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UNIVERSITÀ EDITRICE

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Psychology Hub (2021)
XXXVIII, 1, 21-30

Do antenatal maternal and paternal depression levels affect emotional availability during mother-child and father-child interactions?

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Article info

Submitted: 20 December 2020
Accepted: 15 February 2021
DOI: 10.13133/2724-2943/17434

Abstract

This study determined the predictive value of antenatal maternal and paternal depressive symptoms in emotional availability (EA) during mother-child and father-child interactions. A self-reported measure of depression was administered to 50 couples of primiparous parents during the seventh/eighth month of pregnancy, and an observational system was applied to assess parent-child EA when the children were aged 7 and 9 months. To disentangle the contributions from the mother and fathers, 2 moderated regression models were tested separately for maternal and paternal EA, with maternal and paternal depressive symptoms and their interaction as predictors and the partners' EA and age as controls. In the first model, maternal depressive symptoms ($\beta = -.32$, $p = .01$) and paternal EA ($\beta = .57$, $p < .01$) had a significant effect, and there was a significant interaction between maternal and paternal depressive symptoms ($\beta = -.24$, $p = .03$). No significant effects emerged for paternal depressive symptoms or maternal age. A simple-slope analysis of the interaction showed that for high levels of depression in fathers (+1 SD), the relationship between maternal depressive symptoms and maternal EA was negative and significant ($r = -.56$, $p = .005$), whereas for low levels (-1 SD), this relationship tended to dissipate. Regarding the second model, no significant effects were found for maternal or paternal depressive symptoms or their interaction. Overall, the results suggest that high levels of maternal depressive symptoms during pregnancy impairs the development of adequate maternal EA. This risk increases when maternal and paternal depressive symptoms are high, implicating the importance of the relationship between parents. These effects were not observed for paternal EA.

Keywords: prenatal depressive symptoms; mothers; fathers; parental emotional availability; parent-child interactions.

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Introduction

Studies in Infant Research, attachment theory, and dynamic developmental psychology have widely documented the role of early relational experiences in modeling a child's socioemotional and cognitive development (Emde, 2014; Stern, 1985). The child is a "co-builder" of his relational development. Soon after birth, the infant shows sophisticated perceptivo-sensory and communicative abilities that serve his primary need to attune to significant others and share their subjective experiences (Trevarthen, 2011). The infant also shows articulated affect regulation strategies that are used to modulate his arousal and actively involve parents in dyadic interactions. During interactions, *emotionally available* parents (Emde, 1980) respond to their infant's interactive initiatives and, simultaneously, compensate for the still-immature regulatory strategies that infants use to attain a steady state following stress (Sander, 1987). These processes require the parents to be *sensitive*—that is, able to perceive, interpret, and respond appropriately to the emotional underpinnings of their child's overt behavior (Ainsworth, 1967). Parental *sensitivity* does not emerge when the infant is born; rather, it originates during pregnancy, when mothers and fathers experience dramatic reorganization in developing a parental identity and an emotional bond with their still-unborn infant (Condon et al., 2008; Habib & Lancaster, 2010; Tambelli et al., 2019, 2020).

Depression is a relevant risk condition that can interfere with parental adjustment during pregnancy, impairing the quality of caregiving behaviors (George & Solomon, 1999) that parents should exercise after childbirth to fulfill their child's primary emotional needs (Tambelli et al., 2014). Whereas maternal depression has been examined extensively (during pregnancy and in the postpartum period), paternal depression has been long overlooked.

Maternal depression is more frequent and severe during pregnancy than in the postpartum period (Evans et al., 2001; Sidebottom et al., 2014). Depression symptoms affect 4% to 25% of expectant women (Teixeira et al., 2009) and occur more frequently during the first and third trimesters of pregnancy, when women prepare themselves for delivery (Bunevicius et al., 2009; Gavin et al., 2005; Lee et al., 2007).

The emerging literature on paternal depression suggests that men are more likely to develop depressive symptomatology when their partners are in the early stages of the third trimester of pregnancy (Boyce et al., 2007; Condon et al., 2004). In fathers, the prevalence of perinatal depression, which varies from 2.3% to 12%, is approximately half that in mothers (Bennett et al., 2014; Ramchandani et al., 2008; Top et al., 2016).

There is clear evidence of the harmful impact of prenatal depression on maternal functioning and child development (Field, 2011; Glover, 2014). Such a negative impact is also relevant when considering mothers with depressive levels below the clinical threshold (Ammaniti et al., 2006; Tambelli et al., 2014). Mothers who suffer from perinatal depression exhibit heterogeneous insensitive behaviors, characterized by intrusiveness and emotional withdrawal during interactions with children (Field, 2010; Weinberg & Tronick, 1997). To cope with the negative affects that emerge from these

interactions, children tend to develop a negative emotional core—characterized by anger and sadness—associated with a lack of confidence in his relational competence and in the emotional availability of the mother to act as an external regulator for him (Speranza et al., 2006). These early dysfunctional relational experiences lead to a high risk of externalizing and internalizing psychopathologies in children (Goodman et al., 2011). Notably, it has been showed that maternal sensitivity "is a likely mechanism that links prenatal maternal depression (...) to infant and child bio-behavioral outcomes (e.g., difficult emotion regulation, altered cortisol patterns)" (Warnock et al., 2016; p. 3).

These evidences suggest that prenatal emotional suffering persists after childbirth, undermining the quality of the affective environment that mothers provide to their infants. Although the research on the detrimental effects of prenatal depression on the parent-child relationship has primarily considered mothers, similar scenarios are expected to be observed in fathers. These assumptions are supported by the findings of a recent longitudinal study that demonstrated that prenatal depression persists through 6 months in 75% of mothers and 86% of fathers, indicating the stability of symptom severity over time (Paulson et al., 2016).

Recent research has demonstrated that fathers who suffer from perinatal depression have impairments in parental functioning that are comparable to those in mothers (Davis et al., 2011). Moreover, consistent with the literature on mothers, children of depressed fathers are more likely to develop early behavioral and emotional problems and psychopathological disorders (Ramchandani et al., 2005, 2008).

That perinatal depression in either parent increases the likelihood of the partner developing perinatal depressive symptoms must be considered (Paulson & Bazemore, 2010; Paulson et al., 2016; Wee et al., 2011). This evidence has significant implications for the well-being of children. The risk for negative developmental outcomes rises when children are exposed to both parents suffering from depressive symptomatology (Foley et al., 2001; Nishimura & Ohashi, 2010). Under these conditions, a nondepressed parent cannot mitigate the negative effects of the partner's depression on a child's development, as reported in studies of nondepressed fathers and depressed mothers (Edhborg et al., 2003). Consistent with these findings, the importance of adopting a dynamic developmental systems approach has been stressed in simultaneously examining the paternal influences on maternal parenting and the maternal influences on paternal parenting (Malmberg & Flouri, 2011).

Aims of the Study

Considering the negative impact of prenatal depressive symptomatology on parental functioning and child development (Ammaniti et al., 2006; Tambelli et al., 2014), we tested the feasibility of a perspective investigation on expecting mothers and fathers with depressive levels below the clinical threshold. We were interested to examine whether antenatal maternal and paternal depression levels would predict the

quality of mother-child and father-child relationships. To do this, we analyzed the interaction effects between maternal and paternal depressive symptoms on emotional availability (EA) during mother-child and father-child interactions.

We expected that antenatal paternal depressive symptoms would moderate the relationship between maternal depressive symptoms and maternal EA (controlling for paternal EA). Moreover, we anticipated an analogous moderating effect of antenatal maternal depressive symptoms on the relationship between paternal depressive symptoms and paternal EA (controlling for maternal EA).

Materials and Methods

Participants

Seventy-eight couples of primiparous parents, reporting depressive symptoms below the clinical threshold, were initially recruited in the third trimester of pregnancy from maternity and child health services. Twenty-eight parental couples dropped out of the study, thus the final sample consisted of fifty couples of primiparous mothers (mean age = 33.88 years; SD = 4.58) and primiparous fathers (mean age = 36.90 years; SD = 6.69). All couples were married or cohabitating. Among mothers, 8% reported at least 1 hospitalization during pregnancy, whereas 66% did not report any complications during gestation; 80% did not report any previous abortions.

Prior to data collection, parents received complete information about the study procedures and provided written informed consent to participate in the research per the Declaration of Helsinki. This study was approved by the ethics committee of the authors' institution (Prot. n. 0001002 - 2019 - UOR: SI000092 - Classif. VII/15).

Procedure

At 7–8 months month of pregnancy, a sociodemographic interview was administered to the mothers and fathers, with a self-reported questionnaire that assessed depressive symptomatology [ie, the Edinburgh Postnatal Depression Scale (EPDS)]. At 6–9 months after childbirth, the mother-child and father-child free-play-home interactions (lasting approximately 15–20 minutes) were filmed and coded per the Emotional Availability Scale (EAS).

Measures

Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1987; Italian validation by Benvenuti et al., 1999). The EPDS comprises 10 items that measure the intensity of a set of depressive symptoms during the previous week. Although the EPDS was originally designed to measure maternal postnatal depressive symptoms, several studies have shown evidence of its validity in assessing antenatal depressive symptomatology, in mothers (Lydsdottir et al., 2014) and fathers (Edmondson et al., 2010; Massoudi et al., 2013). The Italian version of the

EPDS has an adequate level of reliability in terms of internal consistency (Cronbach's $\alpha = .79$; Guttman split-half index = .82). A global depression index can be calculated by summing the scores for all items, with the following cutoff values: 12/13 for clinical depression and 9/10 for screening purposes (Benvenuti et al., 1999).

Emotional Availability Scale (EAS; Biringen et al., 2000, 2008). The EAS was used to evaluate the quality of filmed mother-child and father-child interactions. The EAS comprises 6 scales—each ranging from 1 (highly emotional unavailable) to 7 (highly emotional available)—that evaluate various facets of parent-child EA. The first 4 scales assess parental EA toward children (*Sensitivity*, *Structuring*, *Non-Intrusiveness*, and *Non-Hostility*), and the remaining 2 evaluate the quality of the child's behavior toward his parents (*Responsiveness* and *Involvement*). *Sensitivity* regards the parental capacity to be affective, acceptant, flexible, and emotionally regulated toward their children and to be creative and to propose variety during dyadic interactions. *Structuring* refers to the parental ability to provide adequate rules, regulation, and supportive structure to parent-child interactions. *Non-Intrusiveness* concerns the parental capacity to promote the autonomy of children, avoiding excessively directive, stimulating, or protective interventions. *Non-Hostility* is conceived as a parental disposition to show positive emotional reactions in parent-child interactions. *Responsiveness* refers to children's tendency to explore and enjoy during dyadic interaction and to respond to the interactive initiatives of parents. *Involvement* concerns the willingness of children to have relations with their parents. Notwithstanding the theoretical distinction between these 6 scales, Wiefel and colleagues (2005) conducted an explorative factorial analysis (EFA) of all EA scales, revealing that the first factor that was extracted accounted for a high proportion of the variance (66.02%). These results support the possibility of computing a global score for EA. Consistent with these results and considering that high intercorrelation between EA scales was found in our study (ranging from $r = .323$, $p < .05$ to $r = .924$, $p < .01$; Cronbach's $\alpha = .88$), global scores for maternal and paternal EA were computed, calculating the mean of all scales for each participant. These global scores were used for subsequent analyses in our study. The EAS showed adequate inter-rater reliability for all scales, with intraclass correlation coefficient (ICC) values ranging from .81 to .93.

Data Analysis

A series of descriptive statistics were estimated for all variables included in the study (i.e., mean, standard deviation, asymmetry, and kurtosis). To illustrate the relationships among variables, a correlation matrix was reported. To evaluate whether maternal and paternal scores were nested, a canonical correlation was performed. To examine the predictive power of maternal and paternal depression levels during pregnancy on parental EA toward children at 6 and 9 months of life, two separate moderated regression models

were carried out, one on mothers' EA and the other on fathers' EA, respectively. Moreover, considering that maternal and paternal EA are nested, two other moderated regression models were conducted with the same pattern of variables, but also including partner's EA as an additional covariate. In this manner, the effects of maternal and paternal depression levels (as well as of their interaction) were computed, disentangling maternal and paternal EA components. All these analyses were performed using IBM Statistics SPSS v.24. For the multiple moderated regression analysis to reach a statistical power of .80 with medium effects and an alpha level of .05, approximately 50 subjects were needed (G*Power) (69).

Results

Descriptive Statistics

As shown in Table 1, all variables had distributions close to a normal curve (values of skewness and kurtosis were within ± 1), except for paternal EA, which showed a slight deviation for kurtosis. Mean age was significantly higher for fathers than for mothers [$t(49) = 3.82, p < .001$], whereas the mean level of depression was higher for mothers than for fathers [$t(49) = 4.29, p < .001$]. No significant differences in mean EA were found.

Tab. 1. Descriptive statistics

	Mean	SD	Skewness	Kurtosis
EPDS-M	6.34	4.02	.60	-.24
EPDS-P	3.58	2.95	.62	-.46
EA-M	5.78	.85	-.33	-.71
EA-P	5.57	.92	-1.14	2.27
AGE-M	33.88	4.58	-.50	.46
AGE-P	36.90	6.69	.17	.55

Note: EPDS-M: maternal Edinburgh Postnatal Depression Scale; EPDS-P: paternal Edinburgh Postnatal Depression Scale; EA-M: maternal Emotional Availability; EA-P: paternal Emotional Availability; Age-M: maternal Age; Age-P: paternal Age.

Table 2 illustrates the intercorrelations between age, depression levels, and EA for mothers and fathers. Significant correlations emerged between maternal and paternal age and between maternal and paternal EA, whereas a not significant correlation was found between maternal and paternal depressive symptoms during pregnancy. Moreover, a canonical correlation was conducted, including maternal age, maternal EA scores, and maternal depressive symptoms as predictors, and paternal scores for the same variables as criteria, revealing a significant relationship between them (canonical correlation = .64, $p < .001$). Given that that mothers' and fathers' scores on EA were nested, predictive models were conducted (see the following Section), including partners' scores on EA as covariates, to disentangle the unique contribution of maternal and paternal effects.

Tab. 2. Correlations among maternal and paternal measures

	EPDS-M	EPDS-P	EA-M	EA-P	AGE-M	AGE-P
EPDS-M	1					
EPDS-P	.18	1				
EA-M	-.15	.03	1			
EA-P	.08	-.03	.58**	1		
AGE-M	-.24	-.04	-.07	-.10	1	
AGE-P	-.09	.01	-.10	-.07	.56**	1

Note: ** The correlation is significant at 0,01 alpha level (two tailed). EPDS: maternal Edinburgh Postnatal Depression Scale; EPDS-P: paternal Edinburgh Postnatal Depression; EA-M: maternal Emotional Availability; EA-P: paternal Emotional Availability; Age-M: Maternal age; Age-P: maternal age.

Moderated Multiple Regression Models for Emotional Availability

To test the predictive value of paternal and maternal depressive symptoms and their interaction on maternal EA, a first moderated multiple regression analysis (Model 1 in Table 3) was performed, including maternal EPDS, paternal EPDS, and their interaction as predictors, maternal EAS scores as a criterion, and maternal age as a control variable. As shown in Table 3, Model 1 accounted for 13% of the maternal EA. There was a significant main effect of maternal depressive symptoms, a not significant main effect of paternal depressive symptoms, and a significant interaction between maternal and paternal depressive symptoms. No significant effect of age was observed.

The same pattern of variables was included in a second regression analysis (Model 2 in Table 3), adding paternal EAS scores as a further covariate, to estimate the effect of predictors on the residual component of maternal EAS scores (controlling for paternal EA scores). Model 2 accounted for 45% of the maternal EA, revealing significant main effects of maternal depressive symptoms and paternal EA, and a significant interaction between maternal and paternal depressive symptoms. No significant effects were found for paternal depressive symptoms or maternal age.

Tab. 3. Multiple regression models on maternal EA

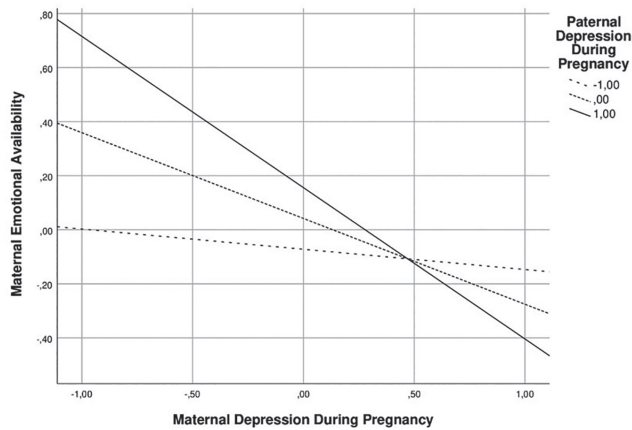
	Beta	SE	T	P	R ²	
Model 1	EPDS-M	-.30	.15	-1.91	.06	.13
	EPDS-P	.09	.14	.64	.53	
	Int	-.29	.13	-2.15	.04	
	Age-M	-.18	.15	-1.21	.23	
Model 2	EPDS-M	-.32	.12	-2.55	.01	.45
	EPDS-P	.11	.11	.99	.33	
	Int	-.24	.11	-2.25	.03	
	Age-M	-.12	.12	-.99	.33	
	EA-P	.57	.11	5.04	.00	

Note: EPDS-M: maternal Edinburgh Postnatal Depression Scale; EPDS-P: paternal Edinburgh Postnatal Depression Scale; EA-P: paternal Emotional Availability; Age-M: Maternal Age; Age-P: Maternal Age. Int: Interaction between EPDS-M and EPDS-P.

To interpret the significant interaction between paternal and maternal depressive symptoms appropriately, a simple slope analysis was performed. As shown in Figure 1, for a high EPDS score for fathers (+1 SD), the relationship between maternal depressive symptoms and maternal EA was negative and significant [$r = -.56, t(49) = -2.94, p = .005$], whereas for a low paternal EPDS scores (-1 SD), the relationship between maternal depressive symptoms and maternal EA disappeared, suggesting a moderate role for paternal depressive symptomatology during pregnancy.

To test the predictive value of paternal and maternal depressive symptoms and their interaction in paternal EA,

Fig. 1. Simple effects of the significant interaction between paternal and maternal depression levels in the prediction of maternal emotional availability



a first moderated regression analysis (Model 1 in Table 4) was conducted, including maternal EPDS, paternal EPDS, and their interaction as predictors, paternal EAS scores as a criterion, and paternal age as a control variable. As shown in Table 4, Model 1 accounted for 2% of paternal EA.

No significant main effects emerged for maternal or paternal depressive symptoms or paternal age. Moreover, no significant interaction effect was found between maternal and paternal depressive symptoms.

A second moderated regression analysis of paternal EAS scores was performed using the same pattern of predictors and including also maternal EAS scores as an additional covariate, to estimate the effects of predictors on the residual component of paternal EA (Model 2 in Table 4). The predictors in this equation accounted for 38% of the variance in paternal EA. We observed not significant main effects for paternal depressive symptoms and age, and a not significant interaction between maternal and paternal depressive symptoms. Notably, maternal depressive symptoms showed a close to be significant effect (beta = .23, $p = .08$), indicating that it may become significant with a larger number of participants. Moreover, a significant main effect was found for maternal EA.

Tab. 4. Multiple regression models on paternal EA

	Beta	SE	T	p	R ²	
Model 1	EPDS-M	.06	.16	.39	.70	.02
	EPDS-P	.04	.15	-.26	.79	
	Int	-.06	.14	-.46	.65	
	Age-P	-.07	.15	-.45	.66	

	Beta	SE	T	p	R ²	
Model 2	EPDS-M	.23	.13	1.75	.08	.38
	EPDS-P	-.10	.12	-.82	.42	
	Int	.11	.12	.93	.36	
	Age-P	.03	.12	.24	.81	
EA-M	.64	.13	5.09	.00		

Note: EPDS-M: maternal Edinburgh Postnatal Depression Scale; EPDS-P: paternal Edinburgh Postnatal Depression Scale; EA-M: maternal Emotional Availability; Age-M: maternal Age; Age-P: Maternal Age. Int: Interaction between EPDS-M and EPDS-P.

Discussion

In this study, we examined the predictive value of antenatal maternal and paternal depression levels on mother-child and father-child relationships. To this end, we analyzed the interaction effects between maternal and paternal depressive symptoms on EA during mother-child and father-child interactions.

Consistent with our original expectations, the negative impact of maternal prenatal depressive symptoms on maternal EA tended to increase if it was associated with prenatal depressive symptoms in their partners. Our findings extend the results of other studies that have reported evidence of the detrimental effects of paternal postpartum depression on mother-child interaction to include the prenatal period (Malmberg & Flouri, 2011).

The evidence that the quality of mother-infant interactions, beyond being impaired by maternal prenatal depressive mood, is also jeopardized when maternal depressive mood is associated with its paternal counterpart implicates the significance of the role that expectant fathers have in facilitating (or compromising) the future relationship between expectant mothers and their children.

We assume that fathers with higher prenatal depressive mood experience greater difficulty in providing their partners with the emotional support that they need to confront their own depressive mood and to prepare themselves to interact positively with their children. These assumptions are consistent with the evidence that women who receive poor emotional support from their partners, beyond being at increased risk for developing depressive symptoms during pregnancy, develop impairments in parental functioning after childbirth (Jeong et al., 2013; Xie et al., 2009). This evidence indicates the importance of examining prenatal risk factors (especially depressive factors) that influence the parent-child relationship, by adopting a transactional approach that considers the influence that one partner has on the other (Schermerhorn & Cummings, 2008).

In contrast to our expectations, maternal prenatal depressive mood did not modulate the quality of father-child interactions after childbirth. These findings differ substantially from those in previous studies during the postnatal period. In such reports, maternal postpartum depression negatively affected the father's ability to be sensitive and emotionally engaged during interactions with his children (Malmberg & Flouri, 2011; Vakrat et al., 2018).

In interpreting our results, we consider the various means by which mothers and fathers address the transition to parenthood. Although deep reorganization is experienced by both parents during pregnancy, compared with fathers, mothers develop more intense and vivid mental representations concerning their infants and future relationship with them (Tambelli et al., 2020). These prenatal representations significantly affect the maternal proneness to be emotionally available to detect and regulate the inner states of their infants soon after birth (Ammaniti et al., 2006; Dollberg et al., 2010; Trentini et al., 2020).

In expectant mothers, these early representational processes are sustained by the *somatic experience of gestation* (Tambelli et al., 2020), which allows them to experience a *direct* interaction with their babies, who express their vitality through intrauterine movements and ultrasound images (Ammaniti et al., 2014; Fava Vizziello et al., 2012; Stern, 1995; Viaux-Savelon et al., 2012). In contrast, the emotional attunement that fathers develop during pregnancy with their still-unborn infants remains *indirect*, because it is mediated by the mothers' willingness to share the perceptions and emotions that emerge from the somatic experience of pregnancy (Schoppe-Sullivan et al., 2008). Consistent with this evidence, a recent study has reported that maternal prenatal representations predict attachment security (or insecurity) in children, during their first year of life (Tambelli et al., 2020). In the same study, father-child attachment was instead predicted by the quality of the father's ability to be emotionally available during the interactive exchanges, rather than by his prenatal representations. Based on these findings, given that expectant mothers *already* have a *direct* interaction (due to somatic experience) with their infants, this early dyadic interaction is likely to be precociously impaired (as observed in the postpartum period) when antenatal depression levels increase jointly in mothers and fathers. In contrast, the influence of a partner's depressive symptomatology on paternal functioning might fully emerge only after childbirth—ie, when fathers have the possibility to *really* interact with the *real* child (Lebovici et al., 1983).

Conclusions

Emerging research on parenting is beginning to recognize the importance of focusing on both mothers and fathers, when studying the effects of parental postpartum depression on parent-child relationships. Nevertheless, the influence of prenatal depressive symptoms on parental EA has not been examined extensively. In this study, we addressed this issue, finding that when both parents report depressive symptoms during pregnancy, the impact of the father's emotional distress has a broader influence on the subsequent mother-child interaction than previously acknowledged. These findings (albeit preliminary) have not been documented by other groups.

A methodological constraint of our study is the low number of couples ($N = 50$), which decreased the power of the statistical tests. As clarified in the data analysis section, the number of participants was sufficient to detect moderate but not low effect size. Future studies should include more participants to increase the statistical power of the significance tests.

Moreover, the limited number of participants did not allow us to apply a structural equation modeling (SEM) analytical strategy to account for the interdependence between partners. In particular, the Actor-Partner Interdependence Model (APIM; Cook & Kenny, 2005) distinguishes the contributions of maternal and paternal depressive symptomatology in the prediction of a partner's EA, comparing various patterns of the effects (ie, no pattern, only actor pattern, only partner pattern, couple-oriented pattern, and contrast pattern).

An additional limitation is that the study was conducted in a nonclinical population, which might have reduced the variability in EPDS and EAS scores and underestimated the regression parameters.

Finally, this study omitted other variables—such as marital satisfaction, perceived social support, and infant characteristics (such as temperament)—that are relevant to parental depressive symptomatology and parenting (Jeong et al., 2013; Takács et al., 2019; Tambelli et al., 2019; Taraban et al., 2017). Their inclusion would have generated a more articulated profile of the predictive value of antenatal maternal and paternal depressive symptoms on maternal and paternal EA.

Notwithstanding these limitations, our study has significant clinical implications, guiding the prevention programs that could be designed during pregnancy to mitigate the adverse sequelae of paternal prenatal depressive symptomatology on mother-child relationships (Field, 2010).

Author Contributions

All authors contributed to the conceptualization and design of the study and agreed to ensure that the questions that were related to the accuracy or integrity of any part of the work were appropriately investigated and resolved. **CT:** Oversaw the composition of this manuscript. As first author, she was primarily accountable for all aspects of the work. **FD:** Analyzed the data and wrote parts of the manuscript (in particular, “Materials and Methods” and “Results”). He revised the paper for intellectual content and approved its final version to be published. **RT:** Monitored the data acquisition and made substantial contributions to the interpretation of data. She revised the paper for intellectual content and approved its final version to be published.

Compliance with Ethical Standards

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Funding

This research was supported by PRIN 2013/2016 - 20107JZAF4 grants from the Italian Ministry for Education, University and Research (MIUR).

Ethical approval

This study was approved by the Ethics Committee of the Department of Dynamic and Clinical Psychology, and Health Studies, Faculty of Medicine and Psychology, Sapienza University of Rome (Italy).

Acknowledgments

The authors thank the families who participated in this research.

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