At risk of depression and anxiety
Landman-Peeters, Karlien Maria Catharina

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2007

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
Landman-Peeters, K. M. C. (2007). At risk of depression and anxiety: studies into the interplay of personal and environmental risk factors s.n.

Copyright
Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

Take-down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): http://www.rug.nl/research/portal. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Download date: 18-01-2019
Chapter 4

Familial liability and offspring emotional problems: The role of parent-offspring relational stress

The present study has examined the role of parent-offspring relational stress in the association between familial liability, as indexed by the number of affected parents, and offspring emotional problems. In a Dutch sample of 407 adolescent and young-adult offspring of parents previously treated for depression, we examined (1) whether a second parent with lifetime emotional problems increased parent-offspring relational stress and, via the latter, the emotional problems of offspring, and (2) whether the strength of the association between parent-offspring relational stress and offspring emotional problems differed depending on the number of affected parents. The results have shown evidence for moderated mediation, that is, the association between familial liability and offspring emotional problems was due to (1) a mediation effect by parent-offspring relational stress such that offspring of two affected parents had higher levels of stress than offspring of one affected parent and (2) a moderator effect of familial liability indicated by a stronger association between parent-offspring relational stress and offspring emotional problems in offspring of two versus one affected parent. These findings suggest that parental depression incorporates a complex interplay between genetic and environmental effects on offspring emotional health in which both mediating and moderating influences are involved.

Acknowledgements - This study was funded by the Netherlands Organization for Scientific Research (NWO-MW). The authors thank Roelie Nijzing, Aukelien Mulder and Jaap Jansen for data collection and Liesbeth Lindenboom, Margo Jongsma, and Jacqueline Reisel for data entry. The authors wish to thank Dr. Sophia Frangou for her very helpful comments on an earlier draft.
Introduction

Offspring of depressed parents are at increased risk of developing mental health problems, particularly depression and anxiety (denoted henceforth as emotional problems) (e.g., Biederman et al., 2001; Lieb et al., 2002; Weissman et al., 1993; Wickramaratne & Weissman, 1998). Parental depression incorporates a complex interplay between genetic and environmental effects on offspring emotional health (Downey & Coyne, 1990; Nomura, Warner, & Wickramaratne, 2001), in which mediating and moderating influences are involved.

Parent-offspring relational stress, as indicated by troubled parent-offspring communication, low parental support, and poor family functioning, is found to partly explain (that is, mediate) the association between parental depression and offspring emotional problems (e.g., Brennan et al., 2002; Davies & Windle, 1997; Goodman, 1992; Kane & Garber, 2004; McCarty et al., 2003; Warner, Mufson & Weissman, 1995). Several studies have shown that parents suffering from depression are more critical and less involved in the lives of their children (Downey & Coyne, 1990; Hammen, Brennan, & Shih, 2004; Nomura et al., 2002). Parental depression is also associated with less cohesion and more conflicts within the family (Downey & Coyne, 1990; Nomura et al., 2002; Whaley, Pinto, & Sigman, 1999). Although parent-offspring relational stress is higher during acute episodes of parental depression than after remission, the adverse impact of parental depression on parent-offspring relations and family functioning may persist for up to ten years following remission (Keitner & Miller, 1990; Timko et al., 2002).

While parental depression thus increases parent-offspring relational stress, it may also alter its impact on offspring emotional problems (that is, moderation) (Fendrich, Warner, & Weissman, 1990; Hammen, Brennan, & Shih, 2004; Nomura et al., 2002; Pilowsky et al., 2006). In a community sample, Hammen, Brennan, and Shih (2004) not only found higher levels of parent-offspring relational stress, but also found a stronger association between parent-offspring relational stress and offspring depression in the offspring of depressed parents than was found in the offspring of non-depressed parents. Parental depression seemed to amplify the effect of parent-offspring relational stress. However, the results in research by Weissman and colleagues (Fendrich, Warner, & Weissman, 1990; Nomura et al., 2002; Pilowsky et al., 2006) are at odds with this. Weissman et al. found higher levels of parent-offspring relational stress in offspring of parents who were clinically treated for moderate or severe depression than was found in offspring of non-depressed parents, but this association between parent-offspring relational stress and offspring depression was significant only in the offspring of non-depressed parents (Fendrich, Warner, & Weissman, 1990; Nomura et al., 2002; Pilowsky et
al., 2006). Compared to parents in the Hammen et al. study, the parents in the study of Weissman et al. were more severely depressed (Hammen, Brennan, & Shih, 2004; Pilowsky et al., 2006). The contradictory findings from these studies could be reconciled if we assumed that the severity of parental depression is a measure of genetic liability to emotional problems and that the contribution of environmental factors, such as parent-offspring relational stress, to the development of offspring emotional problems decreased when liability increased. The impact of parent-offspring relational stress on offspring emotional problems would then be less when genetic liability was high.

To our knowledge, variation in risk among offspring of affected parents has not been studied in relation to the effect of parent-offspring relational stress. It therefore remains unclear whether the relative contribution of parent-offspring relational stress to offspring emotional problems depends on the degree of familial liability. Various studies have shown that the risk of emotional problems increases with the number of affected parents (e.g., Brennan et al., 2002; Foley et al., 2001; Marmorstein, Malone, & Iacono, 2004; Nomura, Warner, & Wickramaratne, 2001; Warner, Mufson, & Weissman, 1995). In the present paper, we have examined the role of familial liability, as inferred by the presence of one versus two emotionally affected parents, and also parent-offspring relational stress in the development of offspring emotional problems. Combining the evidence above, we hypothesize moderated mediation, such that (1) the association between familial liability and offspring emotional problems is mediated by higher levels of parent-offspring relational stress and (2) familial liability interacts with parent-offspring relational stress such that the strength of the association between parent-offspring relational stress and offspring emotional problems differs according to level of familial liability. According to Hammen’s work a higher familial liability should strengthen the association between parent-offspring relational stress and offspring emotional problems, whereas Weissman et al.’s work indicates that a higher familial liability would weaken it. We tested our hypotheses using longitudinal data from a Dutch sample of 407 adolescent and young-adult offspring from families in which at least one parent had a history of treated depression.

**Method**

*Subjects and Procedure*

The present study used data from 407 adolescents and young adults (175 males and 232 females) who participated in the first (T1) and second (T2) waves of assessment of the
Dutch ARIADNE (Adolescents at Risk of Anxiety and Depression; A combined Neurobiological and Epidemiological approach) Study. The study design of ARIADNE has been previously described (Landman-Peeters et al., 2005). Briefly, patients with a) at least one treated episode of emotional disorder, b) no personal history of schizophrenia spectrum diagnoses and c) having biological children aged 13-25 were identified through psychiatric services in the three northern provinces of the Netherlands. At baseline (T1) consenting parents and their children were interviewed in person and were also asked to complete a number of questionnaires. Only the recruited parent was interviewed and he/she provided information about the other biological parent. Approximately one year after the first measurement, offspring were sent a second set of questionnaires for the first follow-up assessment (T2).

At T1 the mean age of the 407 offspring in the present study was 18.35 years (SD=3.25). They came from 286 families where the informant parent had been treated for Major Depression Disorder (MDD) with or without co-morbid anxiety. Eighty offspring came from 62 families with two emotionally affected parents. There were no gender or age differences between the groups of offspring with one or two affected parents.

Measures

Parent-offspring relational stress. Measurement at T1 included offspring reports on parent-offspring communication, parental support, and family functioning as measures of parent-offspring relational stress.

Parent-offspring communication. We used the Dutch version of the Parent-Adolescent Communications Scales (PACS) (Barnes & Olson, 1995; Jackson et al., 1998). The PACS consists of twenty statements that assess either problems (for example, “My mother/father tends to say things to me that are better left unsaid”) or openness (for example, “I feel comfortable with discussing problems with my mother/father”) in parent-offspring communication. Participants indicated their degree of agreement with each statement by choosing one of four options: “Strongly agree,” “Agree,” “Disagree” and “Strongly disagree.” Offspring completed a separate PACS for each parent. The scale yields both an Openness and a Problems score. The Openness score is reversed and then added to the Problems score to yield a composite total score. The higher the total score the more problematic the parent-offspring communication. The combined scales here showed a reliability coefficient of 0.89 for Father-Offspring Communication and of 0.90 for Mother-Offspring Communication.
Parental support. The Social Support Questionnaire – short-form (SSQ) (Sarason et al., 1987) was used to collect information about paternal and maternal support. The SSQ was administered during the interview due to the fact that it requires extensive input. This instrument consists of six items that describe different aspects of social support, for example, “Who can you really count on to be dependable when you need help?” and “Who accepts you totally, including both your worst and your best points?” To establish Paternal Support and Maternal Support scales we counted how often participants respectively mentioned their father or mother as providing support. The interviewers stressed that each item on the SSQ tapped a different form of support and, therefore, should be considered separate from the other items.

Family functioning. This was assessed using the Cohesion and Adaptability scales of the Dutch Family Dimension Scales (FDS) (Buurmeijer & Hermans, 1988). The FDS is based on the Family Adaptability and Cohesion Evaluation Scales (FACES) by Olson, Portner, & Lavee (1985). Family cohesion is described as the degree to which family members feel connected and close to each other on an emotional level. This scale consists of twenty-three items, for example, “Our family makes decisions together.” Family adaptability is described as the family’s ability to change its power structure, roles, and rules in order to adapt to circumstantial and developmental stress. This scale consists of thirteen items, for example, “Rules change in our family.” Offspring used a four-point scale ranging from “Never true” to “Always true” to report to what extent the items applied to their family. Internal consistency reliability was 0.87 for the Cohesion scale and 0.84 for the Adaptability scale.

Parent-offspring relational stress index. The previous scales measured the quality of the parent-offspring relationship in the domains of communication, support and family functioning. In order to create a general measure of parent-offspring relational stress we conducted a factor analysis on a Pearson correlation matrix of the six scales described above, using the maximum likelihood estimation method in SPSS version 11.0. Both the scree-test (Cattell, 1966) and the eigen-value-greater-than-one rule (Kaiser, 1960) indicated a single underlying factor with loadings that ranged from |0.591| to |0.696|, with positive loadings for the Father-Offspring Communication, Mother-Offspring Communication and Adaptability scales and negative loadings for the Paternal Support, Maternal Support, and Cohesion scales. After reverse scoring these latter scales, the standardized scores of the six scales were added together. This parent-offspring relational stress index was then used in all subsequent analyses.

Parental disorder. We gathered information about parental emotional disorder at T1. For parents identified through psychiatric services, we used the World Mental Health (WMH)
Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI) (Kessler & Üstün, 2004) to assess lifetime MDD according to criteria of the Diagnostic and Statistical Manual of Mental Disorders (4th Edition) (DSM-IV) (American Psychiatric Association, 1994). These informant parents were asked about the history of treatment for emotional disorder of the other biological parent by means of vignettes about depressive and anxiety disorders based on DSM-IV diagnostic criteria. Only parents who had received treatment for emotional disorder were classified as “affected.” In the Netherlands approximately seventy-five percent of those individuals with a diagnosable depression and/or anxiety disorders seek treatment (Ten Have et al., 2004); we therefore reasoned that the inclusion of this criterion would minimize any false positive classification and also serve as a proxy measure of equal “illness severity” for the two affected parents.

**Offspring emotional problems.** The DSM-IV Questionnaire (Hartman, 2002; Hartman et al., 2001; Muris, 2006; Muris, Winands, & Horseleben, 2003) was used to assess offspring emotional problems at T2. Respondents were asked to report on a four-point Likert scale to what extent descriptions of symptomatic behavior accurately described their behavior at the time of measurement. The DSM-IV Questionnaire includes items on a broad range of psychiatric symptoms. The Emotional Problems scale includes thirty-two items, for example “I am often unhappy,” “I am low in energy or feel tired for no reason,” and “I suddenly become very anxious or panicky for no reason.” Internal consistency reliability was 0.94.

**Data analyses**

Our two key hypotheses were that (1) the association between familial liability and offspring emotional problems is mediated by parent-offspring relational stress and (2) the strength of the association between parent-offspring relational stress and offspring emotional problems is moderated by familial liability. Prerequisites for mediation are that the independent variable, in this case familial liability, is associated with both the outcome and mediator, in this case offspring emotional problems and parent-offspring relational stress, and that the mediator must significantly contribute to the prediction of the outcome variable in an equation in which both the mediator and the independent variable are included (e.g., Baron & Kenny, 1986). Mediation is indicated if the effect of the independent variable decreases when the mediator is included in the regression equation. However, we expected moderated mediation, such that in our model familial liability
would both be the independent variable and the moderator (see Figure 1). Therefore, the interaction between familial liability and parent-offspring relational stress had to be included in the equation as well (cf. Preacher, Rucker, & Hayes, 2007).

![Figure 1](image)

**Figure 1** Model of moderated mediation by parent-offspring relational stress in the association between familial liability and offspring emotional problems (see Preacher, Rucker, & Hayes, 2007)

We first examined whether familial liability was associated with both offspring emotional problems and parent-offspring relational stress by testing mean differences between the offspring of one and the offspring of two affected parents. We additionally conducted a regression analysis to examine the extent to which familial liability predicted parent-offspring relational stress. Then we conducted a regression analysis to examine moderated mediation, which consisted of two steps. In the first step we entered familial liability and in the second step we added parent-offspring relational stress and its interaction with familial liability. By means of this second step, we were able to examine the decrease in the effect of familial liability after adding parent-offspring relational stress (that is, mediation) and whether the effect of parent-offspring relational stress differed according to familial liability (that is, moderation). If the interaction between familial liability and parent-offspring relational stress was significant, the interaction effect was then plotted and the association between parent-offspring relational stress and offspring emotional problems was separately examined for the offspring of one and the offspring of two affected parents. If not, mediation was tested using the full sample.

At T1, relatively more offspring of two rather than of one affected parent had moved out of the parental home and were living independently (33.8% versus 22.0%; $\chi^2=4.81, p=0.028$) and had experienced parental divorce (25.0% versus 15.3%; $\chi^2=4.26, p=0.039$). These differences may affect associations between familial liability, parent-offspring relational stress, and offspring emotional problems. Therefore, in the regression analyses we adjusted for the effects of offspring age and gender as well as for offspring residential status and parental divorce.
The 407 offspring came from 286 families. To account for this clustering of observations in families, we conducted design-based regression analyses with families as primary sampling units, using the statistical program STATA 8.0 (StataCorp, 2003). All variables were standardized to improve interpretability and prevent computational problems due to multicollinearity that may occur with variables and their products (Aiken & West, 1991).

Results

Group differences in emotional problems and parent-offspring relational stress

The mean total score for emotional problems was higher in offspring with two affected parents ($M= 51.09, SD=14.31$) than in offspring of one affected parent ($M=46.47, SD=12.27$) and this difference was statistically significant ($t(df=405)=2.92, p=0.004$). Similarly, the mean parent-offspring relational stress index was higher ($t(df=405)=2.75, p=0.006$) in offspring of two affected parents ($M=1.22, SD=4.68$) than in offspring of one affected parent ($M=-0.26, SD=4.21$).

Moderated mediation analysis

Our first regression analysis showed that a higher familial liability significantly predicted parent-offspring relational stress ($\beta=0.136 (SE=0.060), p=0.024, 95\% CI=0.018\text{-}0.254$). Thus, after controlling for the effects of offspring gender, age, residential status, and parental divorce, the offspring of two affected parents reported

<table>
<thead>
<tr>
<th>Step 1</th>
<th>$B$ (SE)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family liability</td>
<td>.146 (.050)**</td>
<td>.048 -.243</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family liability</td>
<td>.087 (.040)*</td>
<td>.008 -.166</td>
</tr>
<tr>
<td>Parent-offspring relational stress</td>
<td>.281 (.049)**</td>
<td>.186 -.377</td>
</tr>
<tr>
<td>Family liability $\times$ Parent-offspring relational stress</td>
<td>.100 (.050)*</td>
<td>.001 -.200</td>
</tr>
</tbody>
</table>

Analyses were controlled for the effects of offspring gender, age, and residential status, and parental divorce. All variables were standardized; ** $p<.01$; * $p<.05$.
more parent-offspring relational stress than did offspring of one affected parent. The association was not strong; familial liability accounted for only 2% of the variance in parent-offspring relational stress. Table 1 shows the results of the second regression analysis. This analysis revealed a significant main effect of familial liability in both the first and second step. The inclusion of parent-offspring relational stress and its interaction with familial liability in the second step decreased the effect of familial liability on offspring emotional problems. This indicates that the higher levels of parent-offspring relational stress in offspring of two affected parents could partly explain the higher levels of emotional problems in these children (that is, mediation). In addition, the results showed a significant interaction between familial liability and parent-offspring relational stress. Familial liability moderated the association between parent-offspring relational stress and offspring emotional problems. Regression analyses in the two groups of offspring separately showed a stronger association between parent-offspring relational stress and offspring emotional problems in offspring of two affected parents ($B=0.413 (\text{SE}=0.100), p<0.000, 95\% \text{ CI}= 0.214-0.612$, explained variance 17%) than in offspring of one affected parent ($B=0.252 (\text{SE}=0.056), p<0.000, 95\% \text{ CI}= 0.141-0.363$, explained variance 6%). Figure 2 illustrates the moderator effect of familial liability adjusted for differences in parent-offspring relational stress between offspring of one and two affected parents. The figure indicates that the level of emotional problems is similar in offspring of one versus two affected parents when parent-offspring relational stress is low. However when parent-offspring relational stress is high, offspring of two affected parents show more emotional problems than offspring of one affected parent.

![Figure 2](image_url)

**Figure 2** Regression lines for the association between parent-offspring relational stress and offspring emotional problems in offspring of one affected parent and offspring of two affected parents
Discussion

In a sample of adolescent and young-adult offspring of parents with a history of treated depression, we found evidence for moderated mediation by parent-offspring relational stress in the association between familial liability and offspring emotional problems. Higher levels of parent-offspring relational stress in offspring of two affected parents at baseline partly mediated the association between familial liability and offspring emotional problems at one year follow-up, while the effect of parent-offspring relational stress was stronger in the offspring of two than it was in the offspring of one affected parent (that is, moderation).

Our results indicate that increased familial liability incorporates an interplay between increased genetic liability and increased environmental adversity, in this case parent-offspring relational stress. This is in line with the findings of Hammen, Brennan, and Shih (2004) that parental depressive disorder both increased parent-offspring relational stress and strengthened the association between parent-offspring relational stress and offspring depressive disorder. Our findings, however, contrast with the results of Weissman et al. (Fendrich et al., 1990; Nomura et al., 2002; Pilowsky et al., 2006) where parent-offspring relational stress did not make an independent contribution to psychopathology in the offspring of moderately and severely depressed parents. Our results indicate that increased genetic liability strengthens instead of weakens the association between parent-offspring relational stress and offspring emotional problems. Similar to the Weissman et al. study, the parents in the present study received clinical treatment for depression. In contrast though, the Weissman et al. studies used offspring lifetime depressive and anxiety disorder as outcomes, whereas we used a dimensional measure of current depression and anxiety symptoms. Similarly, Weissman et al. used dichotomous measures of the quality of parent-offspring relations, where we used an aggregated continuous measure. Our measures may have been more sensitive for the following reasons: a) dichotomous measures statistically attenuate associations between variables, b) dichotomous measures of psychopathological symptoms do not capture high but (still) subclinical levels of emotional problems, and c) dichotomous measures of relationship quality may have less validity as most aspects of parent-offspring relations seem to be a matter of degree rather than of presence or absence. Additionally, our measure was a composite of aspects of the parent-offspring relationship. Factor analysis supported the notion of one general underlying factor. Possibly, this composite captures adversity in parent-offspring relations more accurately than separate measures do. High levels of problems in parent-offspring communication may be compensated by parental
support and family cohesion or vice versa, and, similarly, lack of support from one parent may be compensated by support received from the other parent.

The greater reactivity of offspring of two affected parents to adversity in parent-offspring relations probably reflects an interplay between genetic and environmental factors. Parental depression is thought to genetically and/or environmentally affect offspring neurobiological processes, coping abilities, problem-solving skills, and availability of protective resources (Downey & Coyne, 1990; Goodman & Gotlib, 1999; Hammen, Brennan, & Shih, 2004). In comparison to offspring of one affected parent, offspring with two affected parents may be at increased risk since they lack a non-affected parent who is able to compensate, genetically and/or environmentally, for the affected parents’ negative influence on these aspects (Downey & Coyne, 1990).

Our findings should be considered in the context of the following limitations. Firstly, information about parental psychiatric history was based solely on the report of only one parent. Although accuracy improves with the severity of problems, people generally tend to underreport psychiatric illness in their relatives (Heun, Maier, & Müller, 1997). This will reduce the contrast between offspring with one versus two affected parents and, consequently, lead to underestimation of the associations. Secondly, we assumed a unidirectional relationship between parental and offspring emotional problems, but reciprocal associations have been shown, along with offspring emotional problems having a negative impact on parental mental health and parent-offspring relations as well (Ge et al., 1995).

In sum, the findings of the present study indicate that emotional problems in offspring of emotionally disordered parents are increased by the presence of genetic and environmental factors, which act in an additive and interactive fashion. Although we may not be able to decrease genetic vulnerability to parent-offspring relational stress, parent-offspring relations and family functioning can be targeted and improved by prevention and intervention programs.