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Childhood adversities and adolescent depression: A matter of both risk and resilience

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Abstract
Childhood adversities have been proposed to modify later stress sensitivity and risk of depressive disorder in several ways: by stress sensitization, stress amplification, and stress inoculation. Combining these models, we hypothesized that childhood adversities would increase risk of early, but not later, onsets of depression (Hypothesis 1). In those without an early onset, childhood adversities were hypothesized to predict a relatively low risk of depression in high-stress conditions (Hypothesis 2a) and a relatively high risk of depression in low-stress conditions (Hypothesis 2b), compared to no childhood adversities. These hypotheses were tested in 1,584 participants of the Tracking Adolescents’ Individual Lives Survey, a prospective cohort study of adolescents. Childhood adversities were assessed retrospectively at ages 11 and 13.5, using self-reports and parent reports. Lifetime DSM-IV major depressive episodes were assessed at age 19, by means of the Composite International Diagnostic Interview. Stressful life events during adolescence were established using interview-based contextual ratings of personal and network events. The results provided support for all hypotheses, regardless of the informant and timeframe used to assess childhood adversities and regardless of the nature (personal vs. network, dependent vs. independent) of recent stressful events. These findings suggest that age at first onset of depression may be an effective marker to distinguish between various types of reaction patterns.
Childhood adversities are associated with an increased risk to develop a (incident) depressive episode. The relative risk is particularly high in childhood and decreases over time.

Hypothesis 2: Adolescents who survived childhood adversities without getting depressed in childhood or early adoles-

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The relative risk is particularly high in childhood and decreases over time.
cidence (in this study defined as the period until the age of 16) have, compared to adolescents exposed to few adversities: (a) a lower probability to develop a depressive episode in current high-stress conditions and (b) a higher probability to develop a depressive episode in current low-stress conditions.

Note that, although these hypotheses were developed by combining aspects of the SS, SA, and SI models, they cannot be used to test any of these three models directly.

A number of features make our study particularly suitable for investigating long-term effects of childhood adversities on sensitivity to recent stressful events and depression risk: a follow-up period of over 10 years, multi-informant ratings of childhood adversities during various periods, information about the lifetime occurrence of DSM-IV major depressive episodes and their age of onset, and meticulous assessments of stressful events occurring between the ages of 16 and 19 through contextual, interviewer-based ratings.

Methods

Sample

The data were collected as part of the Tracking Adolescents’ Individual Lives Survey, a prospective cohort study of Dutch adolescents (Ormel et al., 2012). Four assessment waves have been completed to date, which ran from March 2001 to July 2002 (Time 1 [T1]), September 2003 to December 2004 (Time 2 [T2]), September 2005 to August 2007 (Time 3 [T3]), and October 2008 to September 2010 (Time 4 [T4]). The study was approved by the Dutch Central Committee on Research Involving Human Subjects. Participants were treated in compliance with American Psychological Association ethical standards, and all measurements were carried out with their adequate understanding and written consent.

At T1, 2,230 (pre)adolescents were enrolled in the study (response rate 76%, mean age = 11.1, SD = 0.6, 51% girls; De Winter et al., 2005), of whom 96% (N = 2,149, mean age = 13.6, SD = 0.5, 51% girls) participated at T2. The response rates at T3 and T4 were, respectively, 81% (N = 1,816, mean age = 16.3, SD = 0.7, 52% girls) and 83% (N = 1,881, mean age = 19.1, SD = 0.6, 52% girls).

The fourth assessment wave included questionnaires, a psychiatric diagnostic interview, and a life events interview. Because the life events interview was costly and labor intensive, it was only administered to part of the sample, in which respondents with a psychiatric diagnosis were oversampled. Of all T4 participants, 84% (N = 1,584) agreed to have the diagnostic interview, 93% of whom gave consent for a life events interview. Of these, 45% (n = 659) met DSM-IV criteria for a psychiatric disorder during the past year. These adolescents were all selected for the life events interview, and 580 (89%) were actually interviewed; for 11% an interview was not feasible within the study period owing to practical constraints. Of the adolescents without a past-year diagnosis (n = 808), 49% were selected for the life events interview and 47% (n = 377) were actually interviewed, yielding a total of 957 interviews (mean age = 19.1, SD = 0.6, 55% girls). As compared to the part of the T4 sample that did not get the life events interview, participants were more often females (55% vs. 50%; χ² = 4.5, p = .04); were younger (mean age = 19.0, SD = 0.6 vs. 19.2, SD = 0.6), t(df = 1,879) = 6.3, p < .01; and reported more depressive symptoms (mean item score 0.33, SD = 0.31 vs. 0.26, SD = 0.29); t(df = 1,694) = −4.4, p < .01, as assessed with the Adult Self-Report (Achenbach & Rescorla, 2003).

Measures

Major depression. The presence of psychiatric disorders was assessed during T4, by means of the World Health Organization Composite International Diagnostic Interview (CIDI), version 3.0. The CIDI is a structured diagnostic interview that yields lifetime and current diagnoses according to DSM-IV (American Psychiatric Association, 2000). The CIDI has been used in a large number of surveys worldwide (Kessler & Ustun, 2004), and it has shown good concordance with clinical diagnoses (Haro et al., 2006; Kessler et al., 2009).

Depression was operationalized as a major depressive episode (MDE). In addition to the lifetime occurrence of an MDE, the CIDI also yields information with regard to the age of first onset, the age at which the last episode started, and the age at which the last episode ended.

Childhood adversities. Exposure to childhood adversities was assessed at T2, by means of parent- and self-reported ratings of overall stressfulness of the child’s life between ages 0–5 and 6–11, respectively. Parents were asked, “How stressful was your child’s life in this phase?” and adolescents were asked, “How many bad things happened to you in this period?” The stressfulness was rated on an 11-point scale (0 = not at all, 10 = very much). Means and correlations of the individual adversity measures are given in Table 1. Based on these four adversity measures, we constructed a factor score (ML extraction) to capture the common core of these measures, which explained 42% of the variance in the individual measures. This factor score was used as the main predictor in subsequent analyses.

Recent stressful events. Stressful life events in the period between T3 and T4 were assessed with Kendler’s Life Stress Interview (LSI; Kendler, Karkowski, & Prescott, 1998), which was based on the Life Events and Difficulties Schedule (Brown & Harris, 1989). The LSI encompasses 11 personal events, that is, events occurring primarily to the respondents themselves, including assault, breakup of romantic relationship, illness or injury, trouble with police, loss of a confidant, and difficulties at work or school. In addition, there are 4 classes of events occurring primarily to an individual in the respondent’s social network (e.g., a serious crisis, illness, or death). Each reported stressful life event was dated as accurately as possible by means of mnemonic aids such as personal calendars. A distinguishing
feature of the LSI is that the events are not rated by the respondent, but by the interviewer. Furthermore, the ratings are contextual, that is, based on what most people would feel about an event given the circumstances and biography, taking no account of respondents’ reaction or any following mental health problems. Interviewer-based contextual ratings are essential to prevent intracategory variability and to disentangle objective event characteristics from the emotions and behaviors evoked by the event (Dohrenwend, 2006).

For each event in the time period between T3 and T4, we rated the severity (i.e., long-term contextual threat) and dependence on respondent’s will or behavior (i.e., planned actions or events directly caused by neglect or carelessness). Severity ratings ranged from 1 = minor to 4 = severe; possible dependence ratings were 1 = clearly independent, 2 = probably independent, 3 = probably dependent, and 4 = clearly dependent. Examples of clearly independent events are death or disease of someone, while breaking up a relationship and being caught for robbery are clearly dependent events. Events like burglary, being discharged, and abuse will often be rated as probably independent or dependent, depending on their specific context. All interviewers were extensively trained and regularly attended booster sessions in order to ensure reliable and valid scores. Furthermore, all interviews were tape-recorded and scored by a second rater blind to the interviewer’s scores. In case of discordant ratings, the two raters discussed the scores until consensus was reached or a third rater made the final judgment.

For the depressed adolescents, we included all events that occurred in the year of the depression onset and the preceding year. The time frame for the life events in the control group also spanned 2 years, and it was chosen in such a way that the distribution of the time lag to the T4 assessment equaled the distribution in the depressed group.

We calculated the summed severity of all events that occurred within the time frame as well as the summed severity of only the independent events, the personal events, and the network events. Descriptive statistics of these measures are presented in Table 2.

Analysis

We examined whether childhood adversities were associated with the onset of an MDE during childhood or adolescence, using a Cox proportional hazards regression model with age of onset of the first MDE as the dependent variable and the factor score representing adversities experienced between age 0 and 11 as the predictor. In addition to the main effect of childhood adversities, we included its interaction with age, to test the hypothesis that the effect of childhood adversities declines over time. Gender was included as a covariate. The analysis was repeated using the adversities measures pertaining to ages 0–5 and 6–11 (averaged across informants), respectively, and the parent reports and self-reports (averaged across timeframes) separately.

Then we examined whether childhood adversities were associated with depressive reactions to stressful life events during adolescence, operationalized as the association between recent stressful events and MDE onset. This was tested in a logistic regression model, with MDE onset as the dependent variable. We did not control for the oversampling of adolescents with a psychiatric diagnosis, because odds ratios are valid regardless of the distribution of the outcome variable. Predictor variables were childhood adversities (overall exposure from age 0 to 11), recent stressful life events prior to onset (total summed severity), and the interaction of the two. Gender was included as a covariate, and gender differences in the effect of any of the predictor variables or their interaction were examined by testing interaction effects, which were maintained in the model if significant. Adolescents who were depressed at T3 were excluded from these analyses, because the LSI did not cover a pre-onset period in their case. The re-

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean (SD)</th>
<th>Range</th>
<th>Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td>All events</td>
<td>4.7 (4.6)</td>
<td>0–10</td>
<td>.15 .26 .43</td>
</tr>
<tr>
<td>Summed severity personal events</td>
<td>2.3 (2.9)</td>
<td>0–23</td>
<td></td>
</tr>
<tr>
<td>Summed severity network events</td>
<td>2.4 (2.9)</td>
<td>0–21</td>
<td></td>
</tr>
<tr>
<td>Independent events</td>
<td>3.0 (3.3)</td>
<td>0–20</td>
<td></td>
</tr>
<tr>
<td>Summed severity personal events</td>
<td>0.8 (1.6)</td>
<td>0–12</td>
<td></td>
</tr>
<tr>
<td>Summed severity network events</td>
<td>2.2 (2.7)</td>
<td>1–19</td>
<td></td>
</tr>
</tbody>
</table>

Note: N = 875.
main sample was split into two subgroups: adolescents with and without an onset of depression before T3. Hypothesis 2 was tested in the group without an onset before T3 (i.e., at risk for a first onset), while the analysis was repeated in the group with a remitted onset before T3 (i.e., at risk for a recurrent onset) for the sake of comparison.

To examine the robustness of the findings with regard to the timing (0–5 vs. 6–11 years) and informant (parent vs. self-report) of the childhood adversities, as well as to the nature (dependent vs. independent) of the recent stressful events, the analysis was replicated using alternative measures. To ease interpretation of the odds ratios, all continuous predictor variables were standardized to a mean of 0 and a standard deviation of 1. The data were analyzed using PASW Statistics software, version 18.0.3.

Results

Childhood adversities and onset of depression

Of the 1,584 adolescents included in this study, 17% had a lifetime diagnosis of MDE. The Cox regression analysis revealed that the factor score representing childhood adversities strongly increased the probability of depression onset early in life (main effect adversities: $B = 2.88, SE = 0.20, p < .001$, hazard ratio $= 17.8$) and that the effect decreased with increasing age (interaction adversities by age: $B = -0.15, SE = 0.01, p < .001$, hazard ratio $= 0.9$) to hazard ratios near 1 in late adolescence (see Figure 1). These effects were the same regardless of the period (i.e., during age 0–11, 0–5, or 6–11 years) and informant (i.e., factor score, parent reports, or self-reports) of the childhood adversities. Comparable results (main effect adversities: $B = 2.80, SE = 0.24, p < .001$; interaction with age: $B = -0.14, SE = 0.02, p < .001$) were found in the subsample of 957 adolescents who participated in the life events interview.

Childhood adversities and sensitivity to recent stressful events

The LSI was administered to 957 adolescents, of whom 801 had not experienced a depression onset before T3. This group was used to test Hypothesis 2. Forty-three adolescents developed a first onset of depression between T3 and T4. Logistic regression analysis revealed a significant negative interaction between the effects of childhood adversities and recent stressful events, indicating that the association between recent stressful life events and MDE onset was weaker in adolescents who had been exposed to high adversity levels during childhood than in those exposed to low adversity levels (Table 3). Inspection of the (joint) distribution of childhood adversities and recent life events indicated that the negative interaction effect was not driven by any outliers. Furthermore, none of the effects differed significantly between boys and girls (all $ps > .19$).

This pattern of associations was found regardless of the time frame (0–5 vs. 6–11 years) or informant (parent vs. self-report) of the childhood adversities, and regardless of the nature of the recent events (personal vs. network, including or excluding person-dependent events): all measures yielded a nonsignificant main effect of childhood adversities, a significant main effect of recent life events and, and a significant ($p < .05$) negative interaction effect of childhood adversities and recent stressful life events (details available upon request). The estimated risk of MDE onset between T3 and T4, conditional on childhood adversities and recent stressful events, is depicted in Figure 2. As hypothesized, at recent low-stress conditions, the probability of depression onset was higher for adolescents exposed to many childhood adversities than for those exposed to few adversities, while at recent high-stress conditions, their probability was lower. The regions of significance ($p < .05$) for the effect of childhood adversities on depression onset covered recent stress levels smaller than $–0.76$ $SD$ below the mean (region of significant positive effect of childhood adversities on probability of depression) and larger than $+ 1.55$ $SD$ above the mean (region of significant negative effect). That the regions were asymmetrically dispersed around the mean is largely due to the skewed distribution of recent life events (Table 2). Simple slopes analyses revealed that recent stressful life events significantly predicted depression onset in adolescents exposed to low ($B = 0.95, SE = 0.20, p < .001$) and mean ($B = 0.55, SE = 0.14, p < .001$) childhood adversity levels, but not in those exposed to high levels of childhood adversity ($B = 0.14, SE = 0.20, p = .48$).

To examine if the negative interaction between childhood adversities and recent life events was specific for adolescents without an early depressive episode, we repeated the analysis for the 74 adolescents who had experienced a depressive episode before T3 but were nondepressed (and so at risk for a re-
current episode) at T3. Of these adolescents, 28 developed an episode between T3 and T4. Sample size differences prohibit a comparison between the two subsamples based on statistical significance, but the small size of the interaction effect ($B = -0.01, SE = 0.21, p = .96, odds ratio = 1.0$) strongly suggests that the modifying effect of childhood adversities did not pertain to the adolescents with an early onset of depression.

**Discussion**

How childhood adversities modify later stress sensitivity and risk of depressive disorder has been described by three seemingly conflicting theories: the stress sensitization, stress amplification, and stress inoculation models. In this study, we aimed to reconcile these models by emphasizing that all may apply, but in different persons and different circumstances. We hypothesized that childhood adversities would increase risk of depression during and shortly after the adversities, but have a waning effect on depression incidence over time (Hypothesis 1). In adolescents without an early onset, childhood adversities were hypothesized to decrease risk of depression in current high-stress conditions (Hypothesis 2a) and increase risk of depression in current low-stress conditions (Hypothesis 2b).

The results provided support for both hypotheses. More specifically, we found that childhood adversities strongly increased the probability of depression onset early in life and that this effect decreased over time. Furthermore, in the subgroup of adolescents without an early onset of depression, those who had been exposed to childhood adversities were less sensitive to the effects of recent stressful life events than were the nonexposed group, as evidenced by a lower probability of depression onset in current high-stress, and a lower probability in current low-stress conditions. This suggests that exposure to adversities either leads to early-onset depressive episodes or programs children for high-stress conditions later in life, at the expense of a relatively higher risk of depression in low-stress conditions. Early-onset depressions may reflect cognitive (Abela, 2001; Hankin & Abramson, 2001) or epigenetic (Essex et al., 2011; Meaney & Szyf, 2005) vulnerabilities causing stress sensitization, stress amplification, or both. At best, adolescents with an early-onset depression are not worse off, but they never seem better off than others. Adolescents who do not develop a depression during or shortly after the childhood adversities cannot be labeled as being at high or low risk in general. Rather, they seem to be optimally adapted to stressful environments, which they can stand better than can adolescents not exposed to childhood adversities (Del Giudice, Ellis, & Shirtcliffe, 2011; Ellis et al., 2011). The apparent price they paid is a relatively high risk of depression in current low-stress environments. Figure 2 suggests that adolescents who were exposed to childhood adversities show less sensitivity to their current environment, both in low-stress and high-stress situations. Given that low-stress environments tend to contain more constructive elements than do high-stress ones (Fergusson et al., 1984), adolescents exposed to childhood adversities may benefit less from the positive influences in their current environment (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007), resulting in lower positive affect.

Combined, our findings provide tentative support for Boyce and Ellis’s (2005) theory of biological sensitivity to

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE</th>
<th>p</th>
<th>Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (girls = 0, boys = 1)</td>
<td>-0.71</td>
<td>0.35</td>
<td>.04</td>
<td>0.5</td>
</tr>
<tr>
<td>Childhood adversities factor score ($z$)</td>
<td>0.09</td>
<td>0.16</td>
<td>.57</td>
<td>1.1</td>
</tr>
<tr>
<td>Recent life events ($z$)</td>
<td>0.55</td>
<td>0.14</td>
<td>&lt;.001</td>
<td>1.7</td>
</tr>
<tr>
<td>Childhood Adversities × Recent Events</td>
<td>-0.40</td>
<td>0.15</td>
<td>.006</td>
<td>0.7</td>
</tr>
</tbody>
</table>

*Note: N = 801.*

![Figure 2](image-url)
A number of limitations should be accounted for when interpreting the associations found. First, exposure to childhood adversities was assessed by retrospective reports of the overall stressfulness of the child’s life, which left much room for respondents’ (imminent) depressive symptoms during the assessment of childhood adversities influenced the ratings of the stressfulness of their childhood, most likely in such a way that the symptoms inflated the stressfulness ratings and hence the association between childhood adversities and depression. Confounding is probably limited, however, because childhood adversities were assessed 5 years before the psychiatric diagnostic interview, and the scores of two independent informants yielded a similar pattern of findings. The essential question to be answered is to which extent the retrospective evaluations of the childhood adversities might have influenced their interaction with recent stressful life events. The most probable report bias is that the childhood adversity ratings reflected (later) stress-vulnerability or sub-threshold depressive symptoms instead of mere exposure to environmental influences. In that case, we would expect a stronger effect of recent life events on depression in adolescents with high childhood adversity ratings. The negative interaction found is in the opposite direction and thus unlikely to be spurious. A second limitation is that the CIDI provides onset ages in years, which precluded a fine-grained analysis of temporal effects of events. The depressogenic effect of stressful events wanes over time and usually loses most of its power after a couple of months (Kendler et al., 1998; Wainwright & Surtees, 2002). The study design ensured that no depression onsets after stressful life events were missed, but it did not prevent the inclusion of events without a depression onset shortly afterward. If anything, this has affected the associations conservatively. The relatively crude dating of depression onsets also made it possible that some of the stressful events occurred after the onset of depression rather than before. Because the findings remained basically similar if we restricted the analyses to person-independent events only, however, reverse causality (depression causing the events rather than the other way around) is not very likely. Third, the life events measures covering childhood and early adolescence were not accurate enough to provide detailed information about the association between stressful events and early onsets of depression. This implies that the notion of increased stress sensitivity in children with an early onset of depression is an assumption that remains to be tested in future research. Fourth, to have unambiguous onset data we focused on depressive disorder only, and ignored possible co-occurring conditions such as anxiety and conduct disorder. Hence, concepts like risk and resilience only pertain to depression, and not to (mental) health and well-being in general.

In sum, our results suggest that there is no unequivocal relation between childhood adversity and depressive reactions to stressful life events during adolescence. Depressive history, notably the age at first onset of the problems, seems to be an effective marker to distinguish between various types of reaction patterns. Nevertheless, much remains to be learned about the actual conditions under which childhood adversities lead to later stress (in)sensitivity and depression. Another issue that deserves further study concerns the implications of (innate or acquired) insensitivity to envi-
Environmental influences for prevention or intervention strategies; possibly individuals characterized by environmental insensitivity require other strategies in order to be treated effectively than sensitive ones. Our study may thus contribute to the further development of evidence-based tailor-made interventions.

References


