months old mediates the relations between parental depressive symptoms (measured throughout pregnancy and the first postpartum year) and the children's developmental outcomes at 18 months of age.

Method

Procedure and Participants

This study is based on data from 1036 families participating in the prospective, multisite *Little in Norway* study (Fredriksen et al. 2017). From September 2011 until October 2012, all pregnant women receiving prenatal care at nine selected well-baby clinics across Norway were invited to participate in the study. There were no exclusion criteria. The response rate was estimated to be 50.7% (Fredriksen et al. 2017). Initially, 1041 women gave their informed consent, but five

later withdrew, leaving 1036 participants. The partners of the pregnant women were also invited to participate, and 878 fathers gave their informed consent. Ten women gave birth to twins. From each pair of twins, only one child, the twin for whom the questionnaire was answered first, was included in this study.

Women were recruited at their first visit to their local well-baby clinic, resulting in enrollment in different phases of pregnancy, as participants' gestational weeks diverged considerably at the time of their first visit (range: weeks 8–34). This study used data from eight data collection waves, however only a proportion of the sample was enrolled in time to participate in the early prenatal data collection waves (T1- T3). Prenatal data collection waves were at average gestational week 21 (T1, $n = 659$, range: 8–24), week 28 (T2, $n = 579$, range: 25–29), week 32 (T3, $n = 906$, range: 30–33) and week 36 (T4, $n = 913$, range: 34–38). The drop in participation at T2 was largely due to shortage of staff members to collect data. Postnatal data collection waves were at 6 weeks postpartum (T5, $n = 930$), 6 months postpartum (T6, $n = 860$), 12 months postpartum (T7, $n = 762$), and 18 months postpartum (T8, $n = 777$), all with a range of +/− 2 weeks. The lowered participation at T7 and T8 likely reflects the fact that parents are returning to work after 1 year of parental leave, with less time to participate in research. There are three categories of missingness, late recruitment as described above, study withdrawal, and intermittent missingness. Regarding study withdrawal, 11, 6, 6, 20, 25, 30, and 13 participants completely withdrew from the study prior to T2 to T8, respectively. The remaining missingness comprises participants that missed one or more data collection waves, but then contributed with data at later time points. Bivariate logistic regression analyses were conducted to investigate selective attrition. Participation at the last data collection point (T8) was predicted by lower levels of mean prenatal (OR = 0.91, 95% CI [0.88, 0.95], $p < 0.001$) and postnatal (OR = 0.95, 95% CI [0.90, 1.00], $p = 0.041$) depressive symptoms for mothers, as well as lower levels of mean prenatal (OR = 0.92, 95% CI [0.87, 0.97], $p = 0.004$) and postnatal (OR = 0.93, 95% CI [0.86, 0.99], $p = 0.030$) depressive symptoms for fathers. Further, high level of the mothers' education slightly increased the probability of participation at T8 (OR = 1.07, 95% CI [1.01, 1.15], $p = 0.030$). Maternal age (OR = 1.00, 95% CI [0.97, 1.03], $p = 0.886$), paternal age (OR = 1.00, 95% CI [0.98, 1.03], $p = 0.763$), maternal parity (OR = 0.87, 95% CI [0.73, 1.04], $p = 0.123$), number of fathers' previous children (OR = 0.98, 95% CI [0.80, 1.21], $p = 0.879$) and fathers' education (OR = 1.04, 95% CI [0.97, 1.11], $p = 0.255$) were not related to attrition at T8.

Ethical Approval

All procedures performed in the study were in accordance with the ethical standards of the institutional and/or national research committee (approved by the Regional Committees for Medical and Health Research Ethics in Norway) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Measures

Depressive Symptoms Depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS) for men and women at T1 to T7. The EPDS is a 10-item self-report questionnaire asking respondents to consider various depressive symptoms during the last 7 days on a four-point scale (range: 0–30). Examples include "I have been so unhappy that I have had difficulty sleeping" and "I have felt sad or miserable". The EPDS was originally developed to screen for depressive symptoms in women during the postpartum period (Cox et al. 1987). It was later validated for prenatal use by Murray and Cox (1990), and in a Norwegian community sample (Eberhard-Gran et al. 2001). It has also been validated on male populations in the postnatal period (Matthey et al. 2001). Although developed with cut-off scores indicating probable depression, the EPDS composite score has also been used as a continuous variable for research purposes (Matijasevich et al. 2015), with the benefit of yielding a more detailed range of depressive symptomatology at both clinical and subclinical levels. In this study, the EPDS composite score was used as a continuous variable. Cronbach's alphas were high at each assessment (ranging from 0.80 to 0.85 for women and from 0.76 to 0.84 for men), indicating good internal consistency.

Parenting Stress Parenting stress was measured with the Parenting Stress Index (PSI; Abidin 1995) when children were 12 months old. The PSI examines the level of stress within the parent-child system, and consists of factors reflecting parental coping and their perceptions of the child. It is measured on a five-point scale, consists of 101 items, and has two domains: the parent domain assesses the personal characteristics and social support of the parent, whereas the child domain reflects the degree to which various child characteristics causes stress to the parents. Total scores are the sum of both domains, with a range from 101 to 505. In this study, we used total scores, as we were primarily interested in the level of parental stress, rather than the source of that stress. The PSI has shown good internal consistency, and has been validated in both normal and diverse clinical populations (Abidin 1995). In this study, Cronbach's alphas for total scores were 0.94 for both mothers and fathers.

Cognitive and Language Development Cognitive and language development was measured by the Bayley Scales of Infant and Toddler Development, Third Edition (Bayley-III; Bayley 2006) when the children were 18 months. The Bayley-III is an individually administered instrument designed to

measure the developmental functioning of infants and toddlers, especially suited to detect developmental delays. In this study, the cognitive and language scales were used (omitting the motor scales). The cognitive scale evaluates abilities, such as sensorimotor development, exploration and manipulation, object relatedness, concept formation, memory, and simple problem solving. There are two language scales: receptive communication, which measures verbal comprehension, and expressive communication such as babbling, utterances, and gestures. Health nurses, who were trained in the method, administered the test at the well-baby clinics under supervision of a clinical psychologist specialized in infant and child development. The administration and scoring rules were digitally implemented, so the sequencing and number of tasks were automatically computed based on the performance of the toddler. The parents were present in the room but instructed not to interfere with the testing.

Social and Emotional Development The Infant-Toddler Social and Emotional Assessment (ITSEA; Carter and Briggs-Gowan 2006) was used to assess social and emotional functioning when the children were 18 months old. The parent or parents accompanying the child to the well-baby clinic answered the questionnaire, resulting in 387 mothers, 32 fathers and 162 jointly answering the questionnaire. The ITSEA is a 168-item instrument measuring areas of social-emotional problems and competencies in 12-to 36-month-olds, with each item rated on a 3-point scale. It yields four domains: an internalizing domain, consisting of depression/withdrawal, general anxiety, separation distress, and inhibition to novelty subscales; an externalizing domain, consisting of activity/impulsivity, aggression/defiance, and peer aggression subscales; a dysregulation domain, consisting of negative emotionality, sleep, eating, and sensory sensitivity; and a competence domain, consisting of compliance, attention, mastery motivation, imitation/play, empathy, and prosocial peer relations. The ITSEA is widely used, and has shown acceptable reliability and validity (Carter et al. 2003). In this study, Cronbach's alphas were 0.65, 0.76, 0.78, and 0.82 for the internalizing, externalizing, dysregulation, and competence domains, respectively.

Statistical Analyses

As this study has a longitudinal design with repeated measurements of depressive symptoms it is important to ascertain whether items of the EPDS were interpreted differently across time points. For this purpose, tests of measurement invariance by means of confirmatory factor analysis were performed. Model fit was evaluated by χ^2 statistics, Confirmatory Fit Index (CFI), Tucker–Lewis Index (TLI), and the root mean square error of approximation (RMSEA), with CFI and TLI values greater than 0.95 and RMSEA values lower than 0.06 indicating good fit (Hu and Bentler 1999). More specifically, based on the ten EPDS items, three parcels were constructed that functioned as indicators for the latent depressive symptom factors, with identical parcel structure across time points. Reducing indicators through parceling has been shown to provide superior tests of structural model parameters as the constructs are defined more precisely (Little et al. 2013). However, the practice of parceling has also been criticized for not taking into account the variability in estimates created by the specific item-to-parcel allocation (Sterba and MacCallum 2010). To reduce this potential bias, we followed suggestions in the methodological literature (Sterba and MacCallum 2010) by re-estimating all models repeatedly with varying item-to-parcel allocations. More specifically, the average of fit indices across 20 identical models with different randomly selected allocations are reported.

Following Widaman et al. (2010), we first tested for configural invariance by constructing latent factors based on the three parcels for each wave, with correlated factors and correlated error variance of identical parcels at different time points. For mothers the basic configural model yielded excellent average fit across the 20 estimations with different item-to-parcel allocations (average fit: $\chi^2[105] = 142.51$; CFI = 0.996; TLI = 0.991; RMSEA = 0.017). Next, weak measurement invariance was tested by constraining factor loadings to be equal across time points. The average model fit showed minor reductions, but was still very good (average fit: $\chi^2[123] = 214.23$; CFI = 0.989; TLI = 0.982; RMSEA = 0.026). Strong invariance was then tested by additionally constraining intercepts of the parcels to be equal across time. Results showed an average decrease in fit for two of the three indices; however, fit indices still indicates acceptable fit (average fit: $\chi^2[141] = 492.72$; CFI = 0.996; TLI = 0.938; RMSEA = 0.049). For fathers the configural model also yielded very good fit across the 20 estimated models (average fit: $\chi^2[105] = 135.72$; CFI = 0.992; TLI = 0.984; RMSEA = 0.017). The weak invariant model, showed average fit indices that were only minimally lower (average fit: $\chi^2[123] = 167.20$; CFI = 0.988; TLI = 0.980; RMSEA = 0.019). When testing for strong invariance by imposing constraints on parcel intercepts, the average fit indices again only showed small decreases, and model fit remained very good (average fit: $\chi^2[141] = 214.64$; CFI = 0.981; TLI = 0.972; RMSEA = 0.024). Taken together, acceptable model fits for the strong measurement invariant models indicate that EPDS items were interpreted rather similarly across time points. However, the reductions in model fit with increasingly restrictive models show a certain degree of difference in interpretations across all seven time points, particularly for mothers.

In a next step, latent factors were modelled by means of confirmatory factor analysis to reflect mothers' and fathers' prenatal and postnatal depressive symptoms. Prenatal latent depression factors were constructed for mothers and fathers, separately, loading on the four EPDS composite scores during

pregnancy. Likewise, postnatal depression factors were modeled for both mothers and fathers, based on the three EPDS composite scores measured at 6 weeks, 6 months and 12 months after birth. All factor loadings were constrained to be equal, thereby forcing depressive symptoms at all the time points to be equally important indicators for the latent factor of depressive symptoms. Imposing equality across time thus prevents any bias in how the timing of symptoms is related to the factor, and yielded adequate fit for both mothers ($\chi^2[18] = 63.95$; CFI = 0.961; TLI = 0.955; RMSEA = 0.050, 90% CI: 0.037, 0.063) and fathers ($\chi^2[18] = 26.60$; CFI = 0.985; TLI = 0.982; RMSEA = 0.023, 90% CI: 0.000, 0.040). These four factors were then used as predictors in structural equation models, with child cognitive, language, social, and emotional developmental measures as outcomes. All analyses were controlled for child's sex and parents' age, parity, and educational level.

To examine whether there was an independent effect of prenatal depressive symptoms after the postnatal symptom load had been accounted for, postnatal symptom paths were adjusted for prenatal symptoms. As pre- and postnatal factors were highly correlated, Cholesky factoring was used to handle potential problems with multicollinearity (de Jong 1999), an approach used in similar contexts previously (e.g., Lervåg et al. 2009). As depicted in Fig. 1, Cholesky factoring decomposes the predictor variables in the structural model into factors that entail a sequential decomposition of variance. In our case, such decomposition provides estimates of the unique variance in child outcomes that is explained by prenatal depressive symptoms when variance due to postnatal depressive symptoms is partialled out. Equivalent to hierarchical regression or fixed-order regressions in standard regression analyses, Cholesky factoring thus provides information about the extra amount of variance accounted for in child outcomes by prenatal depressive symptoms.

Differential effects of maternal and paternal depressive symptoms were investigated by adjusting for the other parent's symptom load when estimating the path coefficients of depressive symptoms on child cognitive, language, and social-emotional outcomes. We further tested whether maternal and paternal associations were significantly different for each of these outcomes, in order to test the specificity of the relations. For this purpose, models where regression coefficients were constraint to be equal across parental gender were compared with models without such constraints by means of Satorra-Bentler scaled χ^2 difference tests.

Further, to explore whether each parent's postnatal depressive symptoms would moderate the relation between the other parent's depressive symptoms and child outcome, latent factor interaction terms were formed and entered in the models using the XWITH command in Mplus, in accordance with the latent moderated structural equations (LMS) approach (Klein and Moosbrugger 2000). Finally, each parent's experience of parenting stress was examined as a mediator of the longitudinal relations between postnatal depressive symptoms and child developmental outcomes. This was done by estimating indirect paths from both parents' latent postnatal symptom factors, through mothers' and fathers' parenting stress to all significant child outcomes in the adjusted analyses. Standard errors of the indirect paths were estimated based upon bootstrapped bias-corrected 95% confidence intervals with 5000 draws (Hayes 2009).

Missing data were handled by the full information maximum likelihood procedure (FIML) accounting for missing at

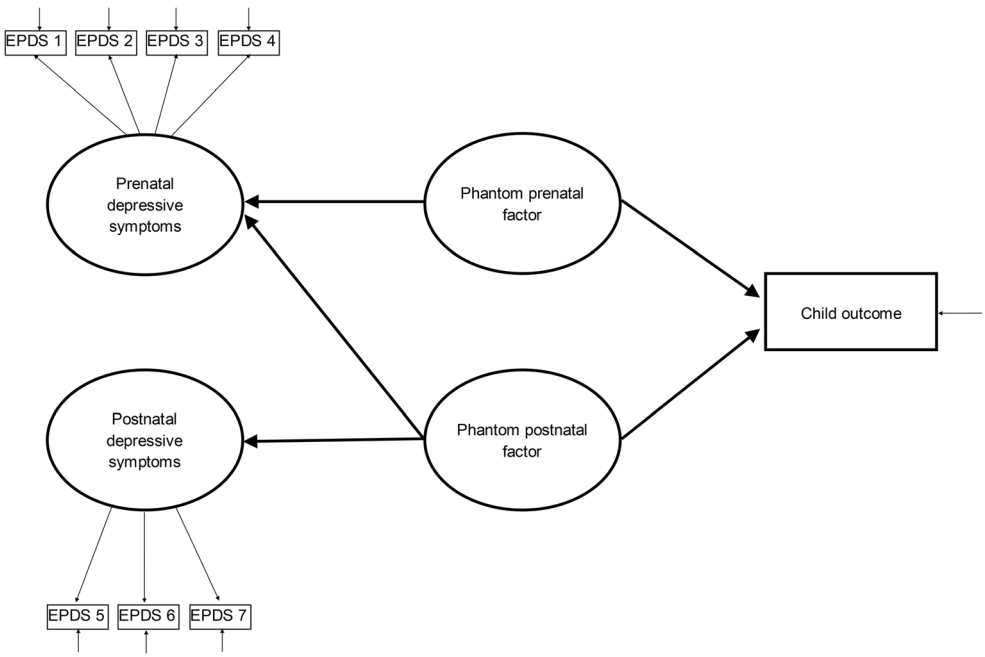

Fig. 1 Path diagram of Cholesky decomposition of intercorrelations among pre- and postnatal symptoms of depression. Phantom factors are introduced to provide estimates of the unique variance in child outcomes that is explained by prenatal depressive symptoms when variance due to postnatal depressive symptoms is partialled out

random (MAR) assumptions, to account for missingness in both indicator, mediating, and outcome variables. All data analyses were performed in Mplus version 7.3 using robust maximum likelihood estimation.

Results

Descriptive Statistics and Bivariate Relations

The mean maternal and paternal age at inclusion was 30.3 years (range: 17–43, $SD = 4.8$) and 32.8 years (range: 16–57, $SD = 5.9$), respectively. Most parents (95.9%) were either married or living with a partner. Of the mothers, 54.8% were nulliparous; 56.2% of the male participants were first-time fathers. Most mothers (77.1%) and fathers (67.1%) had an educational level beyond high school. A majority of participants were of Norwegian descent, although a small proportion (6.1% of mothers, 4.6% of fathers) reported other ethnicities. More boys (52.4%) than girls were born in this sample. The mean gestational period was 39.5 weeks ($SD = 1.9$), and mean birth weight was 3535 g ($SD = 542$).

Table 1 shows the means and standard deviations of all study variables. Depression scores throughout the pregnancy and postpartum period were in the lower end of the scale for both mothers and fathers, as expected in a community sample. Among mothers, depressive symptoms before birth were significantly higher than after birth ($t = 13.06$, $p < 0.001$) whereas fathers had generally lower levels of depressive symptoms than mothers with no significant differences before and after birth ($t = -0.31$, $p = 0.754$). To investigate differences between individual time points, repeated measures ANOVAs with a Greenhouse-Geisser correction were performed. For both mothers ($F(4.93, 975.93) = 15.42$, $p < 0.001$) and fathers ($F(4.81, 586.24) = 2.92$, $p = 0.014$) mean EPDS scores differed significantly between time points. For mothers, post hoc tests using Bonferroni correction revealed that EPDS scores 12 and 6 months postpartum were significantly lower than at all preceding time points, whereas symptom levels 6 weeks postpartum were significantly lower than at the last two measurement points during pregnancy. Post hoc tests for fathers revealed significantly lower EPDS scores 12 month after birth compared to the first time point during pregnancy, even though differences were small. The mean of the composite score on the PSI was at the lower end of the normal range. The Bayley language scales means were close to the norm of 100, whereas the cognitive scale was somewhat elevated. All problem domains of the ITSEA were at the lower end of the scales.

Latent pre- and postnatal depression scores correlated highly for both mothers ($r = 0.82$) and fathers ($r = 0.91$), and there were small to moderate correlations among mothers' and fathers' scores (rs = 0.20 to 0.31, see Table 1). The PSI had moderate to high correlations with depressive pre- and postnatal symptoms for both mothers and fathers. Regarding the Bayley-III, there were no significant relations with maternal symptoms; however, paternal depressive symptoms were related to the language scales in the predicted direction, but not to the cognitive scale. Maternal depression scores correlated consistently with high scores in the externalizing and dysregulation domains of the ITSEA, and evinced weak or non-significant associations with the internalizing and competence domains. There were fewer significant correlations among paternal depression scores and the ITSEA domains, with significant but weak relations in the predicted direction, restricted to the dysregulation and internalizing domains. Inspecting bivariate associations between individual time points of depressive symptoms with outcome measures, the strengths of associations were relatively evenly distributed across time points without any clear patterns indicative of depressive symptoms closer in time being more strongly related with the ultimate outcome.

Longitudinal Relations between Parental Depressive Symptoms and Child Outcomes

Latent factors for maternal and paternal pre- and postnatal depressive symptoms were modeled, based on the mean EPDS scores at each time point. Latent EPDS factors were allowed to correlate across time and parental gender. The model including latent depression factors for both parents showed an adequate fit, $\chi^2[81] = 159.95$, CFI = 0.966, TLI = 0.962, RMSEA = 0.031, 90% CI [0.024, 0.038].

Next, children's social-emotional, cognitive, and language outcomes were regressed on each of these four latent factors to examine the parental effects of pre- and postnatal depressive symptoms on child outcomes. All analyses were adjusted for child's sex and parents' education, age, and parity. As depicted in Table 2, results showed that maternal prenatal depressive symptoms had a significant prospective relation with externalizing, internalizing, and dysregulation problems when the children were 18 months old. For fathers, results showed that elevated prenatal depressive symptoms predicted lower scores on both Bayley-III language scales, as well as higher scores on internalizing and dysregulation problems. When associations between postnatal depressive symptoms and child outcomes were examined, the results were virtually identical, with the same relations being significant and similarly-sized coefficients for both mothers and fathers, compared to prenatal results (see Table 3). The Bayley cognitive scale and the ITSEA competence domain were not significantly related to parental depressive symptoms in either period.

In order to assess whether prenatal symptom load explained unique variance in child outcomes after accounting for postnatal depressive symptoms, first postnatal and then prenatal depressive symptom factors were entered as

Table 1 Intercorrelations and descriptive statistics of all study variables

	M	*SD*	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)
Parental depressive symptoms																					
(1) Prenatal depression Mothers	4.54	3.19	–																		
(2) Prenatal depression Fathers	2.47	2.28	0.29**	–																	
(3) Postnatal depression Mothers	3.30	2.41	0.82**	0.20**	–																
(4) Postnatal depression Fathers	2.45	2.12	0.31**	0.91**	0.37**	–															
Mediating variables																					
(5) Parenting stress Mothers	195.07	31.70	0.51**	0.16*	0.67**	0.25**	–														
(6) Parenting stress Fathers	201.08	32.31	0.22**	0.42**	0.24**	0.54**	0.29**	–													
Child outcome variables																					
(7) Cognitive functioning	112.60	16.12	0.05	−0.05	−0.04	−0.03	−0.08*	−0.08	–												
(8) Expressive language	104.79	13.11	0.00	−0.14**	−0.03	−0.11**	−0.08*	−0.13**	0.45**	–											
(9) Receptive language	103.17	16.32	−0.02	−0.17**	−0.03	−0.14**	−0.10**	−0.18**	0.51**	0.67**	–										
(10) Externalizing problems	0.47	0.20	0.15**	0.05	0.12**	0.03	0.29**	0.13**	−0.09*	−0.06	−0.10*	–									
(11) Internalizing problems	0.44	0.19	0.10*	0.09	0.08	0.13**	0.23**	0.13**	−0.05	−0.07	−0.02	0.21**	–								
(12) Dysregulation problems	0.39	0.17	0.22**	0.07	0.28**	0.14**	0.38**	0.20**	−0.06	−0.06	−0.06	0.35**	0.34**	–							
(13) Competence	1.30	0.22	−0.03	−0.07	−0.10*	−0.02	−0.23**	−0.18**	0.24**	0.31**	0.32**	−0.11**	−0.03	−0.12**	–						
Control variables																					
(14) Age Mothers	30.3	4.78	−0.09**	0.00	0.00	0.06	−0.01	0.00	0.05	−0.06	0.05	−0.12**	−0.05	0.00	−0.12	–					
(15) Age Fathers	32.8	5.87	−0.03	−0.02	0.02	0.04	0.00	−0.03	0.00	−0.02	0.02	−0.16**	−0.08	−0.04	0.03	0.70**	–				
(16) Parity Mothers	0.60	0.78	−0.01	0.08	−0.03	0.09	−0.06	0.03	0.09	−0.05	−0.03	−0.07	−0.06	−0.06	0.07	0.35**	0.28**	–			
(17) Parity Fathers	0.58	0.77	−0.05	0.04	−0.04	0.06	−0.04	−0.06	0.04	−0.04	−0.05	−0.07	−0.08*	−0.09*	0.08*	0.32**	0.42**	0.76**	–		
(18) Education Mothers	16.05	2.13	−0.14**	−0.08*	−0.05	−0.06	−0.03	−0.07	0.03	0.01	0.05	−0.15**	−0.08	0.00	−0.01	0.37**	0.26**	−0.04	0.00	–	
(19) Education Fathers	15.61	2.36	−0.06	−0.08*	−0.02	−0.06	−0.01	−0.04	0.09*	0.01	0.10*	−0.10*	−0.04	0.03	−0.02	0.30**	0.26**	−0.01	−0.02	0.44**	–
(20) Child sex	0.48	0.50	−0.01	−0.01	−0.05	0.03	−0.01	−0.02	0.09**	0.21**	0.22**	−0.06	0.05	−0.05	0.12**	0.00	0.02	0.01	0.01	0.01	0.03

Means and standard deviations for pre- and postnatal depressive symptoms are based on the means of all EPDS composite scores in the pre- and postnatal period. However, intercorrelations and all analyses in this study are based on latent depressive symptom scores

M means, *SD* Standard Deviation

* $p < 0.05$, ** $p < 0.01$

predictors using Cholesky factoring. This was done for all outcome variables for mothers and fathers separately. Results showed that neither the maternal nor paternal prenatal depressive symptom factors explained unique variance in any of the child developmental outcomes after postnatal depressive symptoms had been accounted for (see Table 2). The emerging positive association between maternal prenatal depressive symptoms and cognitive functioning in the child indicates a suppressor effect of small size, as high prenatal depression loads were associated with higher cognitive functioning in the child when accounting for postnatal depression symptoms.

Differential and Moderating Effects of Maternal and Paternal Depressive Symptoms

To examine the differential effect of maternal and paternal depressive symptoms on child outcomes, we fitted a model in which mothers' and fathers' postnatal depressive symptoms were adjusted for the other parent's depressive symptoms, in order to predict the children's social-emotional, cognitive, and language developmental outcomes (see Fig. 2 and Table 3). This was done to examine the unique effect of either parent's depressive symptoms on child outcomes. Increases in maternal depressive symptoms predicted elevated child externalizing and dysregulation problems, as measured by the ITSEA, at 18 months. The effect on internalizing problems found in the unadjusted analyses was attenuated and became non-significant. For paternal symptoms, the adverse effect on the children's receptive and expressive communication, as measured by the Bayley-III, remained. However, the effects on both internalizing and dysregulation problems found in the univariate analyses were reduced and found to be non-significant. We further tested whether maternal and paternal relations were significantly different for each of these outcomes, in order to test the specificity of the relations. Results showed significant differences between mothers and fathers in the associations of depressive symptoms with receptive language ($\Delta\chi^2$ [1] = 13.38, $p < 0.05$), expressive language ($\Delta\chi^2$ [1] = 7.99, $p < 0.05$), and dysregulation problems ($\Delta\chi^2$ [1] = 4.15, $p < 0.05$), whereas associations with externalizing problems did not differ between parental gender ($\Delta\chi^2$ [1] = 1.13, $p > 0.05$).

In the next step, we explored whether the strength of the relation between one parent's depressive symptoms and the child's outcomes would change as a function of the other parent's depressive symptoms (i.e., whether disproportionately larger effect of parental depressive symptoms would be evident when both parents were affected by depressive symptoms). Results from interaction analyses showed that there were no interaction effects on any of the child developmental outcome measures (all $ps > 0.05$).

Table 2 Effects of maternal and paternal prenatal depressive symptoms on children's developmental outcomes at 18 months

	Regression coefficients adjusted for covariates only				Regressions when partialling out variance explained by postnatal depressive symptoms			
	Mothers		Fathers		Mothers		Fathers	
Children's developmental outcomes	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]
Cognitive functioning	0.15 [−0.24, 0.54]	0.03 [−0.05, 0.11]	−0.36 [−0.97, 0.26]	−0.05 [−0.14, 0.04]	2.37* [1.53, 2.11]	0.15 [0.03, 0.26]	−0.63 [−3.11, 1.85]	−0.04 [−0.19, 0.12]
Receptive language	−0.12 [−0.51, 0.28]	−0.02 [−0.10, 0.06]	−1.24** [−1.85, −0.64]	−0.17 [−0.26, −0.09]	0.43 [−1.44, 2.30]	0.03 [−0.09, 0.14]	−1.45 [−3.90, 1.01]	−0.09 [−0.24, 0.06]
Expressive language	−0.06 [−0.38, 0.25]	−0.02 [−0.09, 0.06]	−0.78** [−1.28, −0.28]	−0.14 [−0.22, −0.05]	0.47 [−1.02, 1.95]	0.04 [−0.08, 0.15]	−0.76 [−2.80, 1.29]	−0.06 [−0.21, 0.10]
Externalizing problems	0.01** [0.00, 0.01]	0.14 [0.04, 0.23]	0.01 [−0.00, 0.01]	0.06 [−0.04, 0.16]	0.01 [−0.01, 0.04]	0.07 [−0.05, 0.20]	0.00 [−0.03, 0.04]	0.02 [−0.17, 0.21]
Internalizing problems	0.01* [0.00, 0.01]	0.10 [0.01, 0.19]	0.01* [0.00, 0.02]	0.12 [0.02, 0.21]	0.01 [−0.01, 0.03]	0.05 [−0.07, 0.17]	−0.01 [−0.05, 0.02]	−0.08 [−0.29, 0.14]
Dysregulation problems	0.02** [0.01, 0.02]	0.25 [0.17, 0.34]	0.01** [0.00, 0.02]	0.13 [0.03, 0.23]	−0.00 [−0.03, 0.02]	−0.01 [−0.13, 0.12]	−0.04 [−0.08, 0.01]	−0.18 [−0.47, 0.04]
Competence	−0.00 [−0.01, 0.00]	−0.06 [−0.15, 0.04]	−0.01 [−0.01, 0.00]	−0.08 [−0.17, 0.02]	0.01 [−0.01, 0.04]	0.07 [−0.05, 0.18]	−0.05 [−0.06, 0.01]	−0.11 [−0.27, 0.06]

Variance explained by postnatal depressive symptoms is partialled out by means of Cholesky factoring

Child's sex and parents' age, education and parity are included as covariates in all analyses

* $p < 0.05$; ** $p < 0.01$

Table 3 Effects of maternal and paternal postnatal depressive symptoms on children's developmental outcomes at 18 months

	Regression coefficients adjusted for covariates only				Regression coefficients adjusted for the other parent's postnatal symptoms and covariates			
	Mothers		Fathers		Mothers		Fathers	
Children's developmental outcomes	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]	*b* [95% CI]	β [95% CI]
Cognitive functioning	−0.06 [−0.58, 0.47]	−0.01 [−0.09, 0.07]	−0.45 [−1.14, 0.24]	−0.06 [−0.15, 0.03]	−0.19 [−0.82, 0.45]	−0.03 [−0.12, 0.07]	−0.18 [−1.05, 0.69]	−0.02 [−0.14, 0.09]
Receptive language	−0.18 [−0.70, 0.34]	−0.03 [−0.10, 0.05]	−1.27** [−1.95, −0.59]	−0.17 [−0.25, −0.08]	0.26 [−0.36, 0.89]	0.04 [−0.04, 0.14]	−1.28**[−2.10, −0.46]	−0.16 [−0.27, −0.06]
Expressive language	−0.12 [−0.55, 0.30]	−0.02 [−0.10, 0.06]	−0.81** [−1.36, −0.26]	−0.13 [−0.22, −0.04]	0.14 [−0.36, 0.64]	0.02 [−0.07, 0.12]	−0.80* [−1.42, −0.17]	−0.13 [−0.23, −0.03]
Externalizing problems	0.01* [0.00, 0.02]	0.12 [0.03, 0.21]	0.01 [−0.00, 0.02]	0.06 [−0.04, 0.16]	0.01*[0.00, 0.02]	0.10 [0.00, 0.20]	0.00 [−0.01, 0.01]	0.00 [−0.12, 0.12]
Internalizing problems	0.01* [0.00, 0.01]	0.09 [0.01, 0.18]	0.01* [0.00, 0.02]	0.13 [0.03, 0.22]	0.00 [−0.01, 0.01]	0.03 [−0.08, 0.14]	0.01[−0.01, 0.02]	0.11 [−0.02, 0.24]
Dysregulation problems	0.02** [0.02, 0.03]	0.28 [0.20, 0.36]	0.02** [0.00, 0.03]	0.19 [0.09, 0.28]	0.02** [0.01, 0.03]	0.25 [0.15, 0.35]	0.01[−0.01, 0.02]	0.07 [−0.05, 0.19]
Competence	−0.01 [−0.02, 0.00]	−0.07 [−0.17, 0.02]	−0.01 [−0.01, 0.00]	−0.08 [−0.17, 0.01]	−0.01 [−0.02, 0.00]	−0.08 [−0.19, 0.03]	−0.00 [−0.01, 0.01]	−0.02 [−0.12, 0.08]

Child's sex and parents' age, education and parity are included as covariates in all analyses

*$p < 0.05$, **$p < 0.01$

Parenting Stress as a Mediator

Each parent's experience of parenting stress, measured when the children were 12 months old, was examined as a mediator of the significant adjusted relations between postnatal depressive symptoms and the children's outcomes when they were 18 months old. As depicted in Fig. 3, three significant mediation effects emerged: (a) the relation between maternal postnatal depression and externalizing problems was mediated by maternal parenting stress at 12 months (indirect effect: $b =$ 0.02, 95% CI [0.01, 0.03]; β = 0.24, 95% CI [0.16, 0.33]); (b) a parallel mediation effect was found for dysregulation problems ($b = 0.02$, 95% CI [0.01, 0.03]; β = 0.22, 95% CI [0.15, 0.30]); and (c) for fathers, the relation between postnatal depressive symptoms and child receptive language development was mediated by paternal parenting stress at 12 months ($b = -0.56$, 95% CI [−1.01, −0.13]; β = −0.07, 95% CI [−0.13, −0.02]). The relation between paternal postnatal depressive symptoms and child expressive language was not significantly mediated by parenting stress ($b = -0.31$, 95% CI [−0.71, 0.04]; β = −0.05, 95% CI [−0.11, 0.01]).

Discussion

In this study, we found that parental depressive symptoms, which were highly stable over the perinatal period, predicted children's externalizing, internalizing, and dysregulation problems, as well as language developmental delay at 18 months of age. There was no evidence of independent prenatal effects on children's developmental outcomes, across a broad range of developmental domains. A differential effect was demonstrated, linking fathers' depressive symptoms to language outcomes, and mothers' symptoms to adverse effects in the social-emotional domain. There was no evidence of stronger associations between depressive symptoms and child outcomes when both parents showed high symptom loads. Examining parenting stress at 12 months as a broad mediator between both parents' postnatal depressive symptoms and child outcomes, we found that it mediated the relations between maternal dysphoria and both externalizing and dysregulation problems in the children, as well as paternal postnatal depressive symptoms and receptive communication in the 18-month-old children.

The findings of an association between parental depressive symptoms and aberrant social-emotional development in toddlers are in accordance with the extant literature (Field 2010; Galéra et al. 2011; Ramchandani and Psychogiou 2009; Velders et al. 2011). The effect sizes were generally small, especially regarding internalizing symptoms, which might reflect the fact that the toddlers were only 18 months old (Sanner et al. 2016). Further, we found little evidence to support a hypothesis explicating that depressive symptoms affect

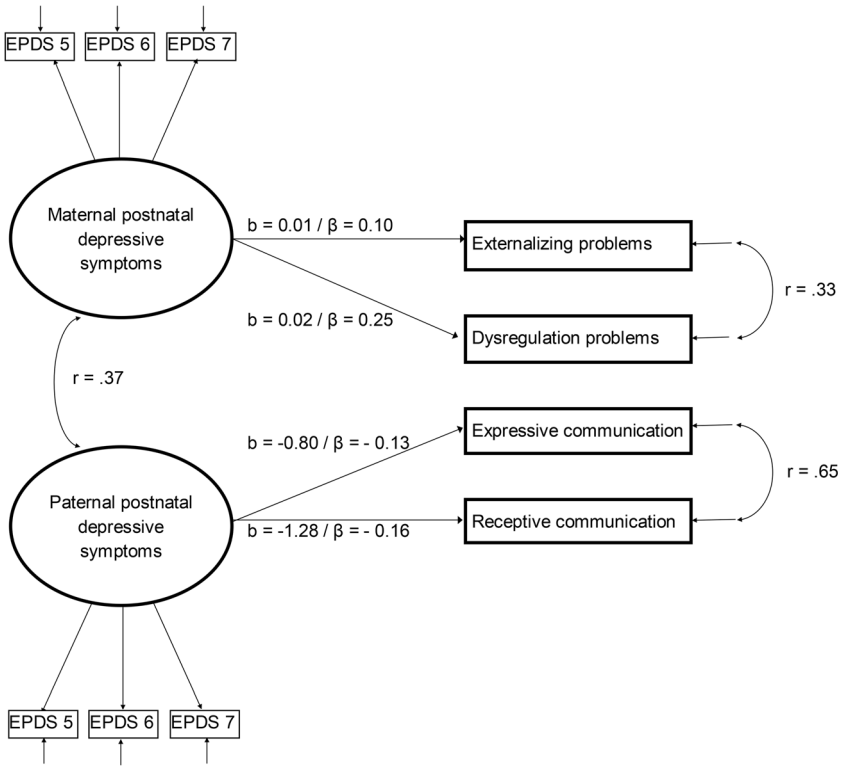

Fig. 2 Children's' developmental outcomes at 18 months predicted by postnatal parental depressive symptoms, adjusted for the other parent's postnatal depressive symptoms. Note. Only significant relations shown ($p < 0.05$). Analyses are adjusted for child's sex and parents' age, education, and parity. Model fit: $\chi^2[56] = 83.82$; CFI = 0.982; TLI = 0.962; RMSEA = 0.022, 90% CI: 0.011, 0.031

social-emotional competence in toddlers. While there are reports of this association (Cummings et al. 2005), findings have been mixed (Conroy et al. 2012); our results add to the interpretation that parental perinatal symptoms are associated with higher levels of social-emotional difficulties in toddlers, whereas the development of social-emotional competence remains unaffected at this age.

Language developmental delays following parental perinatal dysphoria, both for receptive and expressive communication, have been reported elsewhere (Paulson et al. 2009; Quevedo et al. 2012), and, similar to these studies, our effect sizes were generally small. We did not find that parental depressive symptoms predicted developmental delay in the cognitive domain. This is in contrast to some research reporting associations between postnatal depressive symptoms and child cognitive outcomes (Sutter-Dallay et al. 2011); however, several studies did not find these links (Kaplan et al. 2014; Piteo et al. 2012). Research suggests the importance of the persistence of depressive symptoms for child cognitive developmental delay to follow (Quevedo et al. 2012; Sutter-Dallay et al. 2011). Thus, such effects may not have appeared yet because of the young age of the participating children.

Pre- and Postnatal Depressive Symptoms

Parental depressive symptoms were highly stable over the perinatal period. Our findings are in accordance with studies of the entire perinatal period, suggesting that for a sizeable proportion of women there is a continuation of prenatal depressive symptoms to the postnatal period (Fredriksen et al. 2017; Mora et al. 2009; Wisner et al. 2013). Looking at the pre- and postnatal periods separately, there was no evidence of a unique prenatal effect of depressive symptoms independent of postnatal symptoms on any of the child outcomes. With such a high stability in depressive symptoms across periods, this could probably be expected. A reasonable interpretation would be that depressive problems in the perinatal period might be conceived as the manifestation of a common underlying problem across the entire period. Further, there were no apparent differences between mothers and fathers; had there been an exclusive maternal prenatal association, this might have been suggestive of an intrauterine effect. Taken together, our data provide little evidence for a direct physiological influence on the fetal environment leading to aberrant child development, following maternal prenatal depressive symptoms in a community sample, such as this. This corresponds with other reports (Van Batenburg-Eddes et al. 2013; Velders et al. 2011; Waters et al. 2014). However, this study design cannot disentangle whether early developmental risk following parental prenatal depressive symptoms originates in a genetic transmission of risk, an underlying contextual variable, or the continuation of prenatal depressive symptoms into the postnatal period (in which a direct exposure to parental affect, cognitions, and behaviors is likely to occur).

Surprisingly, a positive association between maternal prenatal depressive symptoms and cognitive functioning in the child emerged when postnatal depressive symptoms were

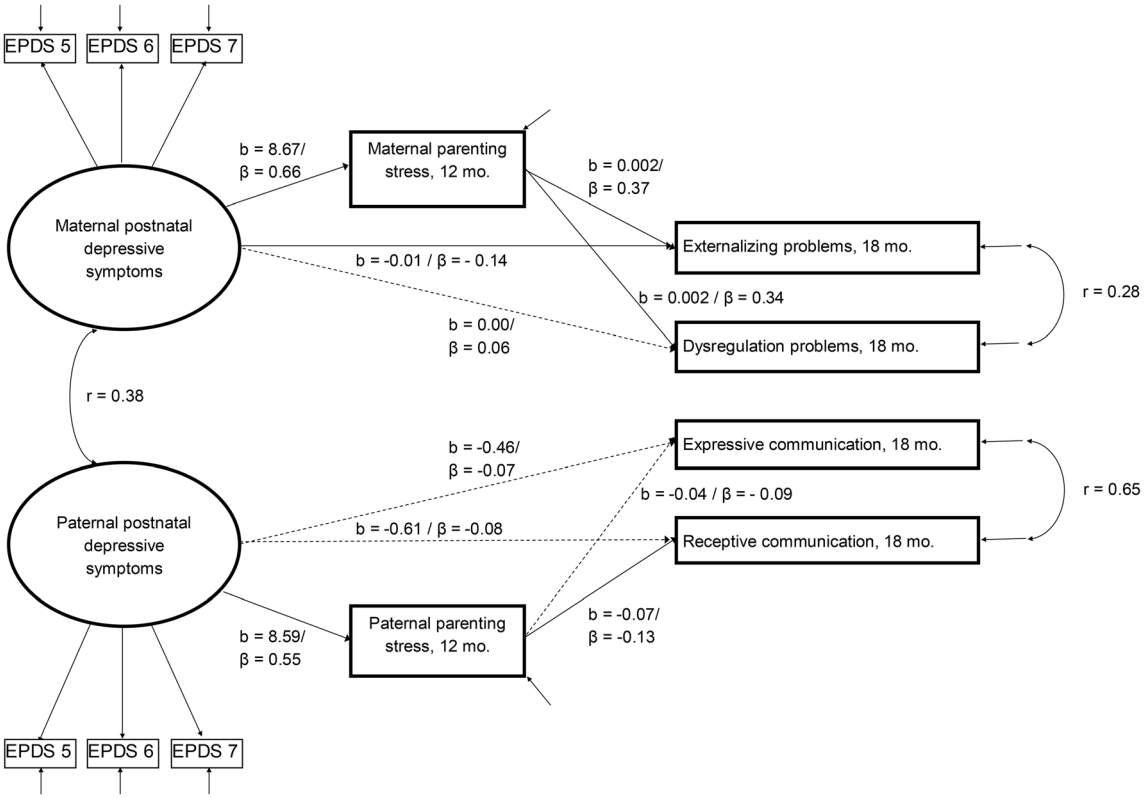

Fig. 3 Parenting stress as a mediator of the relation between postnatal depressive symptoms and child developmental outcomes. Note: (a) Maternal postnatal depression symptoms - > Maternal parenting stress - > Externalizing problems: Indirect effect $b = 0.02$, $\beta = 0.24$, $p < 0.01$. (b) Maternal postnatal depression symptoms - > Maternal parenting stress - > Dysregulation problems: Indirect effect $b = 0.02$, $\beta = 0.22$, $p < 0.01$. (c) Paternal postnatal depression symptoms - > Paternal parenting stress - > Receptive communication: Indirect effect $b = -0.56$, $\beta = -0.07$, $p < 0.01$. Model fit: $\chi^2[82] = 140.87$; CFI = 0.970; TLI = 0.959; RMSEA = 0.026, 90% CI: 0.019, 0.034. Analyses are adjusted for child's sex and parents' age, education, and parity. Solid lines indicate significant relations, broken lines indicate non-significant relations

partialled out. This finding may be a statistical artefact when partialling out the high proportion of variance that is due to postnatal depressive symptoms. However, alternative interpretations may be possible, such as compensatory measures directed toward the families when the mothers suffers from depressive symptoms in pregnancy, which in turn leads to improved cognitive functioning in the child when controlling for postnatal depressive symptoms.

Differential Relations between Maternal and Paternal Symptoms and Child Outcomes

The differential effect of maternal and paternal symptoms found in this study expands the current literature by relating toddlers' dysregulation difficulties, and to somewhat lesser degree externalization problems, to maternal perinatal depressive symptoms, and language developmental delay to paternal depressive symptoms. The finding that language development is associated with paternal (and not maternal) depressive symptoms has been reported in one previous study (Paulson et al. 2009). Moreover, Paulson et al.'s findings indicated that fathers, to a greater extent than mothers, reduced reading activities with their children when affected by depression, which in turn had an adverse effect on language development. In contrast mothers, continued to read a substantial amount to their children even when affected by depressive symptoms. This differential effect has been interpreted within the framework of current gender roles, in which depressed fathers might more easily withdraw from parenting tasks, whereas depressed mothers to a larger degree continue to spend time with their children, as they might not be able to withdraw from childrearing activities (Paulson et al. 2009). These notions might also shed light on the associations between maternal depressive symptoms and toddlers' social-emotional difficulties found in this study. A large body of literature indicates that prolonged caretaking by a depressed parent poses risks of adverse social-emotional development (Field 2010; Tronick and Reck 2009; Wang and Dix 2013). Given that mothers on average spend more time with their children than fathers, the maternal symptom load may comparably have a larger negative impact on child social-emotional development, as depressive symptoms such as feelings of sadness, flat affect, and negative thinking interfere with the behavioral and emotional exchanges between parent and child. Alternative

interpretations should also be considered, such as the possibility of genetic transmission of risk operating differently for each sex (Kendler et al. 2006), or that parental depressive symptoms may affect parenting behavior differently for mothers and fathers (Connell and Goodman 2002).

Moderating Effect of both Parents' Symptom Load

Further, we investigate whether effects of depressive symptomatology on child outcomes are particularly strong when both parents suffered from high symptom loads, as having two dysphoric parents might represent an additional burden for the children, and simultaneously also lack the potential buffering effect provided by the non-dysphoric parent. Few studies have examined this, and of those, the results have been mixed (Goodman et al. 1993; Mezulis et al. 2004; Paulson et al. 2006. We found no evidence of stronger associations between depressive symptoms and child outcomes when both parents showed high symptom loads in this study, suggesting that having two dysphoric parents is not associated with disproportionately larger adverse effects.

Parenting Stress Mediates Relations between Depressive Symptoms and Outcomes

We tested whether parenting stress functioned as a mediator of the significant, adjusted relations between postnatal depressive symptoms and child outcomes, and found that three out of four associations were mediated. This suggests that parenting stress in either parent operates as a broad mechanism between depressive symptoms and diverse child developmental outcomes. Specifically, our findings suggest that fathers' parenting stress mediates the relation between paternal depressive symptoms and children's receptive communication. However, for expressive communication there was no such mediation effect, suggesting that the development of receptive communication is more susceptible to parenting influences than the development of expressive communication. Mothers' parenting stress mediated the relations between maternal postnatal depressive symptoms and both externalizing and dysregulation difficulties in their children. This is in accordance with a recent review showing that parenting stress is more strongly related to externalizing than internalizing problems (Barroso et al. 2017), suggesting that parenting plays a significant role in how maternal depressive symptoms affect social-emotional difficulties in toddlers.

Limitations

There are several methodological issues relevant to understanding the results of this study. The response rate was 50.7%, and as in any community-based research, there is a possibility of self-selection bias with an overrepresentation of healthy and resourceful participants. Further, selective dropout might be a concern, as dropout was related to depressive symptoms in this study. However, by using FIML to handle missing data we attempted to reduce the impact of such selective attrition. Yet another concern regarding representativeness is the specific societal context. Norway has relatively generous perinatal welfare policies, with an emphasis on including fathers. Our findings might not be generalizable to contexts with less generous social policies.

Analyses revealed that even though model fit was satisfactory for models exercising strong measurement invariance on the EPDS across all seven time points, such models did not fit the data as well as less constraint models, especially among mothers. This indicates that the measure of depressive symptoms has been interpreted somewhat differently across time points, thereby calling for some uncertainty in the interpretation of the pre- and postnatal comparisons.

Several of our measures are self-report questionnaires; this includes the EPDS, PSI and ITSEA. Relations among these measures might be inflated due to shared method variance. However, the Bayley-III, a developmental test administered by health nurses, exhibits relations to depressive symptoms comparable to those of the ITSEA. Relatedly, depressive symptoms were measured by self-report only, and therefore provide no information about a clinical diagnosis of depression. Further, although the EPDS has been validated for use among male samples (Matthey et al. 2001), it has been criticized for not being sensitive to typically male symptoms of depression (Madsen and Juhl 2007). This might have led to an underestimation of the severity level of depressive symptoms among men, and we should be cautious to conclude that the mean level difference between male and female depressive symptoms in this study represents the true difference in the population.

Although these findings stem from a longitudinal design and the parental depressive symptoms precede child behavior chronologically, the directionally of effects can not conclusively be teased apart. There might be factors related to the fetuses and infants affecting parental mood that have not been accounted for in this study. Further, there might be unknown contextual factors accounting for both parental mood and child behavior.

Conclusions and Future Directions

Despite the limitations discussed above, this study shows that perinatal depressive symptoms in both mothers and fathers have a wide impact on toddlers' social-emotional and language development. Further, the study documents that there is a differential effect of maternal and paternal symptoms, and that parenting stress operates as a broad mediator in the relations between parental depressive symptoms and elevated early developmental risk.

From a clinical perspective, these findings suggest areas of child and family functioning that may be affected by elevated parental depressive symptoms and emphasize the importance of not only a continued focus on maternal mental health in the perinatal period, but also an awareness of depression in fathers. Given the stability of symptoms across the perinatal period, this study underscores the importance of early intervention.

Research efforts aimed at advancing knowledge on the effect of both parents' depressive symptoms at later stages during childhood are necessary. There is also a need to investigate the effect of chronic exposure to depression and how pre- and postnatal depression might interact to predict child outcomes. Finally, a continued focus on understanding the underlying mechanisms of risk transmission is central to advancing knowledge in this area of research.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in the study were in accordance with the ethical standards of the institutional and/or national research committee (approved by the Regional Committees for Medical and Health Research Ethics in Norway) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent All participants provided informed consent.

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