The sensitive sex
Bouma, Esther Maria Corina

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Stressful life events and depressive problems in early adolescent boys and girls: the influence of parental depressive symptoms, temperament and family environment.

This chapter is based on
Bouma, Ormel, Verhulst, Oldehinkel

‘A women under stress is not immediately concerned with finding solutions to her problems but rather seeks relief by expressing herself and being understood.’

John Gray
ABSTRACT
Stressful life events increase the probability of depressive symptoms in early adolescence. Several genetic and environmental risk factors may change individual sensitivity to the depressogenic effect of these events. We examined modification by parental depression and gender, and mediation of the former by temperament and family environment. Data were collected as part of a longitudinal cohort study of (pre)adolescents (n = 2127). During the first assessment wave at approximately age 11, we assessed parental depression, family functioning, perceived parenting behaviours, and temperamental frustration and fearfulness. At the second wave, about two and a half years later, stressful life events between the first and second assessment were assessed. Depressive problems were measured at both waves. Adolescents with parents who had a (lifetime) depressive episode were more sensitive to the depressogenic effect of stressful events than adolescents without depressed parents (B = .012, p <.001). Furthermore, girls were more sensitive to these effects than boys (B = 0.39, p <.001). The modifying effect of parental depression was not mediated by temperament, family functioning and perceived parenting. Life events were assessed without consideration of contextual information. Depressive problems were measured by questionnaires that did not directly represent DSM-IV criteria. The measure of parental depression was unspecific regarding severity and timing of depressive episodes.
INTRODUCTION

Depressive symptoms in adolescence can be persistent and recurrent (Angst et al., 2000) and represent a potential cause for psychopathology and poor psychosocial adjustments in adult life (Goodyer et al., 2000). The experience of stressful life events is a well-established risk factor for the development of depressive symptoms in adolescence and adulthood (Kendler et al., 1995; Kessler 1997; Larson and Ham 1993; Goodyer et al., 2000; Ormel et al., 2001; Rutter et al., 2000; Silberg et al., 2001). Whether or not stressful life events trigger depressive symptoms depends on adolescents’ sensitivity to stress, which is influenced by a variety of genetic and environmental risk factors. We propose that parental depression is one of the risk factors that may amplify the association between stressful life events and depressive symptoms in adolescence.

Depressive symptoms run in families (e.g. Goodman and Gotlib 1999), which may be due to transmitted vulnerability genes as well as to offspring exposure to an adverse and non-supportive family environment. From twin, adoption and family studies we know that depression is moderately heritable (e.g. Kendler and Prescott 1999; Kendler et al., 1999; Sullivan et al., 2000), and that genetic factors may modify the association between life events and depression (Kendler et al., 1995; Silberg et al., 2001). Consistent with this, recent molecular genetic work suggests that a functional polymorphism in the promotor region of the serotonin transporter gene moderates the depressogenic effects of stressful life events (Caspi et al., 2003; Eley et al., 2004; Jacobs et al., 2006; Zammit and Owen, 2006). Based on these findings we hypothesise that parental depression would amplify the effect of stressful life events on adolescents’ depressive symptoms.

Furthermore, individual differences in sensitivity to stressful life events may be explained by differences in negative affectivity. Negative affectivity, a temperament dimension encompassing frustration and fearfulness, reflects the tendency to experience negative emotions when confronted with environmental challenges (Rothbart et al., 2000). Several studies (e.g. Lonigan et al., 2003), including work from our own group (Oldehinkel et al., 2006) have revealed associations between negative affectivity and depressive symptoms in adolescence. Negative affectivity and depressive symptoms share genetic risk factors (e.g. Kendler et al., 2006). Ormel et al., (2005) showed that the effect of parental psychopathology on offspring psychopathology was partly mediated by preadolescent temperament. Considering this, we expected that (part of) the postulated modification of the depressogenic effect of stressful life events by parental depression would be mediated by temperamental frustration and fearfulness.
The modifying effect of parental depression could also be mediated by family environmental factors. Lack of family closeness, poor communication, absence of supportive relationships, parental rejection and little emotional warmth have all been found to be prevalent in families with depressed parents, and are associated with an increased risk of emotional and behavioural problems in offspring (e.g. Beardslee et al., 1996; Davies and Windle, 1997; Pilowsky et al., 2006). The stress-buffering model (Cohen and Wills, 1985) proposes that, when faced with negative experiences, individuals with greater support from families and friends are less likely to become depressed. Conversely, poor family circumstances may increase the impact of stressful life events. Hence, family environmental factors could also mediate (part of) the postulated modification of the depressogenic effect of stressful life events on adolescents’ depressive symptoms.

Prevalence of depressive symptoms, number and kind of stressful events, and sensitivity to stress, are all different for males and females (e.g. Brown and Harris 1978; Sherrill et al., 1997). The transition into adolescence seems to be the starting point for an increase in depressive symptoms in girls (Angold et al., 1998), probably set off because of a variety of gender specific hormonal and social developments (Larson and Ham, 1993; Cyranowski et al., 2000; Ge et al., 2001; Silberg et al., 2001). Therefore, associations between stressful life events and adolescents’ depressive symptoms cannot be studied without taking into account the possibility of gender differences in each of the effects examined.

The aim of this study was to explore if parental depression and gender modified the sensitivity to the depressogenic effect of stressful events in a large population-based sample of early adolescents. In addition, we investigated if the effect of parental depression was mediated by temperamental frustration and fearfulness, and by family environmental factors, more specifically, perceived parental emotional warmth and rejection and family functioning. The hypothesised associations are presented in Figure 1.

**Figure 1.** Schematic representation of the hypothesised associations.
METHODS

Sample
TRAILS: The TRacking Adolescents’ Individual Lives Survey is a large prospective population study of Dutch adolescents who are measured biennially until at least 25 years of age. The present study involves data from the first and second assessment wave of TRAILS, which ran from, respectively, March 2001 to July 2002, and September 2003 to December 2004. At the first wave, 2230 children were enrolled in the study of which 2149 children participated in the second wave. The mean age at T1 was 11.09 (SD = 0.56), and 50.8% were girls. The mean age at T2 was 13.6 (SD = 0.53), and 51.2% were girls. For more details see De Winter et al., (2005).

Measures
Measures used in this study come from the TRAILS Family Interview held with one of the parents (usually the mother, 95.6%) at the first assessment wave (T1) and self-report questionnaires filled out by parents and adolescents at both the first and second assessment wave (T1 and T2).

Depressive symptoms. Mental health problems were assessed by the Child Behavioral Checklist (CBCL) parent report form (Achenbach 1991b; Verhulst and Achenbach 1995). The CBCL contains a list of 120 behavioural and emotional problems, which parents can rate as 0 = not true, 1 = somewhat or sometimes true, or 2 = very or often true in the past six months. In addition to the CBCL we administered the self-report version of this questionnaire, the Youth Self Report (YSR, Achenbach 1991a). We used the CBCL/YSR Depressive Problems scale (Achenbach et al., 2003), which contains of 13 items (Cronbach’s α T1 CBCL = 0.68, T2 CBCL= 0.73, T1 YSR = 0.77, T2 YSR = 0.77) covering depressed mood, anhedonia, loss of energy, feelings of worthlessness and guilt, suicidal ideation, sleep problems and eating problems. The mean of the standardised parent and adolescent scores was used as a measure of depressive symptoms in this study since a combined score is less sensitive to context and perspective (Kraemer et al., 2003). When information of one informant was missing or unreliable (YSR: T1 n = 34, T2 n = 36, CBCL: T1 n = 153, T2 n = 205) the composite score was based on only one informant.

Stressful life events were assessed at T2 by a questionnaire containing 36 events, of which 25 were stressful. Examples of these events are parental divorce or death, severe illness/death of family members, serious quarrel with a friend, romantic break-up and victimization. (For all events see Appendix III, Chapter 2). The items had a yes/no format to indicate whether or not the event had occurred in the last
two years. Of the 2127 adolescents included in our analysis, 78.2% experienced at least one stressful life event in this period. The severity of an event was rated on a four-point scale ranging from 0 = not unpleasant to 3 = very unpleasant. Of all the events included 39% were categorised as very unpleasant, 28% as unpleasant and 33% as mildly unpleasant. Events that were relatively often rated as very unpleasant were; serious illness, death of a dear one, parental divorce, and conflicts with and between family members. The other events were more or less equally divided across the severity categories. The scores of all 25 events were summed to create a total severity score. Because this total score was highly skewed and we did not expect meaningful differences between low and average levels of stress regarding their association with depressive symptoms, we dichotomise this variable into low/average stress (score \(\leq 6\): 81.4% of the participants) and high stress (score > 6: 18.6% of the participants).

*Parental depressive symptoms* were assessed by means of the TRAILS Family History Interview (FHI). A brief description of the main symptoms of depression was given, followed by a series of questions to assess (lifetime) prevalence, treatment, and presence during the past year. Depressions characterized by professional treatment, medication use, or episodes in the last year were given extra weight, because of their greater (assumed) severity and/or impact on the adolescents. For each parent, parental depression was scored as 0 = no depression, 1 = depression without treatment/medication and last episode more than a year ago, and 2 = depression with treatment/medication or episode in the last year. In our dataset, 14.6% males and 26.8% females had experienced a depressive episode. These numbers are comparable to the DSM-IV lifetime rates (15.4% males and 27.4% females) obtained by CIDI-interviews in NEMESIS, a large population study (Bijl et al., 1998) and in the European Study of Epidemiology of Mental Disorders (2004). We summed the data of both parents which resulted in a parental depression score that ranged from 0 to 4. Missing values (father \(n = 53\), mother \(n = 65\)) were imputed by group means.

*Temperament* was assessed at T1 by the parent version of the short form of the Early Adolescent Temperament Questionnaire Revised (EATQ-R, Hartman 2000, Putnam et al., 2001). Fearfulness (five items, Cronbach’s \(\alpha = 0.63\)) denotes worrying and unpleasant affect related to the anticipation of distress. Frustration (five items, Cronbach’s \(\alpha = 0.74\)) is indicative of negative affect related to interruption of ongoing task or goal blocking.

*Parenting.* At T1, perceived parenting behaviour were assessed with the EMBU-C (Markus et al., 2003), the child version of the EMBU (a Swedish acronym for My Memories of Upbringing, developed by Perris et al., 1980). In the present study, we
used the scales Rejection and Emotional Warmth. The Rejection scale contains 12 items with Cronbach’s $\alpha = 0.84$ for fathers and 0.83 for mothers. The Emotional Warmth scale contains 18 items with Cronbach’s $\alpha = 0.91$ for both parents. The associations for father and mother were high, both for rejection ($r = .68$, $p < .001$) as well as for emotional warmth ($r = .79$, $p < .001$). Therefore we combined them into a single measure. When information of one informant was missing or unreliable (Emotional Warmth: father $n = 70$, mother $n = 30$, Rejection: father $n = 72$, mother $n = 31$) the composite score was based on only one informant.

*Family functioning* was assessed at T1, by a modified version of the General Functioning Scale of the McMaster Family Assessment Device (FAD, Epstein et al., 1983). This scale assesses six dimensions of family functioning; communications, problem solving, affective responsiveness, affective involvement, roles and behaviour control. The modified scale consists of 12 items with a Cronbach’s $\alpha = 0.85$. Parents could rate their agreement on a 4-point scale; 1 = totally disagree, 2 = disagree, 3 = agree, 4 = totally agree.

**Statistical analysis**

All continuous variables were standardised to a mean of 0 and a standard deviation of 1 to obtain internally comparable regression coefficients. Interaction terms were created by multiplying the standardised scores. A p-value smaller than .05 was considered statistically significant. First, means of and correlation between variables were calculated and gender differences in means and percentages were analysed by t-tests and $\chi^2$-square tests respectively. After that, we examined modification of the relationship between stressful events and depressive symptoms by parental depression and gender. In case of a significant interaction of parental depression and stressful life events, we subsequently examined mediation of this effect by fearfulness, frustration, rejection, emotional warmth and family function.

Main and interaction effects of stressful life events, parental depression and gender on adolescent depressive symptoms were investigated by means of a backward stepwise regression analysis with T2 depressive symptoms as dependent variable. The initial model includes all main and interaction effects of stressful life events, parental depression and gender. First, we examined the three-way interaction between stressful life events, parental depression and gender (a significant interaction would imply that the mediation analyses had to be stratified by gender). If not significant, this interaction was dropped from the model. In the second step, non-significant two-way interactions were dropped from the model. The last step examined the main effects. Model terms could only be dropped if the variables were not included in higher-order terms. To examine to what extent the effect of stressful life events might be due to pre-event depressive symptoms, we repeated
the final analysis adjusting for T1 depressive symptoms, by including this variable in the model.

If parental depression significantly modifies the effects of stressful life events on adolescent depressive symptoms, this effect may be mediated by temperament or family-related risk factors. Mediation of an association between a determinant and an outcome can occur only if the mediating variable is associated with both factors, follows the determinant in time and precedes the outcome (Baron and Kenny 1986). In our case, we did not investigate simple mediation, but mediated modification (that is mediation of the modifying effect of parental depression on the association between stressful life events and depressive symptoms). Consequently, our mediators should still be significantly associated with parental depression, but rather than being associated with depressive symptoms, their interaction with stressful life events should be significant. A factor that mediates the effect of parental depression, should be related to sensitivity to stressful life events itself too, that is, increase the effect of life events on depressive symptoms. Putative mediators were only included in subsequent analyses when both associations were found.

To assess the amount of mediation, the (direct) interaction effect of parental depression and stressful events was compared with the interaction effect after including the (main and interaction) effects of the mediator in the model. The larger the relative reduction in the regression coefficient in the interaction between parental depression and stressful life events, the more this effect was mediated by the variable under study.

**RESULTS**

**Descriptive statistics**
Mean scores or percentages of the variables used are shown in Table 1. For descriptive purposes, we presented the mean of the unstandardised total scores of parent and self-reported adolescent depressive symptoms rather than the mean of the standardised scores.

**Bivariate associations**
Correlations between the variables in the study were generally low to moderate (Table 2). In both sexes, depressive symptoms at T2 were significantly associated with all other variables.
The depressogenic effect of stressful life events

Modification of the stress-depressive symptoms association by parental depression and gender

The three-way interactions of stressful events, parental depression and gender was not significant (B = -0.09, p = .36), indicating that the interaction of stressful life events and parental depression was comparable for boys and girl, and that mediation analysis did not have to be stratified by gender. The two-way interaction of parental depression and gender was not significant either (B = 0.02, p = .61).

Parental depression and gender both significantly moderated the association between stressful life events and depressive symptoms. Sensitivity to stress, as indicated by the interaction variable between stressful life events and parental depression, was larger in adolescents whose parents have a history of depression than in adolescents whose parents have no such history. Furthermore, the significant interaction between stressful life events and gender indicated that girls are more sensitive to this effect than boys. Coefficients of the final model are shown in Table 3. These results are visualised in Figure 2 in which the estimated depressive symptoms for boys and girls are shown, both in absence and presence of parental depression and stressful life events.

We performed a posthoc-analyses to examine the effect of stressful life events in absence of parental depression by adding parental depression as unstandardised variable to the model (B = 0.32, p = < .001). This effect was stronger for girls than for boys (B = 0.27, p = .004).

Adjusted for depressive symptoms at T1 (i.e. before the stressful events) the main effects of gender and parental depression were still significant, as well as the interaction between stressful life events and gender (B = .27, p = .01). The interaction between stressful life events and parental depression was no longer significant but showed a trend in the expected direction (B = 0.07, p = .074).

### Table 1. Descriptive statistics

<table>
<thead>
<tr>
<th></th>
<th>Boys</th>
<th>Girls</th>
<th>Gender difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>mean</td>
<td>n</td>
</tr>
<tr>
<td>Dep. problems T1</td>
<td>1027</td>
<td>3.13</td>
<td>1080</td>
</tr>
<tr>
<td>Dep. problems T2</td>
<td>1033</td>
<td>2.47</td>
<td>1075</td>
</tr>
<tr>
<td>Stress (0.1)</td>
<td>1039</td>
<td>14.8%</td>
<td>1088</td>
</tr>
<tr>
<td>Parental depression</td>
<td>1007</td>
<td>0.80</td>
<td>1060</td>
</tr>
<tr>
<td>Fearfulness</td>
<td>930</td>
<td>2.34</td>
<td>976</td>
</tr>
<tr>
<td>Frustration</td>
<td>931</td>
<td>2.83</td>
<td>976</td>
</tr>
<tr>
<td>Emotional warmth</td>
<td>1027</td>
<td>3.17</td>
<td>1082</td>
</tr>
<tr>
<td>Rejection</td>
<td>1027</td>
<td>1.51</td>
<td>1081</td>
</tr>
<tr>
<td>Family functioning</td>
<td>957</td>
<td>2.49</td>
<td>1002</td>
</tr>
</tbody>
</table>

* = Degrees of freedom not equal to n - 1 due to correction for unequal variances.
### Table 2. Bivariate associations

<table>
<thead>
<tr>
<th>Predictor</th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
<th>6.</th>
<th>7.</th>
<th>8.</th>
<th>9.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress (0 = low, 1 = high)</td>
<td>-</td>
<td>.05</td>
<td>.16</td>
<td>.31</td>
<td>.10</td>
<td>.04</td>
<td>.09</td>
<td>.05</td>
<td>.04</td>
</tr>
<tr>
<td>Parental depression</td>
<td>.03</td>
<td>.21</td>
<td>.17</td>
<td>.01</td>
<td>.04</td>
<td>.09</td>
<td>.10</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>Depr. problems T1</td>
<td>.14</td>
<td>.19</td>
<td>.52</td>
<td>.37</td>
<td>.22</td>
<td>.31</td>
<td>.22</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>Depr. problems T2</td>
<td>.19</td>
<td>.18</td>
<td>.60</td>
<td>.28</td>
<td>.16</td>
<td>.19</td>
<td>.12</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td>Rejection</td>
<td>.12</td>
<td>.04</td>
<td>.32</td>
<td>.22</td>
<td>-.33</td>
<td>.26</td>
<td>.24</td>
<td>.03</td>
<td></td>
</tr>
<tr>
<td>Emotional warmth</td>
<td>-.03</td>
<td>-.02</td>
<td>-.16</td>
<td>-.12</td>
<td>-.31</td>
<td>-.05</td>
<td>.02</td>
<td>.004</td>
<td></td>
</tr>
<tr>
<td>Fearfulness</td>
<td>.14</td>
<td>.16</td>
<td>.27</td>
<td>.20</td>
<td>.23</td>
<td>-.05</td>
<td>.43</td>
<td>.05</td>
<td></td>
</tr>
<tr>
<td>Frustration</td>
<td>.11</td>
<td>.13</td>
<td>.25</td>
<td>.23</td>
<td>.26</td>
<td>-.001</td>
<td>.43</td>
<td>.01</td>
<td>-.01</td>
</tr>
<tr>
<td>Family functioning</td>
<td>.04</td>
<td>.07</td>
<td>.05</td>
<td>.09</td>
<td>.03</td>
<td>.01</td>
<td>-.01</td>
<td>-.04</td>
<td></td>
</tr>
</tbody>
</table>

**Bold** = significant association at p < .05.  
*b* = point biserial correlations for associations between a continuous and a dichotomous variable.  
*Note:* Girls’ correlations are printed above the diagonal; boys’ correlations below the diagonal.

### Table 3. Effect modification by parental depression and gender

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress (0 = low, 1 = high)</td>
<td>.39</td>
<td>.001</td>
</tr>
<tr>
<td>Parental depression</td>
<td>.39</td>
<td>.001</td>
</tr>
<tr>
<td>Gender (0 = boys, 1 = girls)</td>
<td>.12</td>
<td>.28</td>
</tr>
<tr>
<td>Stress*parental depression</td>
<td>.25</td>
<td>.04</td>
</tr>
<tr>
<td>Stress*gender</td>
<td>.26</td>
<td>.01</td>
</tr>
</tbody>
</table>
| Dependent variable: depressive problems at T2. Note: \( R^2 = .113 \) (S.E. \( = .81 \))
The depressogenic effect of stressful life events

Figure 2. Estimated problems for boys and girls.  
Note: PD = parental depressive symptoms.

Analysis of mediation
Because the interaction between parental depression and stressful events did not show gender differences, mediation analyses were not stratified by gender. One of the prerequisites of mediation was that the mediator had to be associated with parental depression. Only frustration, fearfulness and family functioning met this first criterion (see Table 2.). However, none of these factors met the second prerequisite and were not included in further analyses. Interesting to note is that rejection did meet the second prerequisite for mediation; the interaction between rejection and stressful life events was significantly associated with depressive symptoms (B = 0.126, p = .003).

DISCUSSION
The first goal of this study was to explore if the relationship between stressful life events and depressive symptoms in adolescence was modified by parental depression and gender in a large population sample of Dutch early adolescents. Secondly, we wanted to investigate if the effect of parental depression was mediated by temperamental frustration and fearfulness, and by family environmental factors. Consistent with our expectations and other studies (e.g. Goodyer et al., 2000; Larson and Ham, 1993), stressful life events were
associated with depressive symptoms in both boys and girls, but considerably stronger in girls than in boys. Furthermore, we found that adolescents whose parents had a history of depression reported more depressive symptoms after the occurrence of stressful life events than adolescents whose parents did not have such history. This effect of parental depression was not mediated by temperament or family environment factors included in this study.

The finding that parental depression was associated with a greater offspring sensitivity to stressful life events is in line with results reported by Silberg and colleagues (2001), despite differences in study design (twin versus population sample, parent versus child report, only girls versus both genders, age differences). This adds to the evidence that familial risk of depression is likely to be expressed by increased stress-sensitivity. A difference between Silberg et al.‘s and our study is that they did not find an association between stressful life events and depression in the absence of parental emotional disorders, while we also found an effect in the group of adolescents whose parents have never had depressive symptoms. Our findings converge with molecular genetic evidence (e.g. Caspi et al., 2003) that genetic polymorphisms contribute to individual differences in sensitivity to stressful experiences.

Children from parents who have a history of depression may be at risk for developing emotional problems not only because of a genetically predisposed sensitivity to stress but also because of the possibility that they experience more stressful events than children from parents without such history. In our sample, however, parental depression was not significantly associated with the number of stressful life events, which could be due to the relatively large proportion of parents who have a history of depression but had no depressive symptoms during the study period.

The association between stressful events and depressive symptoms was much stronger for girls than for boys, both with and without adjusting for pre-existing depressive symptoms. This could indicate that girls become more sensitive to the depressogogenic effects of stressful life events during the transition from childhood to adolescence as suggested several times (e.g. Cyranowski et al., 2000; Silberg et al., 2001). It should be noted, however, that the gender difference in depressive symptoms during early adolescence, was due to a decrease of depressive symptoms in boys, rather than an increase in girls, compared to preadolescent levels. The lack of increase in depressive symptoms in girls in our sample might suggest that their sensitivity to events did not change, while boys became less sensitive. Data from subsequent waves will make it possible to explore this further.
To the best of our knowledge, no one has analysed mediated modification of the stress-depressive symptoms association by parental depression. There are only three studies that examined mediation of the (main) effects of parental psychopathology on offspring psychopathology. Davies and Windle (1997) found that family discord was a modest mediator of the effect of maternal depressive symptoms on middle-adolescent’ depressive symptoms. Ormel et al., (2005), using the same sample as described in the present study, found that one third of the effect of familial loading of parental psychopathology on internalising and externalising problem behaviour in offspring was mediated by offspring temperament. Recently, Burt et al., (2006), reported that parenting and family environmental factors partially mediated the association between parental psychopathology and offspring psychopathology in late adolescence. Although these studies involved mediation of the main effect of parental psychopathology on offspring problem behaviour instead of mediated modification, they seem to provide indirect support that the effect of parental depression on adolescents’ sensitivity to stressful events is mediated by temperament and family environment. Yet we did not find evidence for such mediation. Tentatively, we propose a number of factors that could have contributed to these results.

There were no significant associations between the parental depression measure and the family-related putative mediators emotional warmth, parental rejection, and poor family functioning. This could be due to the fact that our parental depression measure yielded a highly heterogeneous group, regarding both severity of the depressive symptoms and their timing. Mild or long remitted depressive symptoms are not very likely to affect current parenting and family functioning. Our results suggest that the association between parental depression and parenting may be less pronounced or even absent in population samples. Indeed, recent prospective studies found no stress-buffering effects of family support (e.g. Burton et al., 2004).

The absence of associations could also signify methodological weakness of the parental depression measure, which was based on retrospective report. However, this is contradicted by the finding that the parental depression measure was strong enough to moderate the association between stressful events and offspring depressive symptoms. Although we did not evidence for family related factors, it is still possible that parental depression influenced the impact of stressful life events through other family difficulties which were not included in this model.

Temperamental frustration and fearfulness did not mediate the interaction between parental depression and stressful life events either. Tentatively, negative affectivity reflects individual differences in reactivity to daily hassles better than reactivity to more severe and rare life events (Zammit and Owen, 2006). Frustration and
fearfulness may be more relevant with regard to minor provocations, annoyances, and threats than to the bigger facts of life.

Our study has important assets: it was based on a large prospective population sample of pre- and early adolescents and it used multiple informants which decreased the risk of inflated associations. The prospective design allowed us to adjust for depressive symptoms already present before the stressful events occurred, and revealed that the interaction of gender and life events remained significant and that the interaction of parental depression and life events still showed a trend in the expected direction.

There are also limitations. First of all, life events were assessed retrospectively and based on self-reports instead of interview ratings taking into account contextual information (Brown and Harris 1978). Individuals with depressive symptoms tend to over-report number as well as severity of stressful life events ful events (Brewin et al., 1993). Second, depressive symptoms were based on questionnaire data. The CBCL/YSR Depressive Problems scale was not developed to assess depressive symptoms according to DSM-IV criteria, but constructed on the basis of expert ratings of the original, empirically derived, CBCL and YSR scale items. Consequently, the items do not represent one-to-one counterparts with all DSM-IV criteria. Third, as mentioned above, parental depression was assessed retrospectively. Despite these limitations, this study provides an excellent starting point for future research of the mechanisms by which parental depression may be associated with increased stress-sensitivity in children of depressed parents.

To conclude, offspring of depressed parents have a higher stress-sensitivity than offspring of non-depressed parents. This highlights the importance of monitoring children from high-risk families, particularly when they are exposed to high amounts of life stress. Furthermore, early adolescent girls have an increased stress-sensitivity compared to boys. It is important to extend our knowledge about this widely observed gender difference, to provide the best, possibly differential, care for adolescent boys and girls with depressive symptoms. More research is needed on mechanisms by which parental depression influences offspring's increased stress-sensitivity.