Prenatal Maternal Depressive Symptoms, Child Problem Behavior, and Amygdala Volume: The Role of Harsh Parenting

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Abstract

Background: The association between prenatal maternal depression and adverse child outcomes is well established, but the role of potential moderating factors remains poorly understood. **Objectives:** This study investigates the role of harsh parenting in the association between prenatal maternal depressive symptoms and internalizing symptoms, externalizing symptoms, and amygdala volume in preadolescents. **Methods:** A total of 1,436 child-mother pairs from the Generation R study participated. Prenatal depressive symptoms, child internalizing and externalizing symptoms, and harsh parenting were assessed using questionnaires completed by the mothers. To measure amygdala volume, children visited the research center, where T1-weighted brain scans were obtained using a Magnetic Resonance Imaging scanner. Direct associations between prenatal depressive symptoms and internalizing symptoms, externalizing symptoms, and amygdala volume were initially examined using linear regression analyses. The moderating role of harsh parenting was investigated by including an interaction term with prenatal depressive symptoms. **Results:** Higher prenatal maternal depressive symptoms were significantly associated with increased internalizing symptoms (b_{adj} = 0.117, 95% CI [0.043, 0.191], p_{FDR} = .002) and increased externalizing symptoms in preadolescents (b_{adj} = 0.104, 95% CI [0.030, 0.178], p_{FDR} = .006), with internalizing symptoms showing the strongest association. We found no evidence for a moderating role of harsh parenting in these associations. However, harsh parenting was independently associated with externalizing symptoms (b_{adj} = 0.145, 95% CI [0.092, 0.198], p_{FDR} < .001). **Conclusion:** This study found that higher levels of prenatal depressive symptoms in mothers were associated with higher internalizing and externalizing symptoms in preadolescents, regardless of harsh parenting levels.

Keywords: preadolescence, prenatal depressive symptoms, harsh parenting, problem behavior, amygdala, population-based

Samenvatting

Achtergrond: De associatie tussen prenatale maternale depressie en ongunstige uitkomsten bij kinderen is goed vastgesteld, maar de rol van mogelijke modererende factoren is nog onvoldoende begrepen. **Doelstelling:** Deze studie onderzoekt de rol van harde ouderlijke discipline in de relatie tussen prenatale maternale depressieve symptomen en internaliserende symptomen, externaliserende symptomen en amygdala-volume bij preadolescenten. **Methode:** In totaal namen 1.436 moeder-kindparen van het Generation R-onderzoek deel. Prenatale depressieve symptomen, internaliserende en externaliserende symptomen en harde ouderlijke discipline werden gemeten met vragenlijsten ingevuld door de moeders. Voor het meten van het amygdala-volume bezochten kinderen het onderzoekscentrum, waar T1-gewogen hersenscans werden gemaakt met een Magnetic Resonance Imaging scanner. Directe associaties tussen prenatale depressieve symptomen en internaliserende, externaliserende symptomen en amygdala-volume werden eerst onderzocht met lineaire regressieanalyses. De modererende rol van harde ouderlijke discipline werd onderzocht via een interactieterm met prenatale depressieve symptomen. **Resultaten:** Hogere prenatale depressieve symptomen waren significant geassocieerd met verhoogde internaliserende symptomen (b_{adj} = 0.117, 95% CI [0.043, 0.191], p_{FDR} = .002) en externaliserende symptomen bij preadolescenten (b_{adj} = 0.104, 95% CI [0.030, 0.178], p_{FDR} = .006), waarbij internaliserende symptomen de sterkste associatie vertoonden. Er werd geen bewijs gevonden voor een modererende rol van harde ouderlijke discipline in deze associaties. Wel bleek harde ouderlijke discipline onafhankelijk geassocieerd met externaliserende symptomen (b_{adj} = 0.145, 95% CI [0.092, 0.198], p_{FDR} < .001). **Conclusie:** Deze studie toont aan dat hogere niveaus van prenatale depressieve symptomen bij moeders geassocieerd zijn met meer internaliserende en externaliserende symptomen bij preadolescenten, ongeacht het niveau van harde ouderlijke discipline.

Sleutelwoorden: preadolescentie, prenatale depressieve symptomen, harde ouderlijke discipline, probleemgedrag, amygdala, populatie-gebaseerd

Prenatal Maternal Depressive Symptoms, Child Problem Behavior, and Amygdala Volume: The Role of Harsh Parenting

Maternal depression is considered a significant public health concern, as it not only affects the mother, but can also have lasting negative effects on child development (Hentges et al., 2021; Severo et al., 2023). Maternal depression is a mood disorder that affects women during pregnancy and up to one year postpartum (Yonkers et al., 2012). It encompasses a range of symptoms, including persistent sadness, loss of interest in activities, anxiety, and hopelessness, which can significantly impair daily functioning (American Psychiatric Association, 2013). Recent literature suggests that early exposure to maternal depression may affect brain development in children and is associated with an increased risk of emotional and behavioral disorders in preadolescence (Roos et al., 2022).

Maternal depression can be categorized into prenatal and postnatal depression (Wen et al., 2017). Prenatal depression, also known as antenatal depression, occurs during pregnancy (Field et al., 2006) and affects approximately 1 in 10 women (Vlenterie et al., 2021). Although research has increasingly recognized the importance and high prevalence of prenatal depression (Field, 2011; Hein et al., 2014; Underwood et al., 2016), postnatal depression receives more social attention, even though research shows that more women experience depressive symptoms during pregnancy than after childbirth (Vlenterie et al., 2021). This pattern is evident in prevalence rates, as one study found that prenatal depression (29%) was nearly twice as common as postnatal depression (17%; Andersson et al., 2006), while another study reported a significantly higher incidence of prenatal depression (30%) compared to postnatal depression (23%; Edwards et al., 2008).

Given the negative outcomes for child development and the importance of further research, the current study focuses on the relationship between prenatal maternal depression, internalizing and externalizing symptoms, and brain development in preadolescents, to gain a better understanding of this relationship.

Prenatal Depression and Child Development

Accumulating research has shown that prenatal depression can affect not only the mother but also the unborn child, including an increased risk of developing mood disorders later in life (Goodman et al., 2011; Wen et al., 2017). Findings from Plant et al. (2015) and Weissman et al. (2006) indicate that children of mothers who experienced prenatal depression are three times more likely to develop depression themselves. In addition, prenatal exposure to maternal depression has been associated with higher levels of internalizing and externalizing problems in children (Goodman et al., 2011; Roos et al., 2022). Internalizing

problems refer to inward-directed difficulties that primarily affect the individual child (Mervielde et al., 2005; Muller et al., 2007). These problems are characterized by emotional instability and dysregulated affect, including anxiety disorders, mood disorders, and social withdrawal. In contrast, externalizing problems involve outward-directed behaviors that impact the social environment (Eisenberg et al., 2001). These behaviors often manifest as aggression, antisocial tendencies, and impulsivity. This category also encompasses behavioral disorders such as Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD). Adolescents exhibiting externalizing problems are at increased risk of developing criminal or violent behavior later in life (Van Berlo et al., 2016).

To gain a more comprehensive understanding of the associations between prenatal depression and child problem behavior, it is essential to consider the underlying neurobiological mechanisms. Recent studies suggest that the increased risk of internalizing and externalizing problems may be related to alterations in brain development among offspring, particularly in the amygdala (Roos et al., 2022; Soe et al., 2017). The amygdala is a brain structure that plays a crucial role in emotional processing and stress regulation (Wen et al., 2017). Alterations in amygdala volume have also been associated with exposure to prenatal depression (Roos et al., 2022; Soe et al., 2017). Research by Wen et al. (2017) indicates that prenatal maternal depression is linked to a larger amygdala volume in children aged 4.5 to 11 years, which in turn is associated with higher levels of internalizing and externalizing problems (Donnici et al., 2021; Wen et al., 2017). These findings suggest that structural brain changes may contribute to the emotional and behavioral development of children exposed to prenatal depression.

Prenatal Depression, Parenting and Child Problem Behavior

Although the association between prenatal depression, changes in child behavioral problems and brain development has been demonstrated (Donnici et al., 2021; Goodman et al., 2011; Wen et al., 2017), the role of potential moderators remains unclear. One factor that may contribute to internalizing or externalizing symptoms is parenting behavior (Goodman et al., 2011; Hentges et al., 2021; Pinquart, 2017). Poor parenting practices are considered a potential risk factor for the emergence of child problem behavior (Field, 2010). For example, harsh parenting increases the likelihood of externalizing problems in children (Goodman et al., 2011). Harsh parenting refers to parenting practices characterized by physical and/or verbal aggression, such as hitting, yelling, or the use of harsh punishment (Field, 2010). This form of discipline is often used to enforce desired behavior in children but may have unintended consequences, such as child problem behavior.

Previous research has not only demonstrated direct associations between harsh parenting and child problem behavior but has also shown that maternal depression is associated with increased use of harsh parenting (Lovejoy et al., 2000), suggesting that maternal depression may play a mediating role in this relationship (Hentges et al., 2021). A meta-analysis showed that maternal depression is associated with increased negative parenting behavior (Lovejoy et al., 2000). For example, mothers with depressive symptoms are more likely to use harsh punishment (Field, 2010), and maternal depression may also mediate the transmission of prenatal maternal depression to internalizing problems in children (Hentges et al., 2021). While these findings support a mediational pathway, it is important to acknowledge that although mediation has been the primary focus of research, moderation is also theoretically plausible (Field et al., 2006), but remains understudied. Moderation may provide an alternative or complementary explanation for how maternal depression and harsh parenting interact to affect child outcomes.

The 'multivariate cumulative risk model' proposed by Field (1992) emphasizes how early biological vulnerabilities, such as exposure to prenatal maternal depression, interact with adverse social factors to influence child development (Field et al., 2006). Harsh parenting, including physical or verbal aggression, is one such social factor that may be a potential moderator of these associations. According to this model, the accumulation of multiple risk factors increases the likelihood of negative outcomes later in life. Empirical evidence supports this model. For instance, a cohort study demonstrated that higher levels of prenatal risk, including maternal depressive symptoms, were associated with larger amygdala volume and increased emotional and behavioral problems in young children (Acosta et al., 2023). Furthermore, a longitudinal study found that harsh parenting was associated with alterations in the amygdala's functional connectivity, which subsequently increased the likelihood of externalizing behaviors (Koyama et al., 2024).

These findings align with the 'double risk' hypothesis (Sameroff & Seifer, 1983), which suggests that the co-occurrence of multiple adversities can magnify their individual negative effects. Prenatal depression and harsh parenting have both been independently linked to changes in brain structure and child behavior. However, to our knowledge, no prior studies have examined the role of harsh parenting in the association between prenatal maternal depressive symptoms, internalizing and externalizing symptoms, and amygdala volume in preadolescents. According to these theoretical frameworks, harsh parenting is expected to exacerbate the negative effects of prenatal depression on these outcomes. This study aims to fill this gap and to better understand the effect of harsh parenting on the relationship between

prenatal depressive symptoms and child development, in order to inform appropriate prevention and intervention strategies. Early identification and appropriate treatment of prenatal depression are crucial to safeguarding the health and well-being of both mother and child (Vlenterie et al., 2021).

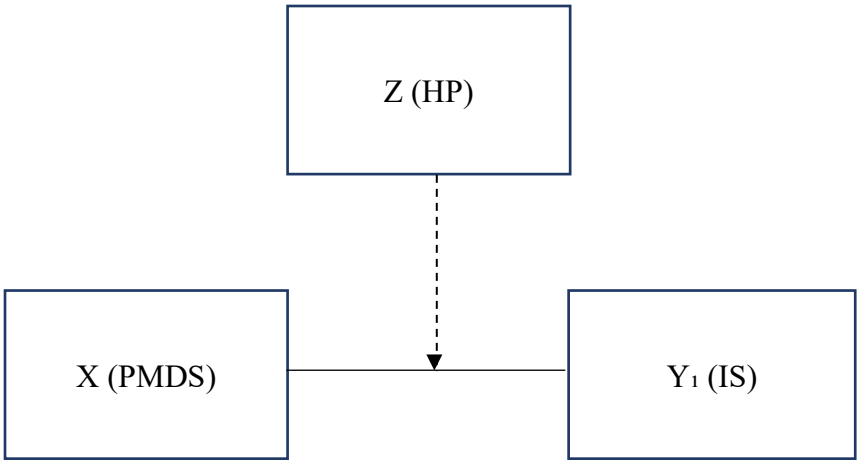
Current Study

The present study examines whether harsh parenting (Z) moderates the association between prenatal maternal depressive symptoms (X) and internalizing symptoms (Y_1), externalizing symptoms (Y_2), and amygdala volume (Y_3) in preadolescents. Previous research has identified amygdala volume as a potential mediator in the association between maternal depression and child problem behavior. However, based on theoretical considerations, the present study focuses on investigating the individual associations and specifically examines the potential moderating role of harsh parenting.

In the current study three sub-questions are addressed regarding the direct associations between the variables: to what extent is prenatal depressive symptoms (X) associated with 1) internalizing symptoms, 2) externalizing symptoms, and 3) amygdala volume in preadolescents? Based on previous research, the following hypotheses are proposed: prenatal maternal depressive symptoms are expected to be associated with (H1) higher levels of internalizing symptoms, (H2) higher levels of externalizing symptoms, and (H3) a larger amygdala volume in children (Donnici et al., 2021; Goodman et al., 2011; Wen et al., 2017). In addition, (H4) harsh parenting is hypothesized to strengthen all of these direct associations (Donnici et al., 2021; Goodman et al., 2011; Hentges et al., 2021; Pinquart, 2017; Roos et al., 2022; Wen et al., 2017). The conceptual models are presented below.

Figure 1

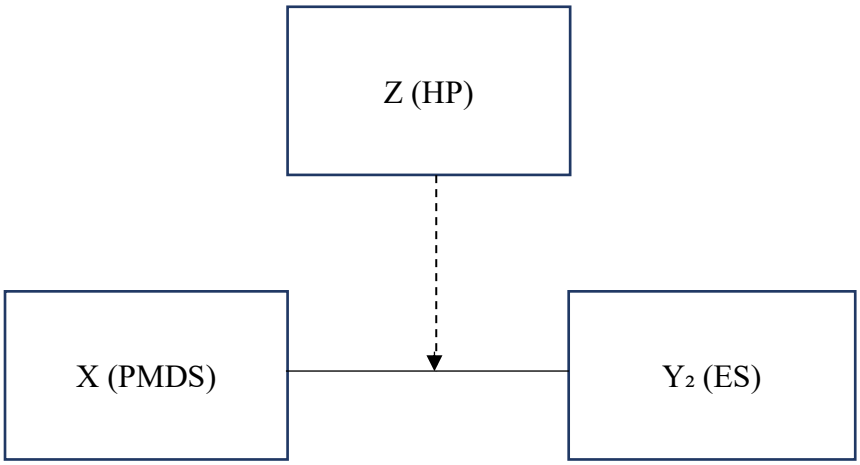
Conceptual framework of the association between prenatal depressive symptoms and internalizing symptoms with harsh parenting as moderator



Note. PMDS = Prenatal Maternal Depressive Symptoms. IS = Internalizing Symptoms. HP = Harsh Parenting.

Figure 2

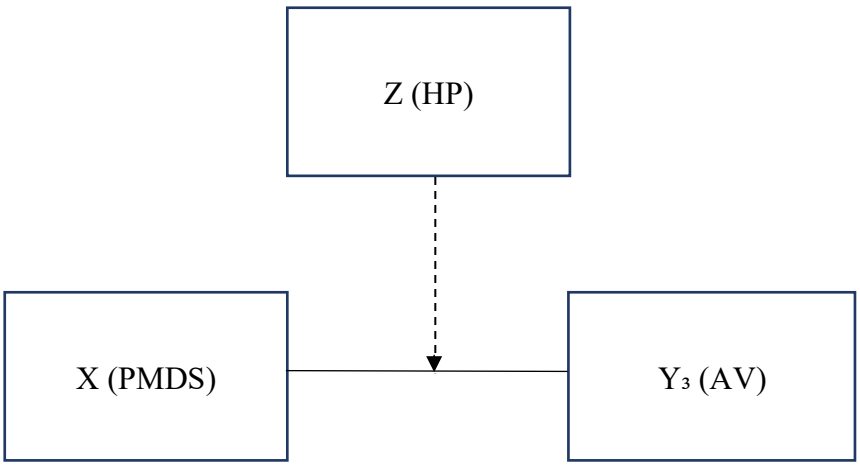
Conceptual framework of the association between prenatal depressive symptoms and externalizing symptoms with harsh parenting as moderator



Note. PMDS = Prenatal Maternal Depressive Symptoms. ES = Externalizing Symptoms. HP = Harsh Parenting.

Figure 3

Conceptual framework of the association between prenatal depressive symptoms and amygdala volume with harsh parenting as moderator



Note. PMDS = Prenatal Maternal Depressive Symptoms. AV = Amygdala Volume. HP = Harsh Parenting.

Method

Study Design

The current study is embedded within the Generation R study, a multiethnic prospective birth cohort in Rotterdam, the Netherlands (Hofman et al., 2004). This population-based study, which includes 9,901 pregnant women (Garcia et al., 2017), is designed to investigate the growth, development and health of urban children from fetal life to young adulthood (Jaddoe et al., 2006). The pregnant women were residents of Rotterdam and had an expected date of delivery between April 2002 and January 2006. The study was approved by the Medical Ethics Committee of the Erasmus Medical Center in Rotterdam (MEC-2012-165, NL40020.078.012). Written informed consent was obtained from parents.

Participants

For this study, participants were included based on data from structural brain scans and a mother-completed questionnaire on behavioral problems, both measured when the children were around the age of 10. Based on prior research in the Generation R cohort utilizing similar data (White et al., 2017), a study population of approximately 3534 children was anticipated. However, the number of participants decreased due to missing data, particularly on the harsh parenting measure, which was based on mothers' self-reports when the children were 3 years old. Of the children with this data, a subset of mothers also completed a questionnaire assessing prenatal depressive symptoms. In this study had 1,436 participants complete data on the main variables of interest.

Figure 3

Flowchart of Study Population

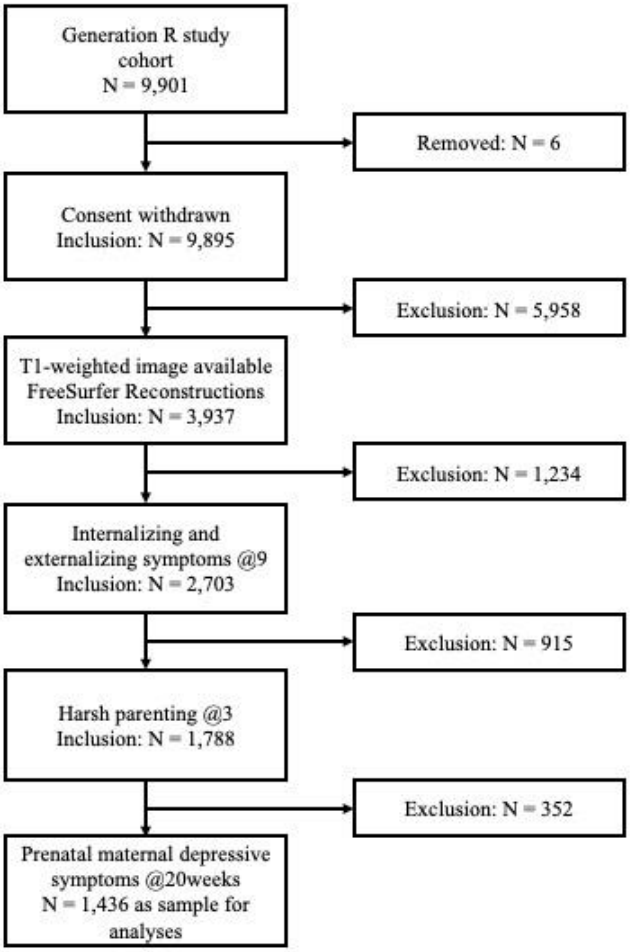


Table 1

Demographic Characteristics of Participants

Variables	*n*	*%*	*M*	*SD*	Min.	Max.
Child characteristics	1436					
Assigned sex at birth	1436					
Male	706	49.2				
Female	730	50.8				
Age (years)						
At CBCL	1430		9.7	0.24	8.9	11.8
Missing	6					
At MRI	1436		10.1	0.55	8.7	12.0
Variables	*n*	*%*	*M*	*SD*	Min.	Max.

Maternal characteristics	1436					
Age (years)	1436		31.9	4.02	17.9	46.3
Pre-pregnancy BMI	1202		23.2	3.74	16.0	45.4
Missing	234					
National origin	1434					
Dutch	1036	72.1				
Moroccan/Turkish	63	4.4				
Surinamese/Antillean	82	5.7				
Other*	253	17.6				
Missing	2	0.1				
Educational level	1421					
Low	35	2.4				
Intermediate	453	31.5				
High	933	65.0				
Missing	15	1.0				
Smoking during pregnancy	1321					
Never	1056	73.5				
Until pregnancy	127	8.8				
Continued	138	9.6				
Missing	115	8.0				
Alcohol use during pregnancy	1370					
Never	404	28.1				
Until pregnancy	200	13.9				
Continued (occassionally)	591	41.2				
Continued (frequently)	175	12.2				
Missing	66	4.6				

Note: *Asian, African, American and Oceanian. *M* = mean (for numeric variables) or percentage (for categorical variables); *SD* = Standard Deviation; *Min* = Minimum*; Max* = Maximum.

In this study, 1,436 children and their mothers participated, with data on various demographic characteristics of both children and mothers presented in Table 1. Of the children, 50.8% were female. The average age of the children was 9.7 years (*SD* = 0.24) at the time of completing the CBCL questionnaire and 10.1 years (*SD* = 0.55) during the MRI scan.

Regarding the mothers, the majority (72.1%) had a Dutch background. More than half of the mothers (65.0%) had a high level of education. Concerning substance use during pregnancy, 73.5% of the mothers reported not smoking, while 9.6% continued to smoke. Alcohol use during pregnancy was reported by some mothers, with 41.2% drinking occasionally and 12.2% frequently.

For comparison, the full dataset in Table A1 includes 9,895 children and mothers, with a similar distribution of boys (49.9%) and girls (48.6%). The children's ages at CBCL (M = 9.7, SD = 0.33) and MRI (M = 10.1, SD = 0.59) were also comparable, though with slightly greater variation. The proportion of mothers with a Dutch background was substantially lower in the full dataset (46.7%) compared to the smaller sample, while the groups of Moroccan/Turkish (14.3%) and Surinamese/Antillean (11.3%) descent were larger. Educational levels also differed markedly, only 37.5% of the mothers in the full dataset were highly educated, compared to 65.0% in the smaller sample. Additionally, 15.2% of mothers in the full dataset continued smoking during pregnancy, which is higher than in the smaller sample, while frequent alcohol use was lower (5.8%). This comparison indicates that the smaller sample included relatively more Dutch and highly educated mothers, who smoked less, suggesting a selective composition of this group within the broader cohort.

Measurement Instruments

Prenatal Maternal Depressive Symptoms

Prenatal maternal depressive symptoms were measured at 20 weeks of gestation using the Brief Symptom Inventory (BSI), a validated self-report questionnaire that assesses psychopathology over the past seven days (Cohen et al., 2023). The BSI consists of 53 items, rated on a 5-point Likert scale (1 = not at all, 2 = a little, 3 = quite a bit, 4 = quite a lot, 5 = continually). A sumscore was computed of the 6 items from the depression subscale, this scale shows strong agreement with the DSM-IV criterion (Derogatis & Melisaratos, 1983; El Marroun, 2014). In this study, the total score was used, which prevented the assessment of reliability within our own sample. However, previous literature indicates that the questionnaire has a Cronbach's alpha of .80 (Cohen et al., 2023; De Beurs & Zitman, 2006).

Internalizing and Externalizing Symptoms

The Child Behavior Checklist (CBCL) was used to assess internalizing and externalizing problems in children aged 9 to 11 years, with mothers completing the questionnaire (Achenbach & Rescorla, 2001; White et al., 2017). The 113 items are rated on a 3-point Likert scale (0 = not true, 1 = somewhat/sometimes true, 2 = often/very true) and are categorized into 'Internalizing' (41 items) and 'Externalizing' (43 items) problems. Higher

sumscores indicate more problems. The CBCL has been internationally validated with a Cronbach's alpha of .68 within this study.

Amygdala Volume

In this study, brain scans were performed on children aged 9 to 11 years using a Tesla 3 Magnetic Resonance Imaging (MRI) scanner (White et al., 2017). The T1-weighted images were analyzed using FreeSurfer software to measure brain structures, such as the amygdala volume. Of the 4087 children who were scanned, 3937 scans were successfully analyzed.

Harsh Parenting

Harsh parenting was measured when the child was 3 years old using an adapted version of the Parent-Child Conflict Tactics Scale (CTSPC; Straus et al., 1998). Mothers completed the questionnaire, which consisted of six items about disciplinary techniques used in the past two weeks, rated on a 6-point Likert scale. The techniques ranged from giving explanations to physical and verbal aggression. Three items on physical punishment (e.g., ‘‘I hit my child with something like a belt, stick or some other hard object’’) were omitted due to potential legal concerns. The total score for harsh parenting was calculated by summing the scores of the following six items: "In the past week/month, I angrily squeezed my child's arm", "I yelled at my child", "I scolded my child", "I threatened to slap my child but didn't", "'I called my child dumb or lazy or some other name like that", and "I shook my child". Higher scores indicate more severe harsh discipline (Jansen et al., 2012). The instrument demonstrates good reliability (Cronbach's alpha = .77; Straus et al., 1998).

Covariates

Several demographic variables were considered as potential factors that may influence the associations: sex of the child, age of the child at MRI and CBCL, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy (serving as a proxy for socioeconomic status; SES), and prenatal substance use, such as maternal smoking and alcohol use during pregnancy. All covariates were included in the analyses, except that in the analyses involving amygdala volume, the child's age at MRI was used instead of age at CBCL, and total intracranial volume derived from the child's MRI scans was additionally included as a covariate.

Statistical Analyses

All statistical analyses were conducted in R version 4.4.1. While 1,436 participants had complete data on the main variables of interest, missing values on covariates were handled in the analyses using Full Information Maximum Likelihood (FIML; Enders & Bandalos, 2001), which allowed the analyses to be performed on the dataset (N = 1437),

including covariates with missing data. Assumption checks and outlier detection were conducted on this dataset.

First, descriptive statistics were calculated for the main variables (prenatal depressive symptoms, internalizing symptoms, externalizing symptoms, and amygdala volume) and moderator (harsh parenting), which were treated as continuous variables in this study. Mean scores and standard deviations were computed for these variables. For the categorical covariates, sex of the child, maternal national origin, maternal educational level during pregnancy, and prenatal substance use, percentages were reported. For the continuous covariates, age of the child at MRI and at CBCL, maternal age at birth, and pre-pregnancy BMI, means and standard deviations were presented.

Secondly, bivariate correlations between the dependent variables (Y_1, Y_2, Y_3), the independent variable (X), the moderator variable (Z), and the covariates were examined using Spearman's correlation coefficient because of a non-normal distribution, to assess the strength and direction of associations. A significance level of .05 was applied.

Thirdly, prior to the moderation analyses, the direct associations between prenatal depressive symptoms (X) and the outcome variables (Y_1, Y_2, Y_3) were investigated. The sem() function in R was used to perform three linear regression analyses while adjusting for the covariates. Assumptions of linearity, multicollinearity, normality, homoscedasticity, and independence of observations were assessed. As several violations of the assumptions normality, linearity and homoscedasticity were observed, a robust Maximum Likelihood Estimator (MLR) was applied to account for these deviations and to produce more reliable parameter estimates. Regarding the assumption of multicollinearity, it was initially violated due to a Variance Inflation Factor (VIF) > 10 for the dummy variable representing educational level. By changing the reference category from 'low' to 'high' education, the VIF values decreased to approximately 1, indicating no multicollinearity.

Finally, moderation analyses were conducted to examine whether harsh parenting moderated the relationship between prenatal depressive symptoms and the outcome variables (internalizing symptoms, externalizing symptoms, and amygdala volume in children at age 10). The sem() function was again used to perform multiple linear regressions, controlling for the covariates. The same assumption checks were applied and due to observed deviations, the MLR estimator was consistently used. An interaction term (X*Z) between prenatal depressive symptoms (X) and harsh parenting (Z) was included for each outcome variable (Y_1, Y_2, Y_3) to test whether harsh parenting moderated the strength of the relationship between prenatal depressive symptoms and the outcome variables.

Given that multiple analyses were conducted on the same dataset, adjustments for multiple testing were applied. Multiple testing increases the risk of obtaining false-positive results (Type I error). To correct for this, the False Discovery Rate (FDR) method was used, as it is less conservative than the Bonferroni correction and better preserves statistical power (Benjamini & Hochberg, 1995).

Results

Descriptive Analyses

The mean scores, standard deviations, and ranges for the main variables and moderator are presented in Table 2. The sample consisted of 1,436 participants. The mean score for prenatal depressive symptoms was low (M = 0.13, SD = 0.32; De Beurs, 2004), ranged from 0 to 3.50. Internalizing symptoms had a mean of 4.50 (SD = 4.63), with scores ranging from 0 to 37.0. Externalizing symptoms had a mean of 3.54 (SD = 4.22), ranging from 0 to 25.0. The average amygdala volume was 3,568.77 mm^3 (SD = 370.57), with a minimum of 2,591.30 and a maximum of 5,064.80 mm^3. Harsh parenting was reported with a mean score of 1.73 (SD = 1.75), ranging from 0 to 12.0.

Table 2

Descriptive Statistics of Main Variables and Moderator

Variables	*M*	*SD*	Min.	Max.
Prenatal depressive symptoms	0.13	0.32	0	3.50
Internalizing symptoms	4.50	4.63	0	37.0
Externalizing symptoms	3.54	4.22	0	25.0
Amygdala volume	3568.77	370.57	2591.30	5064.80
Harsh parenting	1.73	1.75	0	12.0

Note: total N = 1436. M = mean; SD = Standard Deviation; *Min* = Minimum; *Max* = Maximum.

Bivariate Correlations

Table 3 presents the correlations between the main variables, the moderator, and relevant covariates. The strength of the Spearman correlations was interpreted using common guidelines, with values between 0.20–0.39 considered weak, 0.40–0.59 moderate, and 0.60 or higher strong. The results showed that prenatal depressive symptoms were positively and

significantly correlated with both internalizing symptoms (r_S = .12, p < .001) and externalizing symptoms (r_S = .08, p < .01). Additionally, prenatal depressive symptoms were similarly positively correlated with harsh parenting (r_S = .08, p < .01).

Internalizing and externalizing symptoms were moderately correlated with each other (r_S = .50, p < .001), suggesting that children who report more internalizing problems also tend to exhibit more externalizing behaviors. Harsh parenting was moderately positively related to externalizing symptoms (r_S = .16, p < .001) and showed a no association with internalizing symptoms (r_S = .00, p > .05).

Notably, amygdala volume did not show significant correlations with prenatal depressive symptoms, internalizing or externalizing symptoms, nor with harsh parenting.

Table 3

Correlations between Main Variables, Moderator and Covariates

Variables	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*
1. Assigned sex at birth child[a]	-														
2. Age child at CBCL	.00	-													
3. Age child at MRI	-.04	.40***	-												
4. Total intracranial volume	-.55***	.03	.08**	-											
5. Maternal age	-.03	-.04	-.02	.06*	-										
6. Pre-pregnancy BMI	.01	.01	.01	-.03	-.03	-									
7. Maternal national origin[b]	-.02	.06*	-.04	-.09***	-.07*	.01	-								

8. Maternal educational level[c]	.00	-.03	.02	.13***	.21***	-.16***	-.15***	-						
9. Prenatal maternal smoking	.03	.09**	.04	-.06*	-.02	.06*	.01	-.14***	-					
10. Prenatal maternal alcohol use	-.02	.03	.05*	.09***	.23***	-.07*	-.10***	.26***	.17***	-				
11. Prenatal depressive symptoms	-.03	.06*	.03	-.04	-.10***	.07*	.15***	-.09***	.10***	-.04	-			
12. Internalizing symptoms	.02	.01	.00	.00	-.08**	.03	.02	-.04	.01	-.04	.12***	-		
13. Externalizing symptoms	-.06*	.00	.00	.01	-.06*	.06*	.02	-.05	.04	-.02	.08**	.50***	-	
14. Amygdala volume	-.41***	.00	.03	.61***	.05*	.02	-.06*	.07*	-.04	.08**	.00	.00	.01	-

15. Harsh parenting	-.14***	.05	.06*	.04	-.02	.04	.02	-.03	.03	.03	.08**	.05	.16***	.02	-

Note: total $N = 1436$. * $p < .05$, ** $p < .01$, *** $p < .001$. Spearman's rho.

[a]Male = 0, Female = 1. [b]Dutch = 0, Moroccan/Turkish = 1, Surinamese/Antillean = 2, Other (Asian, African, American and Oceanian) = 3. [c]High = 0, Low = 1, Intermediate = 2.

Association between Prenatal Maternal Depressive Symptoms and Child Outcomes

Linear regression analyses were conducted to examine the direct association between prenatal maternal depressive symptoms and child internalizing and externalizing symptoms, as well as amygdala volume. The results are presented in Table 4.

In the adjusted model, which controlled for sex of the child, age at CBCL, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal education, and prenatal smoking and alcohol use, prenatal depressive symptoms were significantly associated with higher internalizing symptoms in children (b_{adj} = 0.117, 95% CI [0.043, 0.191], p_{FDR} = .002), indicating that this association remains significant after controlling for relevant covariates. Similarly, prenatal depressive symptoms were also significantly associated with higher externalizing symptoms in children (b_{adj} = 0.104, 95% CI [0.030, 0.178], p_{FDR} = .006). Based on the standardized coefficient, internalizing and externalizing symptoms can be compared, with internalizing symptoms showing a stronger association (β = .117, p_{FDR} = .002).

In contrast, there was no significant association between prenatal depressive symptoms and amygdala volume in either the unadjusted (b = –0.002, 95% CI [–0.049, 0.045], p_{FDR} = .820) or adjusted model (b_{adj} = 0.005, 95% CI [–0.038, 0.048], p_{FDR} = *.820*), which was controlled for sex of the child, age of the child at MRI, total intracranial volume, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy, maternal smoking and alcohol use during pregnancy.

Table 4

Linear Regression Analysis of the Association between Prenatal Maternal Depressive Symptoms and Child Internalizing and Externalizing Symptoms and Amygdala Volume

	B	*SE*	95% CI		β	*p*	p_{FDR}
			LL	*UL*			
Prenatal depressive symptoms							
			Child Internalizing Symptoms				
Unadjusted	.120	.038	.046	.194	.120	.002	.002
Adjusted*	.117	.038	.043	.191	.117	.002	.002
			Child Externalizing Symptoms				
Unadjusted	.107	.040	.030	.184	.107	.007	.006
Adjusted*	.104	.038	.030	.178	104	.006	.006

			Amygdala Volume				
Unadjusted	-.002	.024	-.049	.045	-.002	.943	.820
Adjusted**	.005	.022	-.038	.048	.005	.820	.820

Note: total $N = 1436$. *B* = unstandardized regression coefficient; *SE* = standard error; *CI* = confidence interval; *LL* = lower limit; *UL* = upper limit; β = standardized regression coefficient.

*Adjusted for: sex of the child, age of the child at CBCL, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy, maternal smoking and alcohol use during pregnancy.

**Adjusted for: sex of the child, age of the child at MRI, total intracranial volume, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy, maternal smoking and alcohol use during pregnancy.

Moderating Effect of Harsh Parenting on the Association between Prenatal Depressive Symptoms and Child Outcomes

To examine whether harsh parenting moderated the association between prenatal depressive symptoms and child outcomes (internalizing symptoms, externalizing symptoms, and amygdala volume), interaction terms were included in linear regression models. The results are presented in Table 5.

Child Internalizing Symptoms

In the adjusted model, controlling for sex of the child, age at CBCL, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal education, and prenatal smoking and alcohol use, prenatal depressive symptoms showed a significant main effect ($b = 0.105$, 95% CI [0.040, 0.170], $p_{FDR} = .004$), indicating that higher prenatal maternal depressive symptoms were associated with increased internalizing symptoms in children. Harsh parenting was not significantly associated with internalizing symptoms ($b = 0.034$, 95% CI [−0.021, 0.089], $p_{FDR} = .342$), and the interaction term between prenatal depressive symptoms and harsh parenting was also non-significant ($b = 0.020$, 95% CI [−0.042, 0.082], $p_{FDR} = .532$).

Child Externalizing Symptoms

For externalizing symptoms, both prenatal depressive symptoms ($b = 0.095$, 95% CI [0.022, 0.168], $p_{FDR} = .015$) and harsh parenting ($b = 0.145$, 95% CI [0.092, 0.198], $p_{FDR} < .001$) were significantly positively associated in the adjusted model. The interaction between

prenatal depressive symptoms and harsh parenting remained non-significant (b = −0.007, 95% CI [−0.062, 0.048], p_{FDR} = .811).

Amygdala Volume

No significant associations were found between prenatal depressive symptoms, harsh parenting, or their interaction and child amygdala volume in the adjusted model. Specifically, prenatal depressive symptoms (b = 0.007, 95% CI [−0.042, 0.056], p_{FDR} = .894), harsh parenting (b = −0.003, 95% CI [−0.050, 0.044], p_{FDR} = .894), and the interaction term (b = −0.003, 95% CI [−0.030, 0.024], p_{FDR} = .894) were all non-significant.

In summary, prenatal maternal depressive symptoms were significantly associated with increased internalizing and externalizing symptoms in children. Harsh parenting was independently associated with externalizing symptoms, but not with internalizing symptoms. No evidence was found for a moderating effect of harsh parenting on these associations. Additionally, neither prenatal depressive symptoms nor harsh parenting were significantly associated with amygdala volume.

Table 5

Moderation of by Harsh Parenting

	B	*SE*	95% CI		β	*p*	p_{FDR}
			LL	*UL*			
	Child Internalizing Symptoms						
Unadjusted							
Prenatal depressive symptoms	.108	.033	.043	.173	.108	.001	.004
Harsh parenting	.038	.028	-.017	.093	.038	.173	.342
Prenatal depressive symptoms x Harsh parenting	.018	.036	-.052	.088	.027	.606	.532
Adjusted*							
Prenatal depressive symptoms	.105	.033	.040	.170	.105	.001	.004
Harsh parenting	.034	.028	-.021	.089	.034	.228	.342
Prenatal depressive symptoms x Harsh parenting	.020	.032	-.042	.082	.030	.532	.532
	Child Externalizing Symptoms						
Unadjusted							
Prenatal depressive symptoms	.094	.038	.020	.168	.094	.012	.015

Harsh parenting	.157	.027	.104	.210	.157	<.001	<.001[e]
Prenatal depressive symptoms x Harsh parenting	-.011	.032	-.073	.051	-.017	.726	.811
Adjusted*							
Prenatal depressive symptoms	.095	.037	.022	.168	.095	.010	.015
Harsh parenting	.145	.027	.092	.198	.145	<.001	<.001[e]
Prenatal depressive symptoms x Harsh parenting	-.007	.028	-.062	.048	-.010	.811	.811
			Amygdala Volume				
Unadjusted							
Prenatal depressive symptoms	-.003	.027	-.056	.050	-.003	.905	.894
Harsh parenting	.046	.029	-.011	.103	.046	.118	.894
Prenatal depressive symptoms x Harsh parenting	-.008	.016	-.039	.023	-.012	.596	.894
Adjusted**							
Prenatal depressive symptoms	.007	.025	-.042	.056	.007	.791	.894
Harsh parenting	-.003	.024	-.050	.044	-.003	.894	.894
Prenatal depressive symptoms x Harsh parenting	-.003	.014	-.030	.024	-.005	.824	.894

Note: total $N = 1436$. *B* = unstandardized regression coefficient; *SE* = standard error; *CI* = confidence interval; *LL* = lower limit; *UL* = upper limit; β = standardized regression coefficient.

*Adjusted for: sex of the child, age of the child at CBCL, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy, maternal smoking and alcohol use during pregnancy.

**Adjusted for: sex of the child, age of the child at MRI, total intracranial volume, maternal age at birth, maternal national origin, pre-pregnancy BMI, maternal educational level during pregnancy, maternal smoking and alcohol use during pregnancy.

Discussion

This population-based study examined how prenatal depressive symptoms in mothers relate to internalizing and externalizing symptoms, as well as amygdala volume, in preadolescents. A key strength of this study is the inclusion of harsh parenting as a potential moderating factor in these associations, a relationship that, to our knowledge, has not yet been

investigated. This inclusion helps to address an important gap in the existing literature. Specifically, this study examined whether harsh parenting moderates the associations between prenatal maternal depressive symptoms and internalizing symptoms, externalizing symptoms, and amygdala volume in children. Based on previous literature, it was hypothesized that prenatal depressive symptoms would be associated with higher internalizing symptoms, higher externalizing symptoms, and larger amygdala volume, and that harsh parenting would strengthen all these associations.

The findings show that prenatal maternal depressive symptoms were significantly associated with higher levels of both internalizing and externalizing symptoms in preadolescents. Harsh parenting was also independently associated with more externalizing behavior. However, no significant interaction was found between prenatal depressive symptoms and harsh parenting for either internalizing or externalizing symptoms. This study found no evidence to suggest that harsh parenting strengthens the association between prenatal depressive symptoms and child problem behavior, contrary to our initial hypothesis.

The first hypothesis, that prenatal depressive symptoms are associated with higher internalizing symptoms in children, was supported and aligns with prior research indicating an increased risk of psychological problems in children exposed to prenatal depression (Goodman et al., 2011; Roos et al., 2022; Wen et al., 2017). This association fits within the developmental psychopathology concept of multifinality, which suggests that a single risk factor (such as maternal depression) can lead to various developmental outcomes (Cicchetti & Rogosch, 1996). It also supports the notion that prenatal depression affects not only maternal well-being but also the child's emotional development. Possible explanations include biological mechanisms such as elevated cortisol levels during pregnancy, which can influence the child's neurobiological development (Field et al., 2006).

The second hypothesis, that prenatal depressive symptoms are associated with higher externalizing symptoms in children, was also supported. This is consistent with previous findings and suggests that prenatal depression contributes to diverse child behavioral problems (Goodman et al., 2011; Roos et al., 2022). Furthermore, harsh parenting was independently associated with more externalizing behavior, which supports existing literature linking harsh parenting with aggression, impulsivity, and oppositional behavior in children (Pinquart, 2017; Hentges et al., 2021) and the fact that this association remains independent of prenatal depressive symptoms suggests that it represents a distinct pathway contributing to child problem behavior. This is in line with Patterson's coercion theory, which posits that harsh and inconsistent parenting can lead to negative, coercive parent–child interactions that

reinforce and maintain externalizing behavior (Smith et al., 2014). Contrary to earlier expectations, the present study found no significant relationship between harsh parenting and internalizing symptoms. This finding aligns with previous results by Mackenbach et al. (2014), which also showed that harsh parenting is primarily associated with behavioral problems (externalizing symptoms) and to a lesser extent with emotional problems. One possible explanation is that parents may be less capable of recognizing internalizing symptoms, which can lead to underreporting, or that children express these complaints less clearly. Furthermore, it was suggested that emotional problems in children are more strongly influenced by genetic factors than by parenting style, which could explain why harsh discipline appears to have less impact in this area (Mackenbach et al., 2014).

The third hypothesis, that prenatal depressive symptoms would be associated with increased amygdala volume in children, was not supported. No significant associations were found between prenatal depressive symptoms and amygdala volume. Additionally, neither harsh parenting nor its interaction with prenatal depressive symptoms showed any significant relationship with amygdala volume. These findings contrast with earlier studies that did report such associations (Wen et al., 2017; Donnici et al., 2021). One possible explanation is that the current sample, drawn from a general population, included lower levels of prenatal depressive symptoms compared to clinical samples with more severe maternal depression (Soe et al., 2017).

The fourth hypothesis, that harsh parenting would strengthen the associations between prenatal depressive symptoms and child outcomes, was not supported. Although it was expected that harsh parenting would strengthen the relationship between prenatal depressive symptoms and internalizing and externalizing symptoms (Field et al., 2006; Hentges et al., 2021), no evidence was found to support such an interaction. The absence of a moderation effect is notable, given prior theories such as Field et al.'s (2006) multivariate cumulative risk model, which emphasize the interaction between biological and social factors (Koyama et al., 2024). One possible explanation is that levels of harsh parenting in this study were relatively low (mean score of 1.73, ranging from 0 to 12.0), resulting in insufficient variation to detect a statistical interaction. Another explanation may be that harsh parenting does not act as a cumulative risk factor, but rather functions within the explanatory pathway as a mediator (Goodman et al., 2020).

Strengths and Limitations

A major strength of this study is the use of a large, population-based sample, which enhances the generalizability of the findings. Additionally, multiple sociodemographic

confounders were controlled for in the analyses, which strengthens the internal validity of the results. The use of structural equation modeling (SEM) and full information maximum likelihood (FIML) to handle missing data helps minimize selection bias.

Nevertheless, the study has several limitations that should be considered. First, the levels of depressive symptoms and harsh parenting in the sample were relatively low, which may have led to an underestimation of effects. Second, the data on internalizing and externalizing symptoms were reported by a single informant (the mother) who also reported on prenatal depressive symptoms. Mothers with higher levels of depressive symptoms may either overestimate or underestimate their children's problems, potentially introducing informant bias.

Furthermore, depressive symptoms during pregnancy were not clinically diagnosed using a structured diagnostic interview. Instead, the depression subscale of the Brief Symptom Inventory (BSI) was used. Although this questionnaire has been shown to be reliable and valid (Derogatis & Melisaratos, 1983), previous research within a subgroup of the Generation R cohort has shown that the BSI is a moderate indicator of depression diagnoses (El Marroun, 2014). This limits the clinical interpretation of the findings.

Another important consideration is the homogeneity of the sample. The mothers in this study were generally highly educated, which may limit the external validity of the findings. Additionally, social desirability bias may have influenced the self-reported measures of harsh parenting and depressive symptoms.

Lastly, selection bias must also be considered, as the original cohort may not fully represent the general population due to differential participation and attrition. Although Full Information Maximum Likelihood (FIML) was employed to handle missing data on covariates, this method does not fully account for selection processes related to key outcomes or exposure variables. Therefore, the potential impact of selection bias on the generalizability of results cannot be ruled out.

Conclusions, Implications and Future Research

This study confirms that prenatal depressive symptoms are associated with both internalizing and externalizing problems in preadolescence. Furthermore, harsh parenting was found to be related to externalizing symptoms. However, the expected moderating role of harsh parenting in the relationship between prenatal depressive symptoms and child internalizing and externalizing symptoms was not supported. Additionally, prenatal depressive symptoms were not found to be associated with amygdala volume. Nevertheless, these findings should be interpreted with caution given the methodological limitations.

These findings emphasize the importance of early identification and screening for depressive symptoms during pregnancy, which should be implemented in primary care. Since prenatal depressive symptoms are associated with both internalizing and externalizing problems in preadolescents, early detection and treatment of maternal depression may help reduce negative developmental outcomes. Additionally, these results support the inclusion of parenting as part of preventive interventions aimed at improving child developmental outcomes. Moreover, the link between prenatal depressive symptoms and internalizing and externalizing problems in preadolescents highlights the need for broad monitoring of child behavior. For clinical practice, this means that professionals should remain vigilant for a wide range of child behavioral problems, even when maternal depressive symptoms are mild.

To better understand causality and developmental pathways, future longitudinal research should incorporate repeated measures of both prenatal and postnatal factors over time. Future studies should also include multiple informants (e.g., partners, teachers, or self-reports from children) to reduce informant bias. Using clinical diagnostic tools rather than self-report questionnaires would enhance the clinical applicability of findings. Including partners in research on parental psychopathology is also essential to gain a fuller picture of the family context. Furthermore, research could focus on the underlying mechanisms of the intergenerational transmission of psychopathology, such as genetic vulnerability, parenting behavior, and the quality of parent-child interactions. This knowledge is crucial for developing targeted interventions that support both mother and child during vulnerable periods.

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Appendix

Bijlage A

Table A1

Participant Characteristics in Original Data

Variables	*n*	%	*M*	*SD*	Min.	Max.
Child characteristics	9895					
Assigned sex at birth						
Male	4935	49.9				
Female	4807	48.6				
Missing	153	1.5				
Age (years)						
At CBCL	5293		9.7	0.33	8.7	12.5
Missing	4602					
At MRI	3185		10.1	0.59	8.6	12.0
Missing	6710					
Variables	*n*	%	*M*	*SD*	Min.	Max.
Maternal characteristics	9895					
Age (years)	9895		29.9	5.37	15.3	46.3
Pre-pregnancy BMI	7201		23.7	4.36	14.4	50.2
Missing	2694					
National origin	9195					
Dutch	4618	46.7				
Moroccan/Turkish	5277	14.3				
Surinamese/Antillean		11.3				
Other*		20.7				
Missing	700	7.1				
Educational level	8658					
Low	968	9.8				
Intermediate	3977	40.2				
High	3713	37.5				
Missing	1237	12.5				

Smoking during pregnancy	8339	
Never	6124	61.9
Until pregnancy	715	7.2
Continued	1500	15.2
Missing	1556	15.7
Alcohol use during pregnancy	7994	
Never	3833	38.7
Until pregnancy	1059	10.7
Continued (occassionally)	2524	25.5
Continued (frequently)	578	5.8
Missing	1901	19.2

Note: *Asian, African, American and Oceanian. *M* = mean (for numeric variables) or percentage (for categorical variables); *SD* = Standard Deviation; *Min* = Minimum*; Max* = Maximum.

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BIJLAGE 4. PLAGIAATVERKLARING

Met ondertekening van deze overeenkomst verklaar ik Anna Vurun (557312) student Pedagogische Wetenschappen van de Erasmus Universiteit Rotterdam, dat ik bij het schrijven van mijn scriptie over het onderwerp Prenatal Maternal Depressive Symptoms, Child Problem Behaviors and Amygdala Volume: The Role of Harsh Parenting geen plagiaat heb gepleegd en dat de scriptie het resultaat is van mijn eigen werk en verwoord is in mijn eigen woorden, behoudens citaten. Daar waar mijn scriptie gebaseerd is op informatie dan wel ideeën van een ander zal ik die ander recht doen door naar diens geraadpleegde werk te verwijzen. Tevens verklaar ik dat ik te allen tijde verantwoordelijk blijf voor het bovenstaande.*

Datum, plaats:

03-07-2025, Schiedam

Handtekening: