Parental depressive symptoms as a risk factor for child depressive symptoms; testing the social mediators in internationally adopted children

Liskola, Krista

2018-12


http://hdl.handle.net/10138/276971
https://doi.org/10.1007/s00787-018-1154-8

Downloaded from Helda, University of Helsinki institutional repository.

This is an electronic reprint of the original article.

This reprint may differ from the original in pagination and typographic detail.

Please cite the original version.
Parental depressive symptoms as a risk factor for child depressive symptoms; testing the social mediators in internationally adopted children

Krista Liskola1 · Hanna Raaska1 · Helena Lapinleimu2 · Marko Elovainio3,4

Received: 18 September 2017 / Accepted: 5 April 2018 / Published online: 12 April 2018
© Springer-Verlag GmbH Germany, part of Springer Nature 2018

Abstract
Parental depressive symptoms have shown to be associated with offspring depression but much of the research has been focused on maternal depression. The aim of our study was to investigate the extent to which depressive symptoms of both parents associate with offspring depressive symptoms and whether social factors mediate these associations using data from adopted children with no shared genetic background. Data were derived from the Finnish Adoption survey study (a subsample of adopted children aged between 9 and 12 years, n = 548). Parental depressive symptoms were measured using short version of the General Health Questionnaire and Children’s Depression Inventory (CDI) was used to measure depressive symptoms in adoptees. Paternal depressive symptoms were related to the total CDI (B = 0.33, p = 0.05) and two dimensions of offspring depressive symptoms: negative mood (B = 0.10, p = 0.03) and interpersonal problems (B = 0.06, p = 0.009). These associations remained significant even when adjusted for child’s age and gender, age at adoption, type of placement before adoption, continent of birth and adoptive family’s SES. No associations were found between maternal and any dimensions of offspring depressive symptoms. No information about the mental health of biological parents was available. We interpret the results as demonstrating that intergenerational transmission of depressive symptoms is not solely related to shared genes. Also, the results highlight the association of paternal depression with offspring depressive symptoms.

Keywords Depressive symptoms · CDI · Adoption · Intergenerational transmission

Introduction
Depression is a common and complex problem with potentially serious economic consequences for affected individuals as well as public healthcare systems. Epidemiological studies show that depressive disorders are diagnosed across all world regions and occur also in children [10, 15, 16]. Previous global burden of disease (GBD) studies in 1990 [37] and 2000 [36, 60] shifted international focus towards depressive disorders as a leading cause of burden in its own right and also in comparison to more recognized physical disorders. According to previous studies, a family history of depression is an important predictor of emotional and behavioral problems in children [51].

Family history of mood disorders and stressful life events are both established risk factors for childhood depression. Children whose parents have mental illness face an elevated risk of psychopathology. This is due to an increased likelihood of carrying a genetic predisposition to depression and the influence of parental mental illnesses on their environment [34, 40]. Already in [42] discussed the association known as passive gene–environment correlation [42]. This means that a depressed parent may not only pass down the genotype associated with depression but may also create a rearing environment that is influenced by the parent’s own heritable characteristics, including depression. So far there is not a lot of direct evidence on the environmental mechanisms mediating the relationship, but modeling, disengagement, negative parenting behaviors, and increased exposure...
to adverse life events, such as family disruption, count as potential mediators [11, 18, 19, 46]. Findings outline the key role of exposure to early stressful life events as a mediator of familial mood disorder risk in preschool onset depression. These findings in a preschool sample provide support for the hypothesis that psychosocial factors may have increased importance as mediators of risk in younger age groups [29]. A meta-analysis on the Children’s Depression Inventory (CDI) conducted in the USA [56] showed no differences in depression across socioeconomic status (SES) of children, although other studies do indicate that a low SES is correlated with a greater prevalence of depression [5].

The majority of investigations in this area have focused on maternal depression and results regarding paternal depression are more mixed [32, 34, 51, 52, 55]. Many researchers have found that paternal depression was associated with concurrent internalizing problems in children of different ages [12, 24, 31, 41]. In a recent study, paternal depressive symptoms were shown to increase the risk of self-reported depressive symptoms in adolescents without any association to maternal depressive symptoms [28]. Furthermore, in one longitudinal study paternal depressive symptoms at age 3, when compared to other paternal characteristics, were the strongest predictor of children’s internalizing problems 3 years later [21]. In many cases, the degree of risk associated with paternal psychopathology is found to be comparable to that associated with maternal psychopathology. However, several researchers have found no association between paternal depression and internalizing problems in children [13, 20, 30], and others have suggested that this association might be accounted for, at least in part, by maternal depression [47].

Past studies have covered the fact that the gender of the child may modify the relationship between parental and offspring depression and views diverge on whether girls or boys are more affected and whether the size of the effect is different for maternal and paternal depression. Most of the research focuses on maternal depression and the results are varying; some argue that girls are more vulnerable to maternal depression than boys [8], others conclude the opposite, especially if the exposure occurred at a young age [9, 14], and some find no significant moderation at all [7]. The scarce evidence on paternal depression implies that boys might be more vulnerable to its effects, especially during early development [46, 47].

Studies have also shown that maternal and paternal depression may have an additive effect on youth psychopathology, implying that the psychopathology of fathers may additionally increase the risk for psychopathology in children when mothers suffer from mental health problems [19, 58]. Furthermore, Laurent et al. [27] found that the double dose of parental depressive symptoms from both adoptive fathers and adoptive mothers was related to lower cortisol levels in the child, which then was linked to higher levels of child internalizing problems.

Most research on the risk for psychopathology in children of depressed parents focuses on biological offspring. As suggested by [40], in such data the isolation of the environmental aspects of the association between parent and child functioning is complicated by the challenges of partitioning genetic from environmental influences. Nevertheless, as implied by Natsuaki et al. there are specific study designs that can tease apart these effects. These designs include (a) adoption studies of parents rearing children to whom they are genetically unrelated and (b) children of twins studies [40].

A number of studies have shown that internationally adopted children have more internalizing symptoms (e.g., symptoms of anxiety and depression) than their non-adopted peers [23]. There are less studies regarding adopted children’s depressive symptoms per se but in a recent Norwegian population-based study 45 internationally adopted adolescents aged from 16 to 19 years reported more depressive symptoms than their non-adopted peers ($n = 10,175$) [2]. Institutional rearing before adoption may increase risk for later anxiety and depressive symptoms (e.g., [6] and female gender may serve as a protective factor for these children with institutional background [6]. However, gender has not been associated with adopted child’s internalizing symptoms in other groups [23]. Age at adoption itself may not increase risk for emotional problems [3] but children with an older adoption age may have been exposed to adverse experiences for a longer time and that may increase their risk for depressive symptoms.

In this study, we investigated mechanisms of intergenerational transmission of depressive symptoms among internationally adopted children and their parents. Thus, we were able to provide a direct test of the extent to which there is only an environmental effect of parental depressive symptoms (that is, separate from genetic influences). Passive gene–environment correlation is presumed to be removed when genetically unrelated parents provide the rearing environment for a child [50]. Furthermore, in previous studies depressive symptoms of two generations are often measured using only one informant (e.g., [35]) and thus suffer from obvious bias. In our study, both parental and offspring reports of depressive symptoms were used; the adopted children filled in the CDI and the parents filled in a five-item GHQ.

**Methods**

**Participants**

This study is part of the ongoing FINnish ADOption (FinAdo) study [44, 45]. The target population of the study
consists of all children internationally adopted through three legalized adoption organizations in Finland between 1985 and 2007. The study was approved by the Ethics committee of the Hospital District of Southwest Finland and written and informed consent was obtained from the parents and the children themselves. Data were gathered with questionnaires exploring data about the child, the adoptive family and the parents themselves. No prenatal information about the children and the life-events they experienced (except for the type and number of placements) before the moment they were adopted was available. The questionnaires were filled in separately by the parents and those adoptees over 9 years of age. Surveys were conducted in December 2007, January 2008 and March 2009 and included two mailings for non-respondents. The study sample of 0–18-year-old included 1450 internationally adopted children. 634 (44%) were boys and 803 girls with a mean age of 7.5 years (SD 4.4) at the time of study entry. The participation rate was 55.7% and older children were more likely to be non-responders, OR 1.06, 95% CI [1.04, 1.08], p < 0.001 [45]. There were no differences in the country of origin between responders and non-responders. In this study, a subsample of children aged between 9 and 12 (n = 548) was used. Response rate in this age group was 46.5% (n = 255). Adequate information (parental GHQ and CDI filled in by the child) was provided by 242 of the participants and they formed the final sample.

**Measures**

**Child-related background factors**

A questionnaire developed for the FinAdo study was used to gain knowledge about the characteristics of the child before and after adoption. The child-related variables included the child’s gender, age at the time of adoption and at the time of responding to the questionnaire, country of birth, the type and number of pre-adoption placements and health history.

**Depressive symptoms in adoptees**

The Children’s Depression Inventory [26] was used to measure the depressive symptoms of the adoptees. The CDI is a 27-item scale that is self-rated and symptom-oriented and it rates the severity of symptoms related to depression or dysthyemic disorder in children and adolescents (7–17). The 27 items on the assessment are grouped into five dimensions which are (a) negative mood, (b) interpersonal problems, (c) ineffectiveness, (d) anhedonia and (e) negative self-esteem. The participants rated themselves based on how they feel and think, with each statement being identified with a rating from 0 to 2, 0 indicating the absence of symptoms, 1 the mild symptoms, and 2 the definite symptoms. The total score ranges from 0 to 54.

The CDI has established good reliability and validity for describing depressive symptoms and has good correlation with other scales [53, 447–463]. In our sample, the Cronbach alpha value for the CDI sum score was 0.84 and 0.57 for the subscale negative mood, 0.24 for interpersonal problems, 0.47 for ineffectiveness, 0.65 for anhedonia and 0.63 for negative self-esteem, respectively.

**Parental depressive symptoms**

The General Health Questionnaire (GHQ) is a self-administered screening questionnaire, designed for use in consulting settings aimed at detecting individuals with a diagnosable psychiatric disorder [17, 139–145]. The 12-Item General Health Questionnaire (GHQ-12) is the most extensively used screening instrument for common mental disorders, in addition to being a more general measure of psychiatric well-being and various versions of the GHQ-12 have been reported to be useful in determining the presence of depression. However, also shorter (five item) versions have shown good predictive validity [1]. In this study, we used a five-item questionnaire requesting whether the parent had recently been able to enjoy his/her daily duties, been thinking of himself/herself as a worthless person, felt unhappy and depressed, lost his/her self-confidence, or felt quite happy. The questions were answered on a 4-point scale: 1 = more than usual, 2 = as much as usual, 3 = less than usual, 4 = much less than usual. The first and last items were reverse coded and all items were summed. In our sample, the Cronbach alphas for the five-item GHQ were 0.76 for mother’s depressive symptoms, and 0.80 for father’s depressive symptoms.

**Statistical analyses**

The associations between parental depression (separately for mother and father) and the dimensions of offspring depressive symptoms were tested using linear regression analyses. A stepwise adjustment was performed. In step one, the results were adjusted for child’s age and gender, in step two for age at adoption, type of placement before adoption, continent of birth, in step three for adoptive family’s socioeconomic status and in the fourth and final step for all the above-mentioned variates. Child’s age and age at adoption were analyzed as continuous variables, child’s birth country was classified according to the continent of birth (Asia, Europe, America, and Africa), type of placement before adoption was classified into three classes (orphanage, foster home, and multiple placements), and based on education adoptive family’s socioeconomic status was classified according to the Official Statistics of Finland (upper middle class, lower middle class, working class, and other). In addition, we tested accordingly the associations between
each parents’ and child’s depressive symptoms in boys and girls separately.

To analyze the effects of maternal and parental depressive symptoms we tested for interaction effects between mothers’ and fathers’ depression. The effects were tested for child’s CDI sum score and the five subscales separately.

The associations were presented as standardized regression coefficients ($\beta$), standard errors (SE) and coefficients of determination ($R^2$). Statistical analyses were conducted using SAS for Windows (version 9.2), with $p$ values below 0.05 considered to be statistically significant.

**Results**

The characteristics of the sample are shown in Table 1. Girls represented 51.6% of the children. Most of the children were adopted from Asia (42.15%) and most of them had been placed in an orphanage before adoption (55%). The mean age at arrival in Finland was 2.74 years and mean age at time of estimation (depressive symptoms measure) was 10.5 years. The families into which the children were adopted were mainly of upper middle class (62.7%) and lower middle class (16.7%).

Correlations among study variables are shown in Table 2. The child’s CDI subscales were correlated with each other and with CDI sum score. The father’s and mother’s GHQ scores were correlated with each other and the paternal GHQ score was correlated with offspring’s CDI sum score and subscale of interpersonal problems. Maternal GHQ score was not correlated with child’s CDI sum score but a correlation emerged with subscale of ineffectiveness. Higher parental SES was correlated with offspring’s younger age.

Table 3 shows the association between parent’s and child’s depressive symptoms. Paternal depressive symptoms were related to offspring’s total depressive symptoms ($B = 0.33, p = 0.05$) and negative mood ($B = 0.10, p = 0.03$) and interpersonal problems ($B = 0.06, p = 0.009$).

These associations remained significant, even when adjusted for child’s age and gender, type of placement before adoption, continent of birth and adoptive family’s SES. No associations were found between maternal and any dimensions of offspring depressive symptoms. Maternal and paternal psychopathology combined were associated with offspring’s interpersonal problems.

Of the potential confounders, only the country of origin (from Asia compared to Europe) was significantly associated with depression ($p = 0.002$ for paternal and maternal depressive symptoms, respectively) in the fully adjusted models.

In the analyses in which only girls were included, we found that mother’s depressive symptoms were not associated with girls’ CDI sum score. However, we found an association between mother’s depressive symptoms and girls’ interpersonal problems ($B = 0.07, SE = 0.03, p = 0.03, R^2 = 0.04$). Accordingly, father’s depressive symptoms were not associated with girls’ CDI sum score but an association emerged with girls’ interpersonal problems ($B = 0.10, SE = 0.03, p = 0.002, R^2 = 0.09$). The associations remained statistically significant after adjustments and in addition, girl’s negative mood was associated with father’s depressive symptoms ($B = 0.17, SE = 0.07, p = 0.02, R^2 = 0.14$) when all the factors were added into the model. Boys’ depressive symptoms were not associated either with mother’s or father’s depressive symptoms.

Furthermore, we tested for interaction effects between mothers’ and fathers’ depression to find out if there was any additive effect of parental depression or maternal protective factor when paternal depression was present. The results for the interaction were not significant with the range of $p$ values varying between 0.26 and 0.85 and the $R^2$ values ranging from 0.02 to 0.03.

**Discussion**

Our study showed that in adoptive families, paternal depressive symptoms were associated with the increased risk of offspring depressive symptoms including negative mood and interpersonal problems. These associations were not
Table 2  Pearson and Spearman correlation coefficients and number of observations

<table>
<thead>
<tr>
<th>Variable</th>
<th>gender</th>
<th>Gender</th>
<th>Age</th>
<th>SES</th>
<th>GHQ mother</th>
<th>GHQ5 father</th>
<th>CDIs</th>
<th>Negative mood</th>
<th>Interpersonal problems</th>
<th>Negative self-esteem</th>
<th>Anhedonia</th>
<th>Ineffectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.04</td>
<td>–</td>
<td>242</td>
<td></td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td>0.02</td>
<td>–0.13*</td>
<td></td>
<td></td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GHQ5 mother</td>
<td>–0.02</td>
<td>–0.03</td>
<td>–0.03</td>
<td></td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GHQ5 father</td>
<td>–0.11</td>
<td>0.06</td>
<td>0.03</td>
<td>0.31**</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDIs</td>
<td>0.01</td>
<td>–0.02</td>
<td>0.03</td>
<td>0.05</td>
<td>0.14*</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative mood</td>
<td>–0.10</td>
<td>0.04</td>
<td>–0.11</td>
<td>0.03</td>
<td>0.15*</td>
<td>0.77**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal problems</td>
<td>0.11</td>
<td>–0.01</td>
<td>0.04</td>
<td>0.11</td>
<td>0.18**</td>
<td>0.75**</td>
<td>0.52**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative self-esteem</td>
<td>–0.11</td>
<td>0.02</td>
<td>0.02</td>
<td>0.04</td>
<td>0.11</td>
<td>0.78**</td>
<td>0.50**</td>
<td>0.57**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anhedonia</td>
<td>0.07</td>
<td>–0.07</td>
<td>0.00</td>
<td>0.03</td>
<td>0.03</td>
<td>0.85**</td>
<td>0.53**</td>
<td>0.51**</td>
<td>0.52**</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ineffectiveness</td>
<td>0.15*</td>
<td>0.02</td>
<td>0.04</td>
<td>0.15*</td>
<td>0.12</td>
<td>0.75**</td>
<td>0.44**</td>
<td>0.54**</td>
<td>0.50**</td>
<td>0.51**</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>

*aSpearman correlation coefficients, all other variables are Pearson correlation coefficients

*p < 0.05, **p < 0.01
explained by multiple potential confounders, including parental socioeconomic position or childhood risks before adoption and mother's depressive symptoms did not moderate the association. Similar associations could not be found between maternal depressive symptoms and offspring psychopathology. The association with paternal depressive symptoms emerged with girls' symptoms in particular.

The results of our study raise two important issues. First, it underlines the role of social and psychological environment in the intergenerational transmission of depressive

Table 3: Associations between depressive symptoms among parents (GHQ5) and adopted children (CDI)

<table>
<thead>
<tr>
<th>Parental depressive symptoms</th>
<th>Depressive symptoms in adopted children</th>
<th>Total CDI</th>
<th>Negative mood</th>
<th>Interpersonal problems</th>
<th>Ineffectiveness</th>
<th>Anhedonia</th>
<th>Negative self-esteem</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>B (SE)</td>
<td>β (SE)</td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td>0.14 (0.2)</td>
<td>0.02 (0.1)</td>
<td>0.04 (0.0)</td>
<td>0.07 (0.0)</td>
<td>0.02 (0.1)</td>
<td>0.03 (0.1)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.02</td>
<td>0.00</td>
<td>0.01</td>
<td>0.01</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Father</td>
<td></td>
<td>0.33* (0.2)</td>
<td>0.10* (0.1)</td>
<td>0.06** (0.0)</td>
<td>0.10** (0.1)</td>
<td>0.02 (1.1)</td>
<td>0.07 (1.1)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.02</td>
<td>0.02</td>
<td>0.03</td>
<td>0.02</td>
<td>0.00</td>
<td>0.01</td>
</tr>
<tr>
<td>Parents’ average depressive symptoms</td>
<td></td>
<td>0.29 (0.2)</td>
<td>0.08 (0.19)</td>
<td>0.07** (0.0)</td>
<td>0.07 (0.0)</td>
<td>0.008 (0.1)</td>
<td>0.06 (0.1)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.01</td>
<td>0.01</td>
<td>0.03</td>
<td>0.02</td>
<td>0.00</td>
<td>0.01</td>
</tr>
<tr>
<td>Step 1: adjusted with child’s age and gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td>0.14 (0.17)</td>
<td>0.01 (0.05)</td>
<td>0.04 (0.02)</td>
<td>0.08* (0.04)</td>
<td>0.03 (0.07)</td>
<td>0.02 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.00</td>
<td>0.01</td>
<td>0.02</td>
<td>0.04</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Father</td>
<td></td>
<td>0.35* (0.17)</td>
<td>0.09* (0.05)</td>
<td>0.07** (0.02)</td>
<td>0.11* (0.04)</td>
<td>0.03 (0.06)</td>
<td>0.06 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.02</td>
<td>0.02</td>
<td>0.05</td>
<td>0.04</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Parents’ average depressive symptoms</td>
<td></td>
<td>0.29 (0.20)</td>
<td>0.07 (0.06)</td>
<td>0.08** (0.03)</td>
<td>0.06 (0.04)</td>
<td>0.017 (0.07)</td>
<td>0.05 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.01</td>
<td>0.02</td>
<td>0.05</td>
<td>0.05</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Step 2: adjusted with age at adoption, type of placement before adoption, continent of birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td>0.13 (0.17)</td>
<td>0.02 (0.05)</td>
<td>0.04 (0.02)</td>
<td>0.07* (0.04)</td>
<td>0.017 (0.06)</td>
<td>0.02 (0.04)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.07</td>
<td>0.03</td>
<td>0.08</td>
<td>0.09</td>
<td>0.07</td>
<td>0.05</td>
</tr>
<tr>
<td>Father</td>
<td></td>
<td>0.38* (0.17)</td>
<td>0.11** (0.05)</td>
<td>0.07** (0.02)</td>
<td>0.07* (0.04)</td>
<td>0.04 (0.06)</td>
<td>0.07 (0.04)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.08</td>
<td>0.06</td>
<td>0.09</td>
<td>0.07</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>Parents’ average depressive symptoms</td>
<td></td>
<td>0.32 (0.20)</td>
<td>0.09 (0.05)</td>
<td>0.08** (0.02)</td>
<td>0.10* (0.04)</td>
<td>0.02 (0.07)</td>
<td>0.07 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.08</td>
<td>0.04</td>
<td>0.09</td>
<td>0.09</td>
<td>0.07</td>
<td>0.06</td>
</tr>
<tr>
<td>Step 3: adjusted with adoptive family’s SES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td>0.13 (0.17)</td>
<td>0.02 (0.05)</td>
<td>0.03 (0.24)</td>
<td>0.07 (0.04)</td>
<td>0.02 (0.07)</td>
<td>0.02 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.01</td>
<td>0.03</td>
<td>0.02</td>
<td>0.03</td>
<td>0.00</td>
<td>0.02</td>
</tr>
<tr>
<td>Father</td>
<td></td>
<td>0.30 (0.17)</td>
<td>0.11* (0.05)</td>
<td>0.05* (0.02)</td>
<td>0.06 (0.04)</td>
<td>0.20 (0.06)</td>
<td>0.05 (0.04)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.03</td>
<td>0.05</td>
<td>0.06</td>
<td>0.05</td>
<td>0.00</td>
<td>0.04</td>
</tr>
<tr>
<td>Parents’ average depressive symptoms</td>
<td></td>
<td>0.27 (0.20)</td>
<td>0.09 (0.06)</td>
<td>0.07** (0.03)</td>
<td>0.09* (0.05)</td>
<td>0.003 (0.08)</td>
<td>0.05 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.01</td>
<td>0.03</td>
<td>0.04</td>
<td>0.03</td>
<td>0.00</td>
<td>0.02</td>
</tr>
<tr>
<td>Step 4: adjusted with child’s age, gender, age at adoption, type of placement before adoption, continent of birth and adoptive family’s SES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td>0.11 (0.17)</td>
<td>0.02 (0.05)</td>
<td>0.04 (0.02)</td>
<td>0.07 (0.04)</td>
<td>0.16 (0.06)</td>
<td>0.01 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.08</td>
<td>0.07</td>
<td>0.09</td>
<td>0.10</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Father</td>
<td></td>
<td>0.37* (0.18)</td>
<td>0.14** (0.05)</td>
<td>0.07** (0.02)</td>
<td>0.07 (0.04)</td>
<td>0.06 (0.06)</td>
<td>0.04 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.09</td>
<td>0.10</td>
<td>0.10</td>
<td>0.10</td>
<td>0.08</td>
<td>0.10</td>
</tr>
<tr>
<td>Parents’ average depressive symptoms</td>
<td></td>
<td>0.30 (0.20)</td>
<td>0.10 (0.06)</td>
<td>0.08** (0.03)</td>
<td>0.10* (0.05)</td>
<td>0.03 (0.08)</td>
<td>0.04 (0.05)</td>
</tr>
<tr>
<td>R²</td>
<td></td>
<td>0.09</td>
<td>0.08</td>
<td>0.08</td>
<td>0.11</td>
<td>0.08</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Figures are standardized regression coefficients (β) and standard errors (SE) and coefficients of determination (R²)

*p < 0.05, **p < 0.01
Symptoms. It is the specific design of our study that enables the partitioning of genetic influence from psychosocial influence. Second, until recently the effect of paternal depression has largely been ignored while the effect of maternal depression has been substantially documented.

It has been argued that depressed men [48] are more likely to express, and elicit, negative emotions in interactions with their partners, and also more likely to disengage from couple interactions at times of stress. Men report some different symptoms when suffering with depression, with higher levels of irritability and anger expressed [33, 59]. In a recent study by Nath et al. [38] higher paternal depressive symptoms at 9 months were significantly associated with children’s emotion regulation at 7 years old, via higher father–child conflict when children were 3 years old. Thus, these factors may be the cause to the difference in the impact of paternal depression on the development of offspring psychopathology compared to maternal depression in our study.

To date, studies that have examined paternal depression [e.g., 39, 49] suggest that, in comparison to the depressive symptoms of adoptive mothers, those of adoptive fathers play a less consistent role as an environmental liability, at least in early childhood. It may be argued that paternal psychopathology increases its salience as children get older, because fathers may be more involved in parenting older children [40]. Taken this into consideration, it is important to contextualize the effect of paternal depression within children’s age and developmental milestones.

In our study, the associations with parental depressive symptoms emerged in the offspring’s subscale of interpersonal problems in particular and it was the only association to emerge with maternal depressive symptoms in girls. Considering the psychological health of adopted children, an important resilience factor is a positive relationship with the adoptive parents, which is believed to protect the adoptee from the negative consequences of adverse pre-adoption experiences [4]. The protective effect of sensitive parenting is suggested in a longitudinal study where maternal sensitivity was found to decrease the risk for internalizing problems in adolescence [57]. Research has indicated [18, 54] the exposure to mother’s depressed emotions as a mechanism through which the risk for internalizing problems is transmitted.

As stated before, results of past studies covering the issue of gender potentially modifying the relationship between parental and offspring depression are mixed (e.g., [43]. In our study, girls were more likely to be affected by parental depression. There was an association between mother’s depressive symptoms and the subscale of interpersonal problems and father’s depressive symptoms and the sub scales of interpersonal problems and negative mood. No associations were found between boys’ depressive symptoms and parental depressive symptoms.

**Strengths and limitations**

The results of our study must be considered in the light of the study’s strengths and its limitations. We did not measure clinical depression of the parents or the offspring. However, depressive symptoms measured with CDI or GHQ have shown to predict clinical depression [22, 25].

In some countries, infants may be given to adoption because of parental mental health problems. Nevertheless, the country of origin of the adopted children did not affect the associations between fathers and offspring depressive symptoms. In addition to lack of information about biological parents, we did not have information about mental health of children’s caretakers in early infancy. The children were adopted to the present families at the mean age of 2.74.

The major limitation was the cross-sectional design of our study. The association between parental depressive symptoms and offspring depressive symptoms may, of course, be due to reversed causality. This does not make this association any less serious. In our study, the offspring depressive symptoms were not reported by their parents and that can be considered as a strength compared to some previous studies.

There was no information available about the mental health of the biological parents but we do not have any reason to suggest that depressed fathers would be more likely to adopt children from depressed biological parents than others.

Some of the subscales showed relatively poor internal consistency, which suggests that the CDI may be more reliable when used as a complete scale rather than as subscales.

**Conclusions**

Our results support the idea that paternal depressive symptoms are associated with offspring psychopathology even without genetic risk.

Our study raises the importance of identifying both parental and maternal depressive symptoms in adoptive (and not only in biological) parents prior to adoption and the need to follow-up on their mental health after the adoption. This is necessary not only to identify depressive symptoms but to prevent the negative impact of parental depression on offspring mental health.

Because of the potential psychosocial transmission of depressive symptoms over generations, some screening of depressive symptoms of the adoptive parents after the adoption process would be useful so that support and services could be targeted and this transmission could be prevented. Further research is needed to find out the actual mechanisms behind the association between parental and offspring depressive symptoms, such as interaction and social processes within families.
Acknowledgements ME was supported by the Academy of Finland (265977). The study was supported by the Foundation of Pediatric Research, Finland and EVO Grant from Turku University Hospital.

Compliance with ethical standards

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

References


