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CHAPTER 6

Postpartum depression predicts offspring mental health problems in adolescence independently of parental lifetime psychopathology

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Abstract

Background

Postpartum depression (PPD) follows 5–15% of the life births and forms a major threat to the child's mental health and psychosocial development. However, the nature, continuance, and mediators of the association of postpartum depression (PPD) with the child's mental health are not well understood. The aim of this study was to investigate whether an association between PPD and adolescent mental problems is explained by parental psychopathology and whether the association shows specificity to the internalizing or externalizing domain.

Methods

2729 adolescents aged 10–15 years from the TRacking Adolescents' Individual Life Survey (TRAILS) were included. Both PPD and parental lifetime history of psychopathology were assessed by parent report. Adolescents' psychopathology was assessed using the Achenbach scales (parent, teacher and self-report). Linear regression was used to examine the association between PPD and adolescent mental health.

Results

We found a statistically significant association of adolescents' internalizing problems with maternal PPD, which remained when adjusted for parental psychopathology. We found no association for externalizing problems.

Conclusions

The association of PPD with internalizing but not externalizing problems extends into adolescence. Parental psychopathology does not explain this association suggesting a direct psychological effect on the child postpartum. If this effect appears causal, early treatment of parental psychopathology may prevent internalizing psychopathology in the offspring, ultimately in adolescence.

Background

Women frequently suffer from mood changes postpartum. When these escalate, a postpartum depression (PPD) may develop.¹⁴⁰ With its high frequency, i.e. following approximately 5–15% of live births, and its substantial negative effects, PPD is a considerable public health problem.¹⁴⁰⁻¹⁴⁵ Negative effects of PPD not only concern the affected women themselves, but also their offspring.^{29,146-153}

Earlier studies have shown that women who suffer from PPD express more negative emotions and are less sensitively attuned to their infants.³³ This may have specific consequences for the child, e.g. attachment insecurity,³⁴⁻³⁶ delay in emotional development,³⁷ social and interaction difficulties,³⁸ and an increased risk of developing violent behaviour.³⁹ Several psychological mechanisms underlying these consequences have been suggested including deficient mother–infant attachment,³⁴⁻³⁶ and temperamental alterations.¹⁵⁴

Although PPD is associated with mental health problems in childhood, it is largely unknown to what extent this association is causal in that the depression negatively affects the child during the postpartum period.¹⁵⁵ A non-causal association between PPD and mental health problems in the child may be produced in at least three ways. First, shared genetic liability to psychopathology between mother and child may cause an indirect non-causal association between PPD and the child's mental health. Second, PPD is associated with maternal¹⁴⁰ and possibly also with paternal¹⁵⁶ psychopathology after the postnatal period, both of which may threaten the child's mental health up to adolescence.⁴¹ Third, numerous studies have shown an association between anxiety, stress, or depression during pregnancy and childhood emotional and behavioural problems.¹⁵⁷ In addition to the causality issue, there is debate whether negative effects extend into adolescence and whether the effects regard predominantly internalizing or externalizing mental health problems.^{37,40-44}

The aim of this study was therefore to investigate whether the association between PPD and the child's psychopathology extends into adolescence, whether it is specific to the internalizing or externalizing domain of psychopathology and whether the association could be explained by parental psychopathology outside the postpartum period.

Methods

Sample

The TRacking Adolescents' Individual Lives Survey (TRAILS) is a prospective cohort study of Dutch (pre)adolescents, with the aim to chart and explain the development of mental health from preadolescence into adulthood, both at the level of psychopathology and the levels of underlying vulnerability and environmental risk.¹⁵⁸ The TRAILS study was approved by the Central Committee on Research Involving Human Subjects (Dutch CCMO). The present study involves data from the first (T1) and second (T2) assessment wave of the population-based cohort of TRAILS, which ran from, respectively, March 2001 to July 2002, and September 2003 to December 2004. In addition, data from the first (T1) and second (T2) assessment wave of the clinical cohort (TRAILS-CC) were used. These waves ran from September 2004 to December 2005, and September 2006 to November 2007.

Sample selection of the general population concerned five municipalities in the North of The Netherlands, including both urban and rural areas. They were requested to give names and addresses of all inhabitants born between 10-01-1989 and 09-30-1991. Of all children approached for enrolment in the general population cohort (i.e., selected by the municipalities and attending a school that was willing to participate, N=3145), 6.7% (N=211) were excluded because of mental or physical incapability or language problems. Of the remaining 2935 children, 74.5% (N=2188, mean age=11.09, SD=0.56, range 10.0–12.0, 50.8% girls) were enrolled in the study (i.e., both child and parent agreed to participate). Responders and non-responders did not differ with respect to the prevalence of teacher-rated problem behaviour, nor regarding associations between socio-demographic variables and mental health outcomes.¹⁵⁹

Of the 2230 baseline general-population participants, 96.4% (N=2149) participated in the first follow-up assessment (T2), which was held two to three years after T1. Mean age at T2 was 13.6 (SD=0.53, range=12.0–15.0).

Sample selection of TRAILS-CC is running in parallel with the TRAILS general population cohort. The cohort includes children who have been referred at least once to the child psychiatric outpatient clinic of the University Medical Center Groningen at any point in their life. Measurements and procedures in TRAILS-CC are identical to those in the general population cohort.¹⁵⁸ As expected, non-response in the clinical cohort was higher than in the population-based cohort. Of the 1264 children, who were eligible to enter the clinical cohort, 42.8% (N=541, mean age=11.1, SD=0.50, range 10.0–12.0, 34% girls) were enrolled in the study (i.e., both child and parent agreed to participate). There were again no significant differences between responders and non-responders in age, gender, level of

education of the parents and age at referral to the clinic. Also did these groups not differ on psychopathology subscales and language performance.¹⁵⁸

Of the 543 baseline participants in the clinical cohort, 85% (N=463) participated in the first follow-up assessment (T2), which was held two years after T1. Mean age at T2 was 12.87 (SD=0.62, range=12.0–15.0).

Measurements

At T1, well-trained interviewers (university graduates, who were extensively trained in interviewing skills, study background and interview content) visited one of the parents or guardians (preferably the mother, 95.6%) at their homes to administer an interview after a complete description of the study was given and written informed consent was obtained from participants. This interview covered a wide range of topics, including developmental history and somatic health, parental psychopathology (to control for recent episodes of depression) and care utilization. Besides the interview, the parent was asked to fill out a self-report questionnaire. Children in the general population cohort filled out questionnaires at school, in the classroom, under the supervision of one or more TRAILS assistants. In the clinical cohort the children visited the outpatient clinic of the University Medical Center Groningen to fill out the questionnaires. Teachers were asked to fill out a brief questionnaire for all TRAILS-children in their class. T2 involved only questionnaires, to be filled out by the children, their parents and their teachers. As in T1, the adolescents completed their questionnaires at school, respectively at the outpatient clinic. Measures that were used in the present study are described more extensively below.

Variables used

Both PPD and parental loading of psychopathology were assessed at baseline (T1) by parent report. The mother was asked whether she had suffered PPD in the first month postnatal, irrespective of having been treated for her condition. To allow adjustment for parental psychopathology outside the postpartum period, we calculated variables representing parental loading of psychopathology as follows. Lifetime parental psychopathology was assessed by means of the TRAILS Family History Interview (FHI).¹⁶⁰ The FHI assessed five dimensions of psychopathology: depression, anxiety, substance dependence, persistent antisocial behaviour, and psychosis. Each dimension was introduced by a vignette (available on request) describing the main DSM-IV characteristics of the dimension, followed by a series of questions assessing lifetime occurrence, professional treatment, and medication use. Information on both biological parents was obtained using a single informant (often the mother). For each dimension, we assigned each parent

to one of the following categories: 0=(probably) never had an episode, 1=(probably) yes, or 2=yes and treatment and/or medication. The prevalence rates in mothers and fathers respectively were: depression (27% and 15%), anxiety (16% and 6%), substance dependence (3% and 7%), and for antisocial behaviour (3% and 7%). We calculated parental loadings for the domains of internalizing and externalizing disorders separately. Both are effectively a count of the number of lifetime disorders within each domain reported by the biological parents. Parental loading on internalizing disorders included depression and anxiety and parental loading on externalizing disorders included substance dependence and persistent antisocial behaviour.¹⁶⁰ The empirical justification for the construction of the familial loadings is twofold (data available on request). First, disorders within each domain were more strongly correlated (on average 0.34) than disorders across domains (0.12), for mothers as well as fathers. Factor analysis of the disorder correlation matrix, for fathers and mothers separately, yielded two factors of internalizing and externalizing problems.¹⁶⁰ These were similar to the two-dimensional structure of common mental disorders.¹⁶¹ Secondly, the pattern of associations between parental disorders and offspring psychopathology was similar for fathers and mothers, suggesting that the paternal and maternal indices could be combined without obscuring relevant details. In line with this also is that paternal disorders were weakly correlated with maternal disorders. For instance, paternal and maternal depression were associated (0.18) and so were paternal and maternal antisocial behaviour (0.26).¹⁶⁰

At T1 and T2, the parents completed the Child Behaviour Checklist (CBCL).^{162,163} It contains a list of 120 behavioural and emotional problems, which could be rated as 0=not true, 1=somewhat/sometimes true, or 2=very/often true in the past 6 months. Adolescents filled out the self-report version of the CBCL, the Youth Self-Report (YSR)^{164,165} and teachers used the Teacher's Checklist of Psychopathology (TCP) based on the Teacher Report Form (TRF).¹⁶⁶ Internalizing and externalizing psychopathology in the adolescents was registered using the combined, equally weighted scores on the Achenbach scales from the children themselves, their parents and their teachers.¹⁶² Combining different sources reduces bias in the prediction of mental health problems and provides more accurate diagnoses than when a single source is used.¹⁶⁷

The following potential confounders were assessed by parent report: smoking (none, less than 1 cigarette per day, 1–10 per day, 11–20 per day, 1–2 packs per day, more than 2 packs per day)¹⁶⁸ and alcohol use during pregnancy (none, less than 1 unit per week, 1–3 units per week, 4–10 per week, 10–20 per week more than 20 per week), socio economic position (SEP) and obstetric factors. SEP included education, income and occupation of both parents, using the International Standard Classification of Occupations.¹⁶⁹ We created a SEP variable by standardizing and averaging

the items. Obstetric factors included in our analyses were gestational age, mode of delivery, birth weight and admission to neonatal care.

Statistical analyses

We calculated descriptive statistics for all variables. We created purified measures of psychopathology by partialling out shared variance between internalizing and externalizing problems. All variables, except for maternal PPD, were transformed to Z-scores. We averaged the scores of children's psychopathology obtained at T1 and T2 for reasons of statistical stability and our interest in the level of psychopathology rather than change over time. In addition, preliminary analyses showed no major differences between T1 and T2 scores (Cohen's d denoting effect size for all dimensions < 0.2). Subsequent analyses were performed using multiple linear regression. First, we assessed the unadjusted associations between PPD and both internalizing and externalizing problems in the adolescent offspring. Second, we adjusted these associations for dimension-specific parental loading and assessed the extent of decrease in the coefficient for PPD. These adjustments were made to investigate to what extent the association between PPD and psychopathology in the adolescent offspring could be explained by a parental history of psychopathology and with that the genetic transmission of mental health problems.

In addition, we performed analyses while adjusting for potential confounders, i.e., smoking and alcohol use during pregnancy, SEP and obstetric factors. Finally, we adjusted for cohort, i.e. general population or clinical cohort. The level of significance was set at 0.05, two-sided. Data were analysed using SPSS 16.

Results

Table 1 shows the descriptive statistics of the main variables we used in this study, divided by the presence or absence of maternal PPD. Seventy-five (2.7%) of the mothers of the adolescents reported a history of a maternal PPD. The demographic statistics show no difference between the children with and without maternal PPD. For both internalizing and externalizing pathology, the distribution was shifted towards the PPD group. This held for parental history as well as adolescent psychopathology.

Table 2 shows a significantly higher level of internalizing problems in adolescents with a history of maternal PPD compared to those without. When adjusted for parental loading the association reduced but remained substantial and statistically significant (difference in z-score: 0.18 (95% CI: 0.04–0.31)).

Table 3 shows no statistically significant association of maternal PPD with externalizing psychopathology in adolescents.

When we adjusted for the potential confounders smoking or drinking during pregnancy, SEP and obstetric factors the association of PPD with either internalizing or externalizing adolescent psychopathology did not materially change. Likewise, adjustment for cohort (general population vs. clinical cohort) did not affect the results.

Table 1: Descriptive statistics according to the presence of a history of maternal postpartum depression (PPD).

	PPD absent (N=2654)	PPD present (N=75)
Female gender, N (%)	1336 (50)	37 (49)
Age in years at baseline, mean (SD, range)	11.1 (0.55, 10.0-12.0)	11.1 (0.52, 10.0-12.0)
Internalising psychopathology*, mean (Z-score, SD)	0.4 (0.0, 0.6)	0.6 (0.3-0.7)
Externalising psychopathology*, mean (Z-scores, SD)	0.4 (0.0, 0.7)	0.5 (0.2, 0.7)
Parental lifetime history of internalising psychopathology, N (%)	1187 (45)	70 (93)
Parental lifetime history of externalising psychopathology, N (%)	432 (16)	18 (24)

* *Internalising and externalising psychopathology according to the Achenbach scales (mean of parent, teacher and self-report).*

Table 2: Internalising psychopathology* in offspring as predicted by maternal postpartum depression, unadjusted (2.1) and adjusted (2.2) for parental loading for internalising psychopathology.

	B (95% CI)	Standard error	t	p-value
2.1:				
Maternal postnatal depression	0.28 (0.14–0.41)	0.07	4.000	<0.001
2.2:				
Maternal postnatal depression	0.18 (0.04–0.31)	0.07	2.506	0.012
Parental loading for internalising problems	0.09 (0.07–0.11)	0.01	7.735	<0.001

* Shared variance with externalising problems was partialled out.

Table 3: Externalising psychopathology* in offspring as predicted by maternal postpartum depression, unadjusted (3.1) and adjusted (2.2) for parental loading for externalising psychopathology.

	B (95% CI)	Standard error	t	p-value
3.1				
Maternal postnatal depression	0.00 (-0.14–0.15)	0.07	0.051	0.960
3.2				
Maternal postnatal depression	0.00 (-0.14–0.15)	0.07	0.053	0.958
Parental loading for externalising problems	0.10 (0.07–0.12)	0.01	8.02	<0.001

* Shared variance with internalising problems was partialled out.

Discussion

Main findings

We investigated the link between PPD and offspring mental health problems in adolescence. For internalizing problems we showed that this relationship extends into adolescence and is only partially explained by parental loading. The relationship appeared to be specific for internalizing problems because there was no association with externalizing problems.

Strengths and limitations

A major asset of this study is that the analyses could be repeated while adjusting for lifetime parental psychopathology. This allowed us to investigate the hypothesis that PPD has a direct effect on the child, separate from psychopathology outside the postpartum period. Another major strength of this study is the use of widely validated composite Achenbach measures of psychopathology based on multiple informants (parent, teacher, child) causing information bias to be limited and reliability to increase.⁴¹ Finally, because of the inclusion of a large population based prospective sample and a high participation rate, our findings have high precision and generalizability.

The findings of this study should be interpreted in the light of three limitations. First, as we already mentioned, the parental loadings for psychopathology are rough approximations of genetic loading and include environmental risk for decreased mental health as well. Nevertheless, the relevance of this potential limitation is questionable as recent findings suggest that the genetic basis for the intergenerational transmission of depression is absent.^{170,171} Second, we did not interview each biological parent in person but interviewed only one parent directly and used this parent as informant for the other parent. Therefore, we assume there is underreporting of lifetime parental psychopathology. Third, it was not registered which adolescents belonged to the same family, so a possible effect related to the rank of the child could not be studied.

Interpretation

Earlier studies have indicated that PPD may increase the risk of emotional and behavioural problems in early childhood.^{33-37,150,153,156} However, there is debate whether negative effects of PPD are lasting and extend into adolescence and what these effects are.^{37,40-44} A commonly recognized limitation was the absence of correction for parental lifetime history of psychopathology, thus leaving the possibility that the association is due to liability shared by mother and offspring.⁴¹ In our study we have overcome

these limitations by correcting for dimension-specific parental loading for psychopathology. Parental loading constitutes a rough approximation of genetic loading and includes environmental risk for decreased mental health, e.g. demographic and socio-economic factors.¹⁶⁰ The observation that in our study the association between PPD and internalizing problems held up after adjustment for maternal internalizing psychopathology outside the postpartum period pleads for a direct psychological effect of PPD. Such a direct psychological effect may be the result of impaired mother–child interaction which has shown to lead to suboptimal attachment.^{33,149-153} Alternative explanations of the association of PPD with mental health problems in the offspring include neglect or even abuse of the child¹⁷² or reduced frequency of breastfeeding.¹⁷³

We found no association between PPD and offspring externalizing problems, which does not agree with findings by Fihrer et al. who observed an increased risk for both internalizing and externalizing problems¹⁵⁰ and by Hay and colleagues who reported on an association between PPD and violent behaviour in offspring.³⁹ This might be explained by the fact that the latter study assessed overt seriously violent behaviour according to DSM-IV criteria only. In contrast, we measured the entire spectrum of externalizing psychopathology as it exists in the general population for which an association with PPD may be weaker. In addition, our findings of raised rates of internalizing problems and no raised rates of externalizing problems are entirely consistent with the recent study of Murray et al.⁴⁴

In our study we found that 2.7% of the mothers had a history of PPD. Recent studies estimated a PPD prevalence of 5–15% of all live births.^{141,143-145} We have three possible explanations for this difference. First, in our questionnaire we defined PPD as a depression with an onset within one month after childbirth, while some other studies used an onset within one year. Second, the level of parental education was higher in our study than in the general population,¹⁵⁸ which may explain the lower than expected frequency of PPD. Third, because PPD is retrospectively assessed at T1, its recall may have been incomplete. Nevertheless, in accordance with Hardt and Rutter who stated that retrospectively assessed adverse experiences involve false negatives but rarely false positives,¹⁷⁴ in our study it is quite likely that those women who did report PPD actually had suffered PPD while an unknown number of women who actually suffered PPD did not report it. Consequently, the association between maternal PPD and psychopathology may have been diluted by non-differential determinant misclassification, i.e. recall of PPD was likely independent of the presence of the adolescents' mental health problems. Therefore, the real association may be stronger than we observed.

Conclusion

The association between PPD and psychopathology in the offspring extends into adolescence, is limited to internalizing problems, and is only partially explained by parental lifetime psychopathology. Therefore, mediation of this association may be largely caused by a direct psychological effect on the child in the postpartum period, e.g. as a result of impaired mother–child attachment.

Early screening for and treatment of maternal PPD may decrease the symptoms of PPD¹⁷⁵⁻¹⁷⁷ and, if the association appears causal, may thereby prevent internalizing psychopathology in the offspring, ultimately in adolescence.¹⁷⁸ In addition, because early management of psychopathology in adolescence may reduce symptoms,^{179,180} the offspring of mothers with a history of PPD could be monitored more closely for internalizing problems in early adolescence.

PART II

Randomised controlled trial



