At risk of depression and anxiety
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Risk of emotional disorder in offspring of depressed parents: Gender differences in the effect of a second emotionally affected parent

In offspring of depressed parents a second parent with emotional problems is likely to increase risk of emotional disorder. This effect may however differ between sons and daughters and between offspring of depressed fathers and offspring of depressed mothers. In adolescent and young-adult offspring of parents with Major Depressive Disorder this study examined the effects of a second affected parent, offspring gender, gender of the depressed parent and their interactions on risk of depression and anxiety disorder. We found that daughters had a higher risk of depression and anxiety than sons and that offspring of depressed mothers had a higher risk of anxiety than offspring of depressed fathers. In addition to these main effects, we found an interaction between parent and offspring gender in as much that sons of depressed fathers had the lowest risk of depression and anxiety relative to the other groups. A second affected parent tended to increase risk of depression and significantly increased risk of anxiety. However, this effect of a second affected parent on offspring anxiety was most prominent in daughters when the second affected parent was the father while risk in sons did not increase if the father was affected as well. Our results indicate that paternal and maternal depression similarly and additively increase daughters’ risk of emotional disorder, but that sons’ risk only increases with maternal depression. Intergenerational transmission of emotional disorder seems strongest when the female gender is involved, either in the form of a daughter or a depressed mother.

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Familial liability and gender

Introduction

Offspring of depressed parents are at increased risk to develop mental health problems, particularly depression and anxiety disorders (further denoted as emotional disorders) (e.g., Biederman et al., 2001; Lieb et al., 2002; Weissman et al., 1993; Wickramaratne & Weissman, 1998). The onset of depression in adolescence and young adulthood is often preceded by anxiety in childhood and early adolescence (e.g., Avenevoli et al., 2001; Cohen et al., 1993) and depression is often co-morbid with anxiety later on (e.g., Angold, Costello, & Erkanli, 1999). Parental depression incorporates a complex interplay of genetic and environmental effects on offspring mental health (Downey & Coyne, 1990; Nomura, Warner, & Wickramaratne, 2001). By examining variation in risk among offspring of depressed parents we can further our understanding of the intergenerational transmission of emotional disorder. In offspring of parents with Major Depressive Disorder (MDD), the present study examined the effect of a second parent with life-time emotional problems on offspring risk.

Depressed patients tend to form relationships with individuals who also suffer from psychiatric problems, which is referred to as assortive mating (e.g., Merikangas et al., 1988). In offspring of depressed parents risk of emotional problems is found to increase when the other biological parent has a history of psychiatric disorder as well (e.g., Brennan et al., 2002; Foley et al., 2001; Marmorstein, Malone, & Iacono, 2004; Nomura, Warner, & Wickramaratne, 2001; Warner, Mufson, & Weissman, 1995). Goodman and Gotlib (1999) formulated four mechanisms by which parental depression can increase offspring risk: a) genetic transmission; b) the development of dysfunctional neuroregulatory mechanisms; c) exposure to the parent’s maladaptive affect, behaviour and cognitions; and d) contextual stressors associated with parental depression. Thus, a second affected parent may add to the genetic and environmental risk (Brennan et al., 2002; Dierker, Merikangas, & Szatmari, 1999; Nomura, Nomura, & Wickramaratne, 2001). Offspring with a second affected parent may also be at increased risk since they lack a healthy parent who is able to compensate, genetically and/or environmentally, for the depressed parent’s influence on offspring functioning (Downey & Coyne, 1990; Tannenbaum & Forehand, 1994). With respect to the development of emotional disorders, a history of depression or anxiety in the other biological parent seems especially relevant. For the present study we expect that risk of emotional disorder in offspring of depressed parents increases when the other parent has life-time emotional problems as well.

The effect of a second parent may however differ between sons and daughters. From adolescence on, risk of emotional disorders is approximately twice as high in
women than in men (Costello & Angold, 1995; Hankin & Abramson, 1999). This gender difference is thought to be caused (partially) by a greater heritability for depression in women and/or a stronger reactivity to (interpersonal) stress in women (Cryanowski et al., 2000; Hankin & Abramsom, 2001; Shih et al., 2006; Silberg et al., 1999). Among offspring of depressed parents, daughters have a higher risk of emotional problems than sons. This finding may reflect the generally observed gender difference (Garber & Flynn, 2001; Goodman & Gotlib, 1999), such that parental depression increases risk for daughters and sons in a similar way. Above and beyond this, daughters may be more strongly affected than sons by parental depression and/or its correlates (e.g., problems in parent-offspring interaction and family functioning) (e.g., Sheeber, Davis, & Hops, 2002). If we assume the latter, a second parent with emotional disorder may increase risk more in daughters than in sons.

A second parent with emotional problems may further differentially increase risk in offspring of depressed fathers and offspring of depressed mothers. Research indicates that the mechanisms formulated by Goodman & Gotlib (1999) are stronger for maternal than for paternal depression (Connell & Goodman, 2002; Field, Hossain, & Malphurs, 1999; Goodman & Gotlib, 1999; Jacob & Johnson, 1997; Johnson et al., 1999; Kendler et al., 2001). This is reflected in the higher risk of emotional problems in offspring of depressed mothers than in offspring of depressed fathers reported by overviews of the literature (Connell & Goodman, 2002; Phares & Compa, 1992). Assuming that offspring are affected more by maternal than by paternal depression, a second affected parent may add less to the risk of emotional disorder in offspring when this second affected parent is the father.

Given the aforementioned gender differences in offspring and parents it is likely that the effect of a second affected parent differs according to the offspring-depressed parent gender dyad. Several studies included the gender of both offspring and the depressed parent to examine gender differences in offspring emotional disorder (Eberhart et al., 2006; Foley et al., 2001; Hops, 1992; Klein et al., 2005; Nomura, Warner, & Wickramaratne, 2001; Thomas & Forehand, 1991). However, results of these studies are inconsistent. Klein et al. (2005) found effects on depression in both sons and daughters but only of maternal and not of paternal depression. Foley et al. (2001) found that paternal depression increased daughters’ but not sons’ depression and anxiety, while maternal depression increased depression only in daughters and increased anxiety stronger in daughters than in sons. Thomas and Forehand (1991) found that maternal depression was related to emotional problems in daughters and paternal depression to emotional problems in sons. The findings of Nomura et al. (2001) indicate a reverse pattern in which paternal depression more strongly affected daughters’ risk of depression and maternal
depression more strongly affected sons’ risk of depression, but they found that paternal and maternal depression similarly increased risk of anxiety disorder in both sons and daughters. Eberhart et al. (2006) found that maternal depression increased risk of depression in both sons and daughters, but paternal depression increased risk only in sons. Hops (1992) reports that associations between parental and offspring depression symptoms were strongest for mothers and daughters, but less consistent and somewhat weaker when fathers or sons were involved. Based on these findings we assume that the effect of a second parent with life-time emotional problems differs according to the depressed parent-offspring gender dyad. Combining our expectations concerning the stronger effect of a second affected parent in daughters and offspring of depressed fathers we hypothesize that a second affected parent increases risk the most in daughters of depressed fathers.

The present examination used data from a Dutch family study among adolescent and young-adult offspring of parents with life-time emotional disorder. For the present study we focused on offspring of parents with MDD to test the following hypotheses: (1) a second parent with life-time emotional problems increases offspring risk of depression and anxiety disorder, (2) a second affected parent increases risk more in daughters than in sons, (3) a second affected parent increases risk more when this second affected parent is the mother than when it is the father, and (4) this latter effect is stronger for daughters.

**Methods**

**Participants**

The present study uses data from 349 (154 male and 195 female) offspring between 16 and 25 years old ($M=19.8$, $SD=2.7$) from 263 families participating in 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 between 1990 and 2002, b) no personal history of schizophrenia spectrum diagnoses, and c) biological children aged 13-25 were identified through 16 psychiatric services in the three northern provinces of the Netherlands. Consenting parents and their children were interviewed in person at their home or at the Department of Psychiatry. Only the recruited parent was interviewed and he/she provided information about the other biological parent. Parents and offspring were interviewed by different interviewers. Interviews were conducted by intensively trained and monitored
The Department of Psychiatry at the University of Groningen is an Expert Training Center for the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI).

The 349 offspring came from 263 families where the recruited parent had MDD; 113 offspring had a depressed father and 236 offspring had a depressed mother. Gender ratio and educational level of parents were comparable to findings from a large Dutch population sample concerning individuals with depression (Ten Have et al., 2004). Sixty-nine offspring came from 54 families where the other biological parent had life-time emotional problems as well. The majority of the participants (95%) were white and of Dutch origin.

Measures

Offspring depression and anxiety. Offspring were interviewed with the World Mental Health (WMH) Survey Initiative Version of the WHO-CIDI (Kessler & Üstün, 2004). It is a structured interview to assess mental disorders designed for use by trained interviewers who are not clinicians. By means of computerized algorithms, it provides diagnoses according to accepted criteria such as the International Classification of Diseases (ICD-10) and the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV). The CIDI has been shown to be reliable and valid (Kessler et al., 1994; Lieb et al., 2002; Wittchen, 1994).

To increase sensitivity to offspring problems we assessed a wide range of depression and anxiety disorders. In the present study 44.4% of the offspring had a life-time diagnosis of emotional disorder (i.e. depression and/or anxiety). Depression was present in 30.1% of the offspring. This included the presence of a DSM-IV lifetime diagnosis of MDD (23.5%), Dysthymic Disorder (4.3%) and/or the presence of minor depression (4.9%) or recurrent brief depression (1.1%). Minor depression incorporates episodes of depression in which dysphoria or anhedonia persisted half the day and/or worst life-time episodes in which at least two symptoms of depression were present (Kessler & Üstün, 2004). Recurrent brief depression incorporates episodes of depression lasting at least three days occurring in most months in a row for an entire year (Kessler & Üstün, 2004). Anxiety was present in 33.5% of the offspring. This included a DSM-IV life-time diagnosis of Generalized Anxiety Disorder (7.4%), Obsessive Compulsive Disorder (8.6%), Social Phobia (9.2%), Separation Anxiety Disorder (4.0%), Adult Separation Anxiety Disorder (4.9%), Panic Disorder (8.6%), Agoraphobia (4.0%) and/or the experience of multiple panic attacks (12%).
Parental depression and life-time emotional problems in the other biological parent. For parents identified through psychiatric services we also used the CIDI (Kessler & Üstün, 2004). We assessed life-time Major Depressive Disorder according to DSM-IV (American Psychiatric Association, 1994). These parents were asked about the history of emotional disorder of the other biological parent by means of vignettes of 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 75% of the individuals with diagnosable depression and/or anxiety disorders seek treatment (Ten Have et al., 2004); we therefore reasoned that the inclusion of this criterion minimised false positive classification and also served as a proxy measure of equal “illness severity” for the two affected parents.

Data analysis
We first calculated the prevalence of depression and anxiety for each combination of offspring gender, gender of the depressed parent and the presence of a second affected parent. As previously mentioned, the 349 offspring came from 263 families. To account for this clustering in families we examined our hypotheses in design-based analyses with families as primary sampling units, using the statistical program STATA 8.0 (StataCorp, 2003).

We examined bivariate associations between our predictor and outcome variables by means of Pearson $\chi^2$-tests. These tests were corrected for the survey design and converted into $F$-statistics (StataCorp, 2003). To test our hypotheses we then conducted logistic regression analyses. This enabled us to simultaneously examine the effects of offspring gender (0=male; 1=female), gender of the depressed parent (0=male; 1=female), the presence of a second affected parent (0=not present; 1=present) and their interactions on the binary outcome measures of offspring emotional disorder. We performed stepwise backward analyses starting with the model including the three-way and all two-way interactions.

Our data indicated that the mean age of offspring with life-time depression ($M=20.8$, $SD=2.7$) was significantly higher than the mean age of offspring without life-time depression ($M=19.4$, $SD=2.6$) ($t(347)=4.30, p<0.001$), but age was not associated with our predictor variables. Age could therefore not be a confounder of associations between predictors and outcome and was not included in our analyses.
Results

Prevalence of depression and anxiety

Prevalence of depression and anxiety are presented for each group of offspring in Table 1.

Table 1: Prevalence of offspring depression and anxiety by offspring gender, gender of the depressed parent and the presence of a second affected parent (total n)

<table>
<thead>
<tr>
<th>Depression</th>
<th>Depressed father</th>
<th></th>
<th>Depressed mother</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mother not affected</td>
<td>Mother affected</td>
<td>Father not affected</td>
<td>Father affected</td>
</tr>
<tr>
<td>Sons</td>
<td>15.6 (45)</td>
<td>12.5 (16)</td>
<td>24.7 (77)</td>
<td>37.5 (16)</td>
</tr>
<tr>
<td>Daughters</td>
<td>41.5 (41)</td>
<td>45.5 (11)</td>
<td>30.8 (117)</td>
<td>50.0 (26)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>8.9 (45)</td>
<td>31.3 (16)</td>
<td>32.5 (77)</td>
<td>18.8 (16)</td>
</tr>
<tr>
<td>Sons</td>
<td>31.7 (41)</td>
<td>54.5 (11)</td>
<td>35.9 (117)</td>
<td>73.1 (26)</td>
</tr>
</tbody>
</table>

Bivariate associations

Daughters had a higher risk than sons of both life-time depression ($F(1,262)=7.98, p=0.005$) and life-time anxiety ($F(1,262)=11.10, p=0.001$). Compared to offspring of depressed fathers, offspring of depressed mothers had a higher risk of anxiety ($F(1,262)=5.62, p=0.019$) but not of depression ($F(1,262)=0.48, p=0.491$). Risk of anxiety was also higher in offspring with a second parent with life-time emotional problems ($F(1,262)=7.82, p=0.006$) but risk of depression was not ($F(1,262)=2.23, p=0.136$).

Logistic regression analyses

Table 2 presents the results of the logistic regression analyses for offspring depression and anxiety. The final model for depression shows that daughters had a higher risk than sons and that offspring of depressed mothers tended to have a higher risk than offspring of depressed fathers, but this latter effect was not significant ($p<0.10$). Similarly, offspring with a second affected parent tended to have a higher risk than offspring without a second affected parent, but this was not a significant effect either ($p<0.10$). The results show a significant interaction between offspring gender and gender of the depressed parent which indicates that sons of depressed fathers had the lowest risk in comparison to the other offspring and that the gender difference in offspring risk of depression was smaller in offspring of depressed mothers than in offspring of depressed fathers (see Figure 1 and
Table 2 Results logistic regression analyses

<table>
<thead>
<tr>
<th></th>
<th>Offspring depression</th>
<th>Offspring anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE)</td>
<td>p</td>
</tr>
<tr>
<td>Offspring gender</td>
<td>1.48 (.48)</td>
<td>.002**</td>
</tr>
<tr>
<td>Gender depressed parent</td>
<td>.81 (.44)</td>
<td>.069</td>
</tr>
<tr>
<td>Second affected parent</td>
<td>.50 (.30)</td>
<td>.090</td>
</tr>
<tr>
<td>Gender offspring ×</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender depressed parent</td>
<td>-1.14 (.57)</td>
<td>.045*</td>
</tr>
<tr>
<td>Gender offspring ×</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second affected parent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender depressed parent × Second affected parent</td>
<td>-2.27 (.98)</td>
<td>.021*</td>
</tr>
<tr>
<td>Gender offspring ×</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender depressed parent × Second affected parent</td>
<td>2.90 (1.36)</td>
<td>.033*</td>
</tr>
</tbody>
</table>

-- non-significant interaction effect, not included in the final model; *p<0.05; **p<0.01

Table 1). The final model did not include interactions with the presence of a second affected parent, indicating that the effect of a second affected parent on offspring risk of depression did not differ according to the gender of offspring and/or depressed parent.

Figure 1 Interaction between offspring gender and gender of the depressed parent in offspring risk of depressive disorder

The final model for anxiety included all main effects and interactions. The main effects indicate that risk of anxiety was higher in daughters, in offspring of depressed mothers and in offspring with a second affected parent. We found two significant two-way interactions: risk of anxiety was lowest in sons of depressed fathers and a second affected
parent increased risk more if this second affected parent was the mother than if it was the father. The third two-way interaction was not significant, suggesting that the effect of a second affected parent did not differ according to offspring gender. However, the significant three-way interaction indicates that the effect of a second affected parent depended on the offspring-depressed parent gender dyad. Figure 2 illustrates that the effect of a second affected parent was strongest in daughters if this second affected parent was the father, while risk of anxiety in sons of depressed mothers did not increase when the father was affected as well.

![Figure 2 Interaction between offspring gender, gender of the depressed parents, and presence of second affected parent in offspring risk of anxiety disorder](image)

**Figure 2** Interaction between offspring gender, gender of the depressed parents, and presence of second affected parent in offspring risk of anxiety disorder

**Discussion**

In offspring of depressed parents, we examined the effect of a second parent with lifetime emotional problems on offspring risk of depression and anxiety. Due to a relatively large sample size, we were able to examine the effect of a second affected parent on offspring according to offspring gender and gender of the depressed parent. We hypothesized that (1) a second affected parent would increase offspring risk of depression and anxiety, (2) a second affected parent would increase risk more in daughters than in sons, (3) risk would increase more if the second affected parent was the mother than if it was the father and (4) that this latter effect would be stronger for daughters. The results for depression did not but the results for anxiety partially did confirm our expectations.
Confirming our first hypothesis, a second affected parent increased offspring risk of anxiety. In line with our third hypothesis, we found that the effect of second affected parent on offspring anxiety was stronger if this was the mother. We did not find confirmation for our second expectation that a second affected parent would increase risk more in daughters than in sons. Although we found that the effect of a second affected parent differed according to the offspring-depressed parent gender-dyad, the differences were not as we expected. Contrary to our expectations, the effect of a second affected parent on daughters’ risk of anxiety seemed stronger if this involved the father rather than the mother. We do not know how to explain this. Moreover, our results show that risk in sons did not increase if the second affected parent was the father, while risk in sons and daughters seemed to increase in a similar way if the second affected parent was the mother.

While we found a significant effect of a second affected parent on offspring risk of anxiety, the effect on offspring risk of depression did not reach significance. This seems to contrast with the findings of Nomura, Warner, and Wickramaratne et al. (2001) who found that a second parent with MDD similarly increased offspring risk of depression and anxiety. Our findings are however in line with those of Foley et al. (2001) who reported a stronger effect for offspring anxiety than for offspring depression. Anxiety is suggested to be a prodromal manifestation of depression (Breier, Charney, & Heninger, 1985). Foley et al. reasoned that the effect of a second affected parent was stronger for anxiety than for depression since their sample, similar to ours, consisted of adolescents and young-adults who may have developed anxiety, but may not yet have developed depression. This may have attenuated the possible effect of a second affected parent on offspring risk of depression.

For both depression and anxiety, we found that sons of depressed fathers were at a lower risk than sons of depressed mothers. Risk did not differ between sons of depressed mothers and daughters of either depressed fathers or depressed mothers. This finding seems to combine the higher risks found in daughters and offspring of depressed mothers somewhat. However, instead of a relatively higher risk in daughters of depressed mothers compared to the other offspring in our sample, we found a relatively lower risk in sons of depressed fathers. Furthermore, our findings concerning the effect of a second affected parent indicate that maternal and paternal emotional disorder similarly and additively increase risk of anxiety in daughters, but that risk in sons increases by maternal emotional disorder only. Overviews of the literature (Connell & Goodman, 2002; Phares & Compas, 1992) indicate that the difference between effects of maternal versus paternal emotional disorder is not substantial. However, these overviews did not distinguish between effects in sons and effects in daughters, because studies examining differences between maternal
and paternal depression generally do not include offspring gender (Connell & Goodman, 2002). We formally tested the gender differences by means of interactions and found no difference between maternal and paternal disorder for daughters but did find a difference for sons. The few studies that include both parent and offspring gender report inconsistent results (Eberhart et al., 2006; Foley et al., 2001; Hops, 1992; Klein et al., 2005; Nomura, Warner, & Wickramaratne, 2001; Thomas & Forehand, 1991). Our findings are partially in line with the results of each of these studies. Klein et al. (2005) also found that maternal depression affected risk in both sons and daughters, but these authors did not find an effect of paternal depression. The results of Foley et al. (2001) concerning anxiety are very similar to ours: they found that maternal depression increased risk in both sons and daughters while paternal depression only increased risk in daughters. Their results concerning depression also show that both maternal and paternal depression increased risk in daughters but they did not find effects for sons. Also in line with our findings, Thomas and Forehand (1991) found that maternal depression affected sons, but in contrast with our study, maternal depression did not affect daughters, while paternal depression only affected sons. Similar to our findings, Nomura et al. (2001) found that sons’ risk of depression only increased with maternal depression. However, they found that paternal depression increased daughters’ risk of depression more than maternal depression, while paternal and maternal depression similarly and additively increased risk of anxiety disorder in both sons and daughters. In line with our results, Eberhart et al. (2006) found that maternal depression increased risk in both sons and daughters, but while we found that paternal depression only increased risk in daughters, Eberhart et al. only found an effect of paternal depression in sons. Based on his results Hops (1992) suggested that the intergenerational transmission of depression is stronger for daughters than for sons and stronger for mothers than for fathers. Our results suggest this for both offspring depression and anxiety but clearly more research is needed to find out which results replicate. Our results also indicate that future studies examining differences according to the gender of the depressed parent and/or offspring should take the possible effect of a second parent with an emotional disorder into consideration. In this context it must also be noted that the effect of paternal depression may manifest itself differently in sons (Cummings & Davies, 1994; Marmorstein, Malone, & Iacono, 2004). For instance, Rohde et al. (2005) found that sons of depressed fathers had higher rates of suicidal ideation and suicide attempts than sons of depressed mothers and daughters of depressed fathers or mothers. Moreover, sons were more likely than daughters to develop substance abuse or dependency.

Our results must be considered in the light of four additional limitations. The first limitation is that the offspring in our study were 16-25 years old. Therefore, many may not
have developed anxiety or -in particular- depression yet. This can lead to underestimation of associations. Secondly, information about parental psychiatric history was based solely on the report of 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 without and offspring with a second parent with life-time emotional problems and consequently lead to underestimation of effects. Thirdly, possible differences between groups in severity, chronicity and timing of parental disorder may have affected the strength of associations. The literature suggests that offspring confronted with parental depression early in life and offspring of chronically depressed and/or highly impaired parents are at increased risk (Hammen & Brennan, 2003; Warner, Mufson, & Weissman, 1995). A final limitation is that our data was cross-sectional. We assumed a uni-directional (genetically and environmentally mediated) effect of parental disorder on offspring mental health. However, associations between parental and offspring mental health may have bi-directional origins (Ge et al., 1995), which can only be detected in longitudinal designs.

The experience of depression or anxiety in adolescence or young-adulthood often not only forebodes recurrent episodes in later life, but is also associated with difficulties in the areas of social relationships, education and work. The present paper indicates that offspring risk of depression and anxiety increases when the female gender is involved, either in the form of a daughter or a depressed mother. Early recognition of symptoms reminiscent of depression or anxiety in childhood and appropriate intervention seem important for offspring of depressed parents, particularly for daughters. However, to make this possible, clinical practice should more often inquire after patients’ children, especially when it concerns mothers.