Childhood abuse and the two-year course of late-life depression

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Abstract

Introduction: Late-life depression often has a chronic course, with debilitating effects on functioning and quality of life; there is still no consensus on important risk factors explaining this chronicity. Cross-sectional studies showed that childhood abuse is associated with late-life depression, and in longitudinal studies with chronicity of depression in younger adults. We aim to investigate the impact of childhood abuse on the course of late-life depression. Methods: Two-year longitudinal cohort study. Setting: Data were derived from the Netherlands Study of Depression in Older Persons (NESDO). Participants: 282 participants with a depression diagnosis in the previous 6-months (mean age 70.6 years), of whom 152 (53.9%) experienced childhood abuse. Measurements: Presence of childhood abuse (yes/no) and a frequency-based childhood abuse index (CAI) were calculated. Dependent variable was depression diagnosis after two-years. Results: Multivariable mediation analysis showed an association between childhood abuse and depression diagnosis at follow-up. Depression severity, age at onset, neuroticism and number of chronic diseases were important mediating variables of this association, which then lost statistical significance. For childhood abuse (yes/no), loneliness was an additional, significant mediator. Depression severity was the main mediating variable, reducing the direct effect with 26.5% to 33.3% depending on the definition of abuse (respectively 'yes/no' abuse and CAI). Conclusions: More depressive symptoms at baseline, lower age at depression onset, higher levels of neuroticism and loneliness and more chronic diseases explain a poor course of depression in older adults who reported childhood abuse. When treating late-life depression it is important to detect childhood abuse and consider these mediating variables.

Key words: childhood abuse; late-life depression; chronic course; longitudinal cohort study; chronicity.
Introduction

The course of depression in older adults in the general population as well as in a cohort of depressed persons is often unfavourable (Solhaug et al., 2012; Stek et al., 2002). This concerns depressive symptoms (Luppa et al., 2012) as well as formal depression diagnoses (Magnil et al., 2013). Recently, Comijs et al. (2015) found that 48.8% of the 285 older persons (aged 60-93 years) with a depression at baseline also had a depressive disorder after two years. More than half (63%) of the sample had chronic depressive symptoms over a period of two years. Apparently, late-life depression is often a chronic condition, for which the risk factors are still largely unclear.

In adults, the course of depression has been shown to be more chronic in case of childhood abuse (Wiersma et al., 2009; Hovens et al., 2012; Nanni et al., 2012). Wiersma et al. (2009) concluded: “multiple childhood traumas can be seen as an independent determinant of chronicity of depression”. Childhood abuse and an earlier age at onset were also negatively associated with remission of major depressive disorder (MDD) in younger adults (Kelly & Mezuk, 2017). Likewise Brown and Moran (1994) demonstrated a strong relationship between chronicity of depression (≥ one-year duration) and childhood abuse in 404 working-class mothers. They suggested that childhood abuse plays an important role on its own, but also influences chronicity via interpersonal difficulties, such as difficulties with their partner. One longitudinal study showed, indeed, that childhood trauma was associated with chronicity in the course of depression in adults aged 18-65 years (Hovens et al., 2012). In addition, they showed that baseline clinical characteristics, such as age at onset and the severity of depression, were important mediating factors in the relationship between childhood abuse and the course of depression. However, they did not consider personality characteristics, whereas personality characteristics, especially neuroticism, have been linked to childhood abuse (Moran et al., 2011) as well as to the course of depression (Rhebergen et al., 2011). Recently, Vinkers et al. (2014) showed that stressful experiences, including childhood abuse, contribute to depression among adults (<65 years), particularly in participants with high levels of neuroticism. Finally, Shevlin et al. (2015) showed that loneliness mediated the relationship between childhood abuse and amongst other adult psychopathology, depressive disorders. A recent review identified most of these (among others) as risk factors for a chronic course of depression in younger adults (all studies were conducted in adults aged <60 years; Hölzel et al., 2011).

To our knowledge, no study has investigated the impact of childhood abuse on the course of depression in older adults, and the possible factors that mediate this relationship. Recently, cross-sectional studies have shown an association between late-life depression and childhood abuse (Comijs et al., 2013; Ege et al., 2014). Ege et al. (2014) showed that childhood abuse, particularly repeated physical abuse and forced sexual intercourse, was significantly associated with depression in older adults. Whereas, Comijs et al. (2013) showed that late-life depression,
even when having a late onset (>60 years), was associated with all types of childhood abuse. Interestingly, the association between childhood abuse and late-life depression with an onset ≥60 years was partly explained by the number of chronic diseases (Comijs et al., 2013). This might indicate that deteriorating health may make older adults who reported childhood abuse vulnerable to developing a depressive episode in late life. Age-related changes, such as loss of physical health and loss of close relatives and friends, are very common in this age group. Therefore, chronic diseases and psychosocial factors may be important mediators to consider, in addition to previously - in younger adults - identified putative mediators, including the severity of depression symptoms, age at onset, neuroticism and loneliness.

The main aim of the present study is to investigate whether childhood abuse is associated with a poor course of late-life depression. First, we hypothesize that depressed older adults who reported to be abused during childhood have a more chronic two-year course of depression. Second, we hypothesize that the association between childhood abuse and the course of depression is mediated by the severity of depression, the age at onset of depression, neuroticism, the number of chronic diseases, and psychosocial factors such as loneliness and social network size.

Methods

Study sample
This longitudinal study was based on data from the Netherlands Study of Depression in Older Persons (NESDO; https://nesdo.onderzoek.io/). More detailed information can be found in the design paper (Comijs et al., 2011). In short, for the baseline measurement, 378 participants with a depression diagnosis in the previous 6-months and 132 nondepressed controls from five regions in the Netherlands were interviewed. To ensure that persons with late-life depression differed in severity and stage of the disorder, participants were recruited from mental health care institutes as well as from general practitioners (Comijs et al., 2011). Depressed participants were included if they met the DSM-IV-TR criteria (APA, 2000) for MDD, dysthymia or minor depression. A (possible) diagnosis of dementia, a primary psychotic disorder or a bipolar disorder based on clinical judgment, a score under 18 out of 30 on the Mini Mental State Examination (MMSE; Folstein et al., 1975) and insufficient command of the Dutch language were exclusion criteria. The nondepressed comparison group had no lifetime diagnoses of depression.

Two years later all participants were invited for a second face-to-face assessment; 83.4% of the first assessment participated in the second interview (Comijs et al., 2015). Attrition was due to death and mental problems in the patient group and having no interest or no time in the control group. Attrition was significantly more common among persons who had lower education, who had more severe psychopathology and lower cognitive functioning at baseline (Comijs et al.,
had more severe psychopathology and lower cognitive functioning at baseline (Comijs et al., 2015). For the present study, we selected participants that were depressed at baseline and participated in the second interview (n=285). From this group, three subjects had missing data on childhood abuse and had to be excluded. This resulted in a total of 282 participants included in the analyses. The study design was approved by the Ethical Review Board of the VU University Medical Center and written informed consent was obtained from all the participants.

**Measurements**

**Depression characteristics**

A formal diagnosis of depression, according to the DSM-IV-TR criteria (APA, 2000), was determined using the Composite International Diagnostic Interview (CIDI; WHO-version 2.1; lifetime version). This is a structured clinical interview that has high validity for depressive disorders and is particularly used in research settings (Wittchen et al., 1991). For this study, we included participants that fulfilled the criteria for a MDD, dysthymia diagnosis or minor depression diagnosis at one point in time during the previous six months. In the statistical analyses, we considered only the presence or absence of a depression diagnosis at two-year follow-up.

**Childhood abuse**

Childhood abuse was assessed using a structured interview, namely the Childhood Abuse Inventory. This measurement, previously used in the Netherlands Mental Health Survey and Incidence Study (NEMESIS; de Graaf et al., 2004), retrospectively inquires about people’s childhood (before the age of 16 years). The occurrence and frequency of several types of childhood abuse are recorded, namely emotional neglect (lack of parental attention/support and ignorance of one’s problems), psychological abuse (verbal abuse, punishment without reason, being blackmailed), physical abuse (being kicked or hit) and sexual abuse (sexually touched against one’s will, or forced to touch someone sexually). In our analyses, we used a dichotomized (yes/no) variable, where “no” reflects no abuse at any type of abuse. In addition, we calculated a childhood abuse index (CAI) from the sum of the recorded frequencies of these events (never=0; once, sometimes=1; regularly, often or very often=2), ranging from 0-8. Higher scores indicate a higher frequency of childhood abuse (Wiersma et al., 2009).

**Mediating variables**

Several variables were considered as mediators, including clinical and personality characteristics. Clinical characteristics included the severity of depression, which was assessed with the Inventory of Depressive Symptomatology – Self-report version (IDS-SR; Rush et al., 1996), and the age at onset of depression, which was obtained from the CIDI. Neuroticism was measured using a subscale of the NEO-Five Factor Inventory (FFI; Costa & McCrae, 1995). The subscale neuroticism scores range from 12 to 60, higher scores indicating more neuroticism. The Loneliness scale was used to assess feelings of loneliness (de Jong Gierveld & Kamphuis, 1985). This scale consists of 11 questions leading to a score ranging from 0 (“no loneliness at all”) to 44. Higher scores indicate a higher frequency of loneliness.
to 11 ("very severe loneliness"). Social network size was determined using the Close Person Inventory (CPI; Stansfeld & Marmot, 1992), which asks participants to indicate how many important contacts they have on a regular basis, such as family and friends. This was categorized in six answering options: 0-1 (1), 2-5 (2), 6-10 (3), 11-15 (4), 16-20 (5), more than 20 (6). For our purpose, we dichotomized these options into small social network (0-5 people), and large social network (6-20 people or more). The number of chronic diseases was measured with a self-report questionnaire asking for the presence of several chronic diseases, namely cardiac disease, peripheral atherosclerosis, stroke, diabetes mellitus, COPD (i.e. asthma, chronic bronchitis or osteoarthritis) and cancer. For more detailed information on validity and reliability of the instruments used in the NESDO study, we refer readers to Comijs et al. (2011).

**Covariates**

Several variables, including sex, age, years of education, partner status, comorbid anxiety disorder and recent life events, were considered as confounding variables. Partner status was obtained with standard questions asking whether the participant had an intimate relationship with someone they considered as their partner. Current anxiety disorder (last 6-month diagnosis) was determined by the CIDI, including social phobia, panic disorder, agoraphobia and general anxiety disorder (GAD). The number of recent negative life events in the past five years was assessed using the Brugha questionnaire (Brugha et al., 1985).

**Statistical analyses**

Differences in characteristics between participants with and without a depression diagnosis at two-year follow-up were analyzed using independent samples t-tests, Mann-Whitney U-tests or Chi-squared tests. When differences (p≤.05) between these groups were found or when the strength of the association between childhood abuse and depression diagnosis at follow-up changed 10% or more when the variable of interest was added to the regression model, they were used as covariates in subsequent analyses. Multicollinearity was investigated using a correlation matrix, where a correlation coefficient equal to or greater than 0.50 was considered as an indication of multicollinearity. In addition, Variation Inflation Factors (VIFs) were calculated as an indication of how much variance of the regression coefficient is increased as a result of collinearity. A VIF of 3 or greater was considered as an indication of multicollinearity.
The associations between childhood abuse and two-year follow-up depression diagnosis were investigated using multivariable mediation analysis (Figure 1). Following Preacher & Hayes (2008) we used a multiple mediation model quantifying the indirect effect of the independent variable (childhood abuse) on the dependent variable (two-year follow-up depression diagnosis) through one or more mediator(s) by testing the effect of childhood abuse on the mediator (a) and by testing the effect of the mediator on depression diagnoses at two-year follow-up (b). The product “a x b” quantifies the indirect effect through the mediators, and was obtained using a bootstrapping procedure with 5000 bootstrap samples. The direct effect of childhood abuse on two-year follow-up depression diagnoses with consideration of the mediation effect was quantified as “c’”. When the bias corrected 95%-confidence interval did not contain zero, we considered the mediating effect significant. First, we investigated the mediating effect of all mediating variables adjusted for relevant confounders separately. Secondly, we created a mediation model with all variables in one complete model. In addition, we calculated the change in effect using the following formula: \( \Delta B = \frac{(c - c')}{c} \times 100 \). This process was repeated for the associations between the CAI and two-year follow-up depression diagnosis.

All p-values were tested two-tailed and p-values ≤.05 were considered as statistically significant. Statistical Package of the Social Sciences version 20.0 (SPSS 2011) was used to conduct all statistical analyses.

Figure 1. Direct effect of childhood abuse on depression diagnosis at two-year follow-up (c); and multivariable mediation analysis of the association between childhood abuse and depression diagnosis at two-year follow-up (c').
Results

Sample characteristics

Characteristics of the study sample at baseline according to two-year follow-up depression diagnosis are shown in Table 1. Of the 282 participants, 137 persons (48.6%) met the criteria for a depression diagnosis (MDD, dysthymia or minor depression). Forty-six (33.6%) of the 137 participants with a MDD at two-year follow-up also had a dysthymia diagnosis. Persons who were depressed at follow-up had significantly higher depression severity scores at baseline and a lower age at onset of depression. A history of childhood abuse was significantly more present in persons that were depressed at two-year follow-up, specifically psychological, physical and sexual abuse. They also scored significantly higher on neuroticism, felt lonelier, had a smaller social network and more chronic diseases. Of these, only age was considered as a confounding variable in subsequent mediation analyses based on a 10% change of the regression coefficient of the dependent variable. Collinearity was ruled out by correlations below 0.50. None of the correlation coefficients exceeded 0.50 (data not shown). All VIFs were below 3.

Multivariable analyses were performed by means of mediation analysis investigating the association between childhood abuse and depression diagnosis at two-year follow-up. Table 2 and 3 show the results of the mediation model with outcome defined as childhood abuse yes/no (Table 2) and total scores on the CAI (Table 3), all adjusted for age (also Figure 2a and 2b). First, we investigated the role of several putative mediators, namely severity of depression, age at onset of depression, neuroticism, loneliness, social network size and number of chronic diseases.
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Table 1. Baseline characteristics of persons that had a depression at baseline and participated in the follow-up (N=282), grouped as persons with a depression diagnosis after two years (N=137) and persons without a depression diagnosis after two years (N=145).

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>N=282</th>
<th>No depression diagnosis after two years N=145</th>
<th>Depression diagnosis after two years N=137</th>
<th>Test value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographics</strong></td>
<td></td>
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</tr>
<tr>
<td>Female, N (%)</td>
<td>282</td>
<td>95 (65.5)</td>
<td>89 (65.0)</td>
<td>X²=0.01 (1)</td>
<td>.92</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>282</td>
<td>70.34 (7.12)</td>
<td>70.89 (7.94)</td>
<td>t=-0.62 (280)</td>
<td>.54</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>282</td>
<td>10.66 (3.25)</td>
<td>10.52 (3.65)</td>
<td>t=0.35 (280)</td>
<td>.73</td>
</tr>
<tr>
<td>Partner status, N (% yes)</td>
<td>280</td>
<td>80 (55.9)</td>
<td>64 (46.7)</td>
<td>X²=2.39 (1)</td>
<td>.12</td>
</tr>
<tr>
<td><strong>Childhood abuse</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Childhood abuse, N (%)</td>
<td>282</td>
<td>65 (44.8)</td>
<td>87 (63.5)</td>
<td>X²=9.89 (1)</td>
<td>.002</td>
</tr>
<tr>
<td>Emotional neglect, N (%)</td>
<td>282</td>
<td>55 (37.9)</td>
<td>66 (48.2)</td>
<td>X²=3.02 (1)</td>
<td>.08</td>
</tr>
<tr>
<td>Psychological neglect, N (%)</td>
<td>282</td>
<td>30 (20.7)</td>
<td>43 (31.4)</td>
<td>X²=4.20 (1)</td>
<td>.04</td>
</tr>
<tr>
<td>Physical abuse, N (%)</td>
<td>282</td>
<td>14 (9.7)</td>
<td>26 (19.0)</td>
<td>X²=5.03 (1)</td>
<td>.03</td>
</tr>
<tr>
<td>Sexual abuse, N (%)</td>
<td>282</td>
<td>23 (15.9)</td>
<td>35 (25.5)</td>
<td>X²=4.05 (1)</td>
<td>.04</td>
</tr>
<tr>
<td>Childhood abuse index, median (IQR)</td>
<td>282</td>
<td>0.00 (2)</td>
<td>1.00 (4)</td>
<td>U=8021.00</td>
<td>.003</td>
</tr>
<tr>
<td><strong>Mediating variables</strong></td>
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<tr>
<td>Age at onset depression², mean (SD)</td>
<td>276</td>
<td>51.42 (19.38)</td>
<td>44.10 (20.79)</td>
<td>t=3.03 (274)</td>
<td>.003</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>279</td>
<td>25.79 (11.73)</td>
<td>33.81 (12.41)</td>
<td>t=-5.55 (277)</td>
<td>&lt;.001</td>
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<tr>
<td>Neuroticism</td>
<td>267</td>
<td>37.34 (5.83)</td>
<td>40.77 (6.67)</td>
<td>t=-4.73 (265)</td>
<td>&lt;.001</td>
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<tr>
<td>Loneliness</td>
<td>268</td>
<td>5.67 (3.48)</td>
<td>7.64 (3.16)</td>
<td>t=-4.86 (264.25)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Social network size, N (% small)</td>
<td>279</td>
<td>77 (54.2)</td>
<td>91 (66.4)</td>
<td>X²=4.33 (1)</td>
<td>.04</td>
</tr>
<tr>
<td>Number of chronic disease, median (IQR)</td>
<td>282</td>
<td>2.00 (2)</td>
<td>2.00 (3)</td>
<td>U=7687.00</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Other covariates</strong></td>
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<td></td>
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<tr>
<td>Anxiety disorder, N (%yes)</td>
<td>282</td>
<td>47 (32.4)</td>
<td>57 (41.6)</td>
<td>X²=2.56 (1)</td>
<td>.11</td>
</tr>
<tr>
<td>Number of negative life events (past 5 yrs), median (IQR)</td>
<td>282</td>
<td>2.00 (2)</td>
<td>2.00 (2)</td>
<td>U=9625.50</td>
<td>.65</td>
</tr>
</tbody>
</table>

²Continuous variables are analyzed using independent samples t-tests, categorical variables are analyzed using Chi-squared statics and non-normally distributed variables are analyzed using independent samples Mann-Whitney U-test.

³Age at onset depression = first age MDD or dysthymia was recorded.

Abbreviations: IDS = Inventory of Depressive Symptomatology, BAI = Beck's Anxiety Inventory, IQR = interquartile range.
Figure 2a. Direct and indirect effects of childhood abuse on depression diagnosis at two-year follow-up using a mediation model, adjusted for age.

Note: *p<.05; **p<.01; ***p<.001; ‘significant based on 95% confidence interval, bootstrap p<0.05

c= direct effect of childhood abuse on depression diagnosis at two-year follow-up; a= effect of childhood abuse on mediator (using standardized z-scores); b= effect of mediator (using standardized z-scores) on depression diagnosis at two-year follow-up; a x b: indirect effect of childhood abuse on depression diagnosis at two-year follow-up; c'= direct effect of childhood abuse on depression diagnosis at two-year follow-up in mediation model.
Figure 2b. Direct and indirect effects of the childhood abuse index (CAI) on depression diagnosis at two-year follow-up using a mediation model, adjusted for age.

Note: *p<.05; **p<.01; ***p<.001; †significant based on 95% confidence interval, bootstrap p<0.05
c= direct effect of childhood abuse on depression diagnosis at two-year follow-up; a= effect of childhood abuse on mediator (using standardized z-scores); b= effect of mediator (using standardized z-scores) on depression diagnosis at two-year follow-up; a x b: indirect effect of childhood abuse on depression diagnosis at two-year follow-up; c'= direct effect of childhood abuse on depression diagnosis at two-year follow-up in mediation model.
Associations between childhood abuse and depression diagnosis at follow-up

The direct association between childhood abuse (yes/no) and depression diagnosis at two-year follow-up was significant. Including every mediator separately showed that depression severity, age of depression onset, neuroticism, loneliness and number of chronic diseases significantly mediated the relationship between childhood abuse (yes/no) and depression diagnosis after two years (Table 2). Social network size was no mediating variable for this association. Looking at the separate mediators, the largest change in direct effect came from depression severity (ΔB= -26.5%), followed by neuroticism (ΔB= -20.8%), age at onset of depression (ΔB= -16.5%), loneliness (ΔB= -15.4%) and the number of chronic diseases (ΔB= -10.7%). Including all variables in the same model showed that particularly age at depression onset and loneliness were strong and significant mediators. The direct effect adjusted for a x b (c') of childhood abuse on depression diagnosis at two-year follow-up was no longer significant in a complete model; adding all mediators to our model led to a significant reduction of the direct effect (c) (ΔB= -55.1%).

Associations between the childhood abuse index (CAI) and depression diagnosis at follow-up

The direct association between the CAI and depression diagnosis at follow-up was significant. Including every mediator separately, we found that depression severity, age at onset of depression, neuroticism and number of chronic diseases significantly mediated the relationship between the CAI and depression diagnosis at follow-up (Table 3). Of these, depression severity seemed to have the strongest indirect effect, while it fully mediated the association between the CAI and depression diagnosis after two years. Loneliness and social network size were no mediating variables. Comparable to the changes in effect size for childhood abuse (yes/no), the largest change in effect size between the CAI and depression diagnosis at follow-up was found for depression severity (ΔB= -33.3%), followed by neuroticism (ΔB= -23.5%) and age at onset of depression as well as chronic diseases (both ΔB= -16.7%). In a complete model with all significant mediators, the depression severity, age of depression onset and neuroticism had a significant mediating effect in the relation between the CAI and depression diagnoses at follow-up. The direct effect corrected for a x b (c') of the CAI on depression diagnosis at two-year follow-up was no longer significant in a complete model, indicating the influence of these baseline characteristics on the course of depression in older adults with a history of childhood abuse.
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Table 2. Multivariable mediation analysis on the association between childhood abuse and depression diagnosis at two-year follow-up through depression severity, age at onset of depression, neuroticism, loneliness, social network size and number of chronic diseases.

<table>
<thead>
<tr>
<th></th>
<th>a*</th>
<th>b*</th>
<th>c</th>
<th>c'</th>
<th>a x b (bootstrapping)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>B (SE)</td>
<td>p</td>
<td>B (SE)</td>
<td>p</td>
</tr>
<tr>
<td>Separate mediators</td>
<td></td>
<td></td>
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<tr>
<td>Depression severity</td>
<td>279</td>
<td>0.44 (0.12)</td>
<td>&lt;.001</td>
<td>0.66 (0.14)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Age at onset depression</td>
<td>276</td>
<td>-0.43 (0.12)</td>
<td>&lt;.001</td>
<td>-0.35 (0.13)</td>
<td>0.01</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>267</td>
<td>0.36 (0.12)</td>
<td>0.003</td>
<td>0.62 (0.14)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Loneliness</td>
<td>268</td>
<td>0.32 (0.13)</td>
<td>0.01</td>
<td>0.57 (0.14)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Social network size</td>
<td>279</td>
<td>-0.33 (0.12)</td>
<td>0.01</td>
<td>-0.21 (0.13)</td>
<td>0.09</td>
</tr>
<tr>
<td>Chronic diseases</td>
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<td>0.30 (0.12)</td>
<td>0.02</td>
<td>0.43 (0.13)</td>
<td>0.001</td>
</tr>
<tr>
<td>Complete model†</td>
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<tr>
<td>Individual effects</td>
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<td>&lt;.001</td>
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<td>0.05</td>
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<td>- Neuroticism</td>
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<td>0.38 (0.12)</td>
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<td>0.24 (0.17)</td>
<td>0.16</td>
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<tr>
<td>- Loneliness</td>
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<td>0.03</td>
<td>0.36 (0.15)</td>
<td>0.02</td>
</tr>
<tr>
<td>- Chronic diseases</td>
<td></td>
<td>0.33 (0.13)</td>
<td>0.01</td>
<td>0.21 (0.15)</td>
<td>0.15</td>
</tr>
</tbody>
</table>

*Mediation model based on significant separate mediators: complete model mediated by severity of depression (IDS-score), age at onset depression, neuroticism, loneliness and number of chronic diseases (without social network size since it was no mediator, nor a confounder)

*significant based on 95% confidence interval (CI), bootstrap p<0.05

+ all mediating variables were standardized, meaning 'a' en 'b' are based on standardized z-scores.

NB. Bootstrapping: 5000 bootstrap samples; BC 95% CI= bias corrected 95% confidence interval.

Note. All analyses were adjusted for age. Mediation analyses were based on Preacher & Hayes, 2008.
Table 3. Multivariable mediation analysis on the association between childhood abuse index (CAI) and depression diagnosis at two-year follow-up through depression severity, age at onset of depression, neuroticism, loneliness, social network size and number of chronic diseases.

|                        | n   | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  | B (SE)       | p  |
|------------------------|-----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|--------------|----|
|                        |     |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |
| Separate mediators     |     |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |
| Depression severity    | 279 | 0.11 (0.03)  | <.001 | 0.66 (0.14)  | <.001 | 0.18 (0.06)  | 0.004 | 0.12 (0.06)  | 0.006 | 0.07 (0.03-0.13)* |
| Age at onset depression| 276 | -0.12 (0.03) | <.001 | -0.35 (0.14) | 0.01  | 0.18 (0.06)  | 0.003 | 0.15 (0.06)  | 0.02  | 0.04 (0.01-0.09)* |
| Neuroticism            | 267 | 0.08 (0.03)  | 0.004 | 0.62 (0.14)  | <.001 | 0.17 (0.06)  | 0.006 | 0.13 (0.06)  | 0.04  | 0.05 (0.02-0.10)* |
| Loneliness             | 268 | 0.05 (0.03)  | 0.10  | 0.59 (0.14)  | <.001 | 0.17 (0.06)  | 0.006 | 0.15 (0.06)  | 0.02  | 0.03 (-0.003-0.07) |
| Social network size    | 279 | -0.04 (0.03) | 0.18  | -0.24 (0.12) | 0.05  | 0.17 (0.06)  | 0.004 | 0.17 (0.06)  | 0.01  | 0.01 (-0.002-0.04) |
| Chronic diseases       | 282 | 0.09 (0.03)  | 0.002 | 0.42 (0.13)  | 0.001 | 0.18 (0.06)  | 0.003 | 0.15 (0.06)  | 0.02  | 0.04 (0.01-0.08)* |
| Complete model¹, CAI   | 260 |              | 0.18 (0.06) | 0.004 | 0.07 (0.07)  | 0.32  | 0.13 (0.07-0.22)* |
| Individual effects     |     |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |              |    |
| - Depression severity  |     | 0.12 (0.03)  | <.001 | 0.35 (0.17)  | 0.04  | 0.04 (0.01-0.10)* |
| - Age at onset depression | 0.12 (0.03) | <.001 | -0.31 (0.15) | 0.04  | 0.04 (0.004-0.09)* |
| - Neuroticism          |     | 0.09 (0.03)  | 0.002 | 0.33 (0.17)  | 0.05  | 0.03 (0.001-0.08)* |
| - Chronic diseases     |     | 0.10 (0.03)  | 0.001 | 0.25 (0.15)  | 0.09  | 0.02 (-0.001-0.07) |

¹Mediation model based on significant separate mediators: complete model mediated by severity of depression (IDS-score), age at onset depression, neuroticism and number of chronic diseases (without loneliness and social network size since they were no mediators, nor confounders)

*significant based on 95% confidence interval (CI), bootstrap p<0.05

+ all mediating variables were standardized, meaning 'a' en 'b' are based on standardized z-scores.

NB. Bootstrapping: 5000 bootstrap samples; BC 95% CI= bias corrected 95% confidence interval.

Note. All analyses were adjusted for age. Mediation analyses were based on Preacher & Hayes, 2008.
**Discussion**

In accordance with our hypothesis, a significant association was found between childhood abuse and a chronic two-year course of late-life depression. Our results are consistent with earlier findings in younger adults (18-65 years) (Wiersma et al., 2009; Hovens et al., 2012; Rhebergen et al., 2011). Although measurements, timeframes and definitions for a chronic course differed and hamper comparisons with these previous studies, they also strengthen the general idea that childhood abuse negatively influences the course of depression even in late life.

Furthermore, we hypothesized that the association between childhood abuse and depression diagnosis at follow-up would be mediated by several baseline characteristics. Indeed, this association was fully mediated by a more severe depression, a younger age at the onset of depression, higher levels of neuroticism, feeling more lonely and a higher number of chronic diseases, indicating that there is predominantly an indirect effect of childhood abuse on depression diagnosis after two years. The age at depression onset and loneliness were the strongest mediators considering the association with childhood abuse (yes/no); with respect to the CAI, depression severity, the age at depression onset and neuroticism were the strongest mediators.

Depression characteristics played an important mediating role. The direct effect between childhood abuse and depression diagnosis at follow-up was predominantly reduced by the severity of depression, leading to a reduction of 33.3% of the direct association, suggesting childhood abuse is associated with a more severe depression, which in turn negatively influences the course of depression. This is in line with a similar study in younger adults aged 18-65 years (Hovens et al., 2012), where depression severity was also identified as the strongest mediator. In addition, childhood abuse has been associated with more depressive symptoms cross-sectionally in older adults (Gamble et al., 2006). Likewise, irrespective of childhood abuse more depressive symptoms were associated with a poor course, as well as a longer duration of the late-life disorder (Comijs et al., 2015). Additionally, the age at depression onset partly mediated the association between childhood abuse and depression diagnosis at follow-up, reducing the direct effect by ±16.5%. It has been shown that childhood abuse leads to an earlier onset of depression (Bernet & Stein, 1999). This earlier onset of depression has been associated with more lifetime depressive episodes and more severe depressive symptoms (Zisook et al., 2007) and chronicity of depression (Rhebergen et al., 2012), illustrating the interplay between these depression characteristics as well.

Along with these depression characteristics, we showed that neuroticism was an important mediator since the direct effect was reduced by about 22%. Childhood abuse has been previously associated with neuroticism in older adults (Wilson et al., 2006), and neuroticism is...
known to be associated with a poor course of depression as well (Bukh et al., 2016). A recent study in younger adults also found a mediating role of higher levels of neuroticism between childhood abuse and the four-year course of depression, showing that these effects are still visible after a longer period of time (Hovens et al., 2016).

Of our psychosocial variables, only loneliness was a mediating variable. A study by Shevlin et al. (2015) already identified that loneliness mediated the association of childhood abuse and adult depression. In our study, however, this mediation effect was only found for childhood abuse (yes/no) and not for the CAI, which might indicate that the presence of childhood abuse is more important than the frequency of abuse. In our study, social network size was not a significant mediator. One possible explanation might be that it is not about the number of social contacts but the quality of the contacts that influence the course of depression (Schwarzbach et al., 2014). Still, as a consequence of an unfavorable course of depression, it has been shown that social networks become smaller and levels of loneliness increase (Houtjes et al., 2014). This might indicate a vicious circle, where childhood abuse and these mediators are only a starting point for more chronicity and other negative developments.

Finally, we found a mediation effect of the number of chronic diseases. Previously, childhood abuse has been associated with different physical parameters that reflect physical health (Walker et al., 1999). In addition, it was demonstrated that more somatic illnesses predicted a poor course of depression (Comijs et al., 2015). It seems that declining health at later age may make victims of childhood abuse more vulnerable to a chronic course of depression. It has been suggested that chronic diseases contribute to a poor course of depression via cognitive styles such as hopelessness instead of via a direct organic effect (cognitive vulnerability theory; Alloy et al., 1999) and hopelessness has been shown to mediate the association between childhood abuse and a poor course of depression in younger adults (Hovens et al., 2016). This might also explain the significant but small effect of chronic diseases as mediator (respectively 10.7% and 16.7% reduction of the direct association for childhood abuse yes/no and CAI).

Another point for consideration is that putative predictors of the course of depression may differ across different age groups. Many of our mediating factors have already been found in younger adults, which might indicate that some mediators are applicable to all age groups such as depression severity and a younger age at depression onset. Other factors might be more specific to younger or older adults, or the strength of the association might differ across age groups; however, this is largely unknown. In addition, there may also be other potential mediators, such as inflammation, brain circuitry or genetics (Teicher & Samson, 2013; Teicher et al., 2016). Therefore, more research is needed to disentangle this issue, especially in older adults and for comparison with other age groups.
Strengths and limitations
Major strengths of this study were the large number of participants and the possibility to investigate several mediators and to control for various confounders. Another strength of our study was the categorical as well as dimensional measurement of childhood abuse. These strengths enabled an in-depth analysis of the longitudinal association between childhood abuse and the course of late-life depression. Several limitations need to be mentioned as well. First of all, childhood abuse was investigated retrospectively making it subject to possible over- or underreporting (Fergusson et al., 2000) that might be due to the mental health status. However, psychopathology has been associated with neither less reliable nor less valid reports of childhood experiences (Hardt & Rutter, 2004). Still, we need to be cautious in the interpretation of our results. Another limitation is that we considered only the presence or absence of depression diagnosis and did not look at specific course types of depression, such as remitted MDD, intermittent MDD and a chronic depression, nor did we combine this with depression severity over time. It might be interesting to investigate whether these course types are differentially associated with childhood abuse. Finally, due to the naturalistic design of the study, the impact of various treatment regimens on the association between childhood abuse and the course of late-life depression could not be thoroughly examined.

Clinical relevance
Our findings strongly underscore the importance of detecting a history of childhood abuse in case of depression in older adults. Childhood abuse is not only an important predictor for psychopathology, but also for a poor course of the disorder. Next, it is clinically important to consider the role of mediating variables in the course and persistence of late-life depression. These mediators may also be helpful in treatment selection and future profiling. For example, loneliness and possibly neuroticism might be targeted by therapeutic interventions (García-Martín, 2004). Other characteristics, such as the severity and an early onset of depression, call for more preventive strategies. Another possibility is to integrate these findings into specific treatments for chronic forms of depression, of which childhood traumas seem to be independent determinants (Wiersma et al., 2009), such as cognitive behavioral analysis system of psychotherapy (CBASP).

Conclusion
Childhood abuse is strongly associated with a poor course of late-life depression. This study showed that a considerable part of the association (about 60%) can be explained by a more severe depression at baseline, lower age at depression onset, higher levels of neuroticism, loneliness and more chronic diseases. In the treatment of late-life depression it is important to detect childhood abuse and to consider mediating characteristics that influence its course negatively.

Childhood abuse and the two-year course of late-life depression | 119
References


Reliability of retrospective reports on childhood abuse and its determinants in older adults during a 6-year follow-up

Ilse Wielaard, Max L. Stek, Hannie C. Comijs, Didi Rhebergen

Abstract

Introduction: Controversy exists concerning the reliability of retrospective self-reports on childhood abuse since this method might be subject to under- or overreporting. Until now, no studies have been done in older adults, although reasons for under- or overreporting could be even more prominent in this age group. In this first study in older adults, test-retest reliability of retrospective measurements on childhood abuse and the influence of age, cognitive functioning and depression on this test-retest reliability was investigated. Methods: A longitudinal cohort study, the Netherlands Study of Depression in Older persons (NESDO), obtained information on childhood abuse at baseline and at a 6-year follow-up interview. Our sample consisted of 277 adults (mean age 68.5 years at baseline) of which 118 (42.6%) reported childhood abuse at baseline. Results: The largest proportion of the answers was consistent (yes-yes or no-no) for every type of childhood abuse, varying from 85.2 to 93.5%. Looking more closely, ‘yes’ answers were more fluctuating than ‘no’ answers. Sexual abuse was most reliably reported in two separate interviews. There was no significant effect of age nor cognition on the test-retest reliability. Only test-retest reliability of emotional neglect was significantly associated with depression diagnosis and depression severity at baseline. Conclusions: In conclusion, test-retest reliability of retrospective self-reports on childhood abuse seems moderate to good, but slightly dependent on the type of abuse. The test-retest reliability was influenced neither by age nor cognition, only reporting of emotional neglect was associated with depression state.

Key words: test-retest reliability; answer consistency; proportion of specific agreement; retrospective self-reports; childhood abuse; late-life depression.
Introduction

Childhood abuse has a large negative impact on physical and mental health in all age groups, even in old age (Comijs et al., 2013; Cuijpers et al., 2011; Ege et al., 2015; Kessler et al., 2010; Lindert et al., 2014; Springer et al., 2007). Most studies in this field used retrospective, self-report measurements to obtain information on childhood abuse history, such as the Childhood Abuse Inventory that has been used in several population-based and clinical cohorts (Comijs et al., 2011; De Graaf et al., 2004; Penninx et al., 2008). Controversy exists concerning the reliability of these retrospective, self-report measurements of childhood abuse, since this method might be subject to under- or overreporting.

Until now, most research into the reliability of childhood abuse measurements has been done in adults aged 18 to ±60 years, using different sample sizes and methodology. A review (Hardt & Rutter, 2004) included eight studies that investigated the reliability of adult recall of adverse childhood experiences such as sexual abuse, and concluded that retrospective reports on adverse childhood experiences most likely underestimate the occurrence of childhood abuse. More recently, several other studies investigated the consistency of retrospective reports on childhood abuse. One study used three measurements over a period of 1.5 years, on average 30 years after the participants’ childhood (Da Silva & Da Costa Maia, 2013). Although their sample was relatively small (N=30), they concluded that the temporal stability for assessment of childhood abuse (with the exception of sexual abuse) was acceptable. Some other studies concluded that measurements of childhood abuse are more reliable when the questions focus on events that are concrete, specific and unambiguous (Hardt et al., 2006; Yancura & Aldwin, 2009). Recently, Naicker et al. (2017) also concluded that retrospective reports of adverse childhood experiences are sufficiently valid. Especially childhood neglect and abuse are more consistently reported than experiences that are subject to broad interpretation and one’s own judgement.

Several reasons have been given for under- and overreporting in retrospective self-reports, such as being unable or unwilling to report embarrassing and painful events, and fallibility of memory (Brennen et al., 2010 in Colman et al., 2016; Widom & Morris, 1997; Williams, 1994). Furthermore, cognitive problems and a negative mood might also influence the consistency of self-reports (Colman et al., 2016; Eikelenboom et al., 2014). Thus far, studies in younger adults considered some of these factors; however, these factors might be even more prominent later in life. For instance, age could be an important factor in older adults, while more time has passed since childhood, which might influence these self-reports (Fergusson et al., 2000; Newbury et al., 2018). Age has only been considered in studies with younger adults. Naicker et al. (2017) investigated prospective informant-reports and retrospective self-reports over time in children, adolescents and young adults (maximum of 26 years). They found that the prevalence of abuse reports increased in adolescence but decreased in young adulthood, which may be due
to a longer time-lag. However, they suggested it could be due to a change of environment, schools and peers leading to new experiences. On the other hand, Yancura et al. & Aldwin (2009) reported moderate to high reliability of self-reports over time in a sample aged 22-61 years and found that age was not predictive for inconsistencies.

With age, retrospective self-reports on childhood abuse may also be hampered by cognitive decline, especially in older adults. Cognitive problems, as a consequence of ageing, become more prominent in older age and might therefore influence the consistency of retrospective self-reports. However, although older adults may have problems with episodic memory (learning and remembering new information), retrospective autobiographical memories that contain factual information about things that happened earlier in their lives are recalled much better (e.g. Drag & Bieliauskas, 2010). Yancura et al. & Aldwin (2009) aimed to study the underlying process of changing memory capacity with increasing age, but examined the impact of age on the test-retest reliability of childhood abuse reports instead of cognition and included a younger sample (maximum age of 61 years). Although, it is generally known that cognition declines with age, no study has investigated the role of cognition on the reliability of retrospective reports of childhood abuse in adults 65 years and older.

Finally, several studies have investigated whether psychopathology influences the reliability of retrospective reports. Some studies in adults younger than 60 years indicated that the test-retest reliability of childhood abuse reports is unrelated or weakly related to psychiatric status (current, past or future) (Brewin et al., 1993; Fergusson et al., 2000; Yancura et al. & Aldwin, 2009). However, Susser & Widom (2012) reported that, in most studies, retrospective self-reports, as compared to official records of abuse, are more strongly related to adult psychopathology. In addition, Colman et al. (2016) found that, among others, the development of depression was related to inconsistent reporting of childhood adverse events. These findings might indicate that depression influences the retrospective self-reports in older adults.

To conclude, thus far no studies have examined the reliability of retrospective childhood abuse reports and the influence of age, cognition and depressive status on retrospective self-reports in older adults. We therefore asked participants of the Netherlands Study of Depression in Older persons (NESDO) twice, with an interval of six years, to recall possible childhood abuse. This allows us to investigate the answer consistency of retrospective recall of these memories. The main goal of the present study is to examine the test-retest reliability of retrospective recall of childhood abuse over these two measurement moments and to assess whether this reliability is comparable for different types of childhood abuse, e.g. emotional neglect, psychological, physical and sexual abuse. We also aim to investigate whether this is state-dependent, meaning whether age, cognition and depression predict test-retest reliability.
Reliability of retrospective reports on childhood abuse and its determinants | 129

Methods

Study sample
Data was used from the Netherlands Study of Depression in Older persons (NESDO; https://nesdo.onderzoek.io/). Detailed information on design and (baseline) measurements can be found in the paper of Comijs et al. (2011). In short, NESDO is a longitudinal cohort study investigating depressive disorders in adults aged 60 years and older. So far, NESDO has consisted of three face-to-face interviews, starting with a baseline interview and then follow-up interviews at two and six years. At baseline, 510 older adults were included from the Netherlands. Participants were included when they fulfilled the DSM-IV-TR criteria for major depressive disorder (MDD), dysthymia or minor depression (APA, 2000). A non-depressed control group consisted of participants that had no lifetime depression diagnosis. Exclusion criteria were having a (possible) diagnosis of a dementia, psychotic or bipolar disorder, a score under 18 out of 30 on the Mini Mental State Examination (MMSE; Folstein et al., 1975) or insufficient command of the Dutch language. During the third interview, six years later, 277 participants answered questions concerning childhood abuse for the second time and were selected for the present study. The same childhood abuse questionnaire was used in the follow-up interview, enabling us to compare the answers. At the 6-year follow-up, in the depressed group most participants (nearly 47%) dropped out due to mortality and mental health reasons, such as cognitive impairment, while in the control group (nearly 26%) this was due to refusal. Only 13% of the depressed group had a full remission. More detailed information about the third interview (and attrition) can be found in Jeuring et al. (2018). The Ethical Review Board of the VU University Medical Center and of participating centres approved the study design. All participants gave written informed consent.

Measurements
Childhood abuse:
Childhood abuse was assessed using the Childhood Abuse Inventory, which is a semi-structured interview designed to gain insight into the occurrence and frequency of several types of childhood abuse prior to the age of 16 (De Bijl et al., 1998; De Graaf et al., 2004). This semi-structured interview has previously been used in large-scale studies, including the Netherlands Mental Health Survey and Incidence Study (NEMESIS) and the Netherlands Study of Depression and Anxiety (NESDA) (De Graaf et al., 2004; Penninx et al., 2008). It asks questions about four types of childhood abuse, namely emotional neglect, and psychological, physical and sexual abuse. Each of these types of childhood abuse are further specified by concrete examples of abuse. Emotional neglect concerns not being listened to, ignoring problems and emotions, feeling unwelcome, getting little or no attention and support from the parents. Psychological abuse includes name calling, yelling, unfair punishment, subordinate or disadvantageous treatment compared to brothers/sisters and blackmailing. Physical abuse concerns being kicked or hit with the hands or an object, or other forms of physical abuse. Sexual abuse consists of
being sexually touched against one's will or being forced to sexually touch someone else. After participants reported the presence of this type of abuse, the frequency of childhood abuse was scored as 'never' (0), 'once' and 'sometimes' (1) and 'regularly', 'often', and 'very often' (2). For this study, we only used the answer on presence (yes/no) of (types of) childhood abuse. Scores at baseline and at the 6-year follow-up were compared. Four options were possible, including consistent positive (yes/yes) or negative (no/no) answers for the two measurements, or inconsistent answers, including 'yes' on baseline and 'no' on the follow-up interview or vice versa, for each type of childhood abuse.

Age, cognition, depression:
Age was determined by the birth year of the participants given at the baseline measurement. A depression diagnosis, according to the Diagnostic and Statistical Manual of Mental Disorders (4th Edition, text revision) was assessed by the Composite International Diagnostic Interview (CIDI; WHO Version 2.1; lifetime version). This is a structured interview, especially used in research settings, with high validity for depressive and anxiety disorders (Wittchen et al., 1991). For this study, participants with a 6-months diagnosis of MDD, dysthymia and/or minor depression were included. The Inventory of Depressive Symptomatology – Self-Report (IDS-SR) version was used to assess the severity of depression (Rush et al., 1996), with scores ranging from 0-80. Global cognitive functioning was tested by the MMSE (Folstein et al., 1975), which consists of 11 questions with a maximum score of 30. Memory was tested using the 10 Words Test, which is a modified version of the Auditory Verbal Learning Test (Rey, 1964). The interviewer reads a list of ten words and asks the participant to repeat as many of these words as possible, and repeats this five times, with a maximum score of 10 per trial. Twenty to thirty minutes later the interviewer again asks the participants to recall as many words as possible (delayed reproduction), with a score ranging from 0-10. For this study, we included the total score over all five trials and we computed a retention-score which is “the score on the delayed reproduction / maximum score on the five trials”, thereby reflecting the rate of forgetting.

Statistical analyses
Baseline differences between the study sample and the persons who were lost to follow-up (attrition sample) were analysed using an independent t-test, chi-squared test, and Kruskal-Wallis test when appropriate. To investigate the answer consistency of the retrospective reports on childhood abuse, we used two different approaches. First, we checked the answer consistency of participants on the childhood abuse questionnaire over the two measurements. Next, we computed Cohen's Kappa for dichotomous variables to compare the reliability between interview 1 and 3. Its value varies from zero to 1.00. Interpretation was based on Landis & Koch (1977), where '0' means poor reliability, 0-0.20 'slight', 0.21-0.4 'fair', 0.41-0.60 'moderate', 0.61-0.8 'substantial', and above 0.81 'almost perfect'. Cohen's Kappa is based on chance frequencies, which means that in the case of skewed distributions of variables, these results could be biased and it is not useful for computing Cohen's Kappa. In addition, whereas
reliability is the “ability of a measure applied twice upon the same respondents to produce the same ranking on both occasions, agreement requires the measurement tool to produce twice the same exact values” (Berchtold, 2016, page 1). Therefore, we additionally examined agreement between two measurements, based on De Vet et al. (2013). They concluded that the agreement for positive and negative ratings would be the most appropriate absolute measure with respect to this answer consistency issue and would be most informative for clinicians. The formula for positive agreement (PA) concerning ‘yes’ answers in our study, is $2a/(2a+b+c)$, and for negative agreement (NA) concerning ‘no’ answers this is $2d/(2d+b+c)$ (see Figure 1). In addition, the effect of age, cognition and depression on the consistency or inconsistency of the answers between two interviews was investigated using chi-squared tests and ANOVA for normally distributed variables and Kruskal-Wallis test for non-normally distributed variables. We considered these variables on baseline and follow-up measurements. In addition, we computed the difference between the scores on interview 1 and interview 3 as an indication of change between these two moments. All statistical analyses were conducted using the Statistical Package of the Social Sciences version 22.0 (SPSS 2013).

### Results

#### Attrition

Of the 510 participants at baseline, 230 participants were lost to follow-up at the time of the third interview. Baseline characteristics are shown in Table 1. The attrition sample concerned participants (N=230) that were significantly older and had fewer years of education. Depression was also more common and more severe in the attrition group, and they scored worse on the cognitive tests (MMSE and 10 Words Test). There was no significant difference concerning childhood abuse, except for physical abuse. Persons that reported physical abuse at baseline dropped out of the study significantly more often.

#### Sample characteristics

In total, 277 persons participated in the follow-up interview and provided information on childhood abuse. Of our sample, 96 participants had no depression diagnosis at both interviews, 50 participants (18.1%) had a depression diagnosis at both interviews. 131 participants (47.3 %)
had a depression diagnosis at baseline and no diagnosis at follow-up, 6 years later. In our sample, 191 participants (69%) answered consistently on all types of childhood abuse, while 86 participants (31%) answered inconsistently on one or more types of childhood abuse.

**Table 1. Baseline characteristics of persons that participated in interview 3, our study sample (N=277), and participants that fell out the study (N=230).**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>N=277</th>
<th>Study sample N=277</th>
<th>N=230</th>
<th>Attrition N=230</th>
<th>Test value¹ (df)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographics</strong></td>
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<td></td>
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<td></td>
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<tr>
<td>Female, N (%)</td>
<td>277</td>
<td>181 (65.3)</td>
<td>230</td>
<td>147 (63.9)</td>
<td>X²= 0.11 (1)</td>
<td>.74</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>277</td>
<td>68.50 (6.13)</td>
<td>230</td>
<td>73.05 (7.89)</td>
<td>t= 7.14 (427.11)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>277</td>
<td>11.60 (3.53)</td>
<td>230</td>
<td>10.20 (3.46)</td>
<td>t= -4.47 (505)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Childhood abuse</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Childhood abuse, N (% yes)</td>
<td>277</td>
<td>118 (42.6)</td>
<td>230</td>
<td>103 (44.8)</td>
<td>X²= 0.24 (1)</td>
<td>.62</td>
</tr>
<tr>
<td>Emotional neglect, N (% yes)</td>
<td>277</td>
<td>86 (31.0)</td>
<td>230</td>
<td>78 (33.9)</td>
<td>X²= 0.47 (1)</td>
<td>.49</td>
</tr>
<tr>
<td>Psychological abuse, N (% yes)</td>
<td>277</td>
<td>57 (20.6)</td>
<td>230</td>
<td>50 (21.7)</td>
<td>X²= 0.10 (1)</td>
<td>.75</td>
</tr>
<tr>
<td>Physical abuse, N (% yes)</td>
<td>277</td>
<td>26 (9.4)</td>
<td>230</td>
<td>36 (15.7)</td>
<td>X²= 4.60 (1)</td>
<td>.03</td>
</tr>
<tr>
<td>Sexual abuse, N (% yes)</td>
<td>277</td>
<td>48 (17.3)</td>
<td>230</td>
<td>44 (19.1)</td>
<td>X²= 0.28 (1)</td>
<td>.60</td>
</tr>
<tr>
<td><strong>Depression and cognition variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>277</td>
<td>181 (65.3)</td>
<td>230</td>
<td>194 (84.3)</td>
<td>X²= 23.57 (1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>274</td>
<td>21.71 (14.87)</td>
<td>225</td>
<td>27.64 (14.98)</td>
<td>t= 4.42 (497)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>277</td>
<td>28.00 (3.00)</td>
<td>230</td>
<td>28.00 (3.00)</td>
<td>U= 24319.50</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>10 WT total score (trial 1-5), mean (SD)</td>
<td>276</td>
<td>34.06 (6.57)</td>
<td>229</td>
<td>29.93 (6.99)</td>
<td>t= -6.83 (503)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>277</td>
<td>75.55 (20.30)</td>
<td>230</td>
<td>69.72 (25.9)</td>
<td>t= -2.78 (429.49)</td>
<td>.01</td>
</tr>
</tbody>
</table>

¹Continuous variables are analyzed using independent samples t-tests, categorical variables are analyzed using Chi-squared statics and non-normally distributed variables are analyzed using independent samples Mann-Whitney U-test.

**Reliability and specific agreement**

In Table 2, the answer consistencies on all forms of childhood abuse are shown. Most importantly, the majority of people answered consistently (yes-yes, no-no) on both interviews. Emotional neglect and psychological abuse had more or less equal numbers of people that answered consistently, respectively 85.2% and 86.7%. The highest number of consistent reports were on sexual abuse, namely 93.5%. There were many consistent ‘no-no’ responses (see Table 2), which led to skewed distribution and influenced the value of Cohen’s Kappa. Although test-retest reliability of forms of abuse, reflected by Cohen’s Kappa, was considered moderate to substantial/good (see Table 2), the finding may be biased due to the skewed distribution. Therefore we also calculated the proportion of specific agreement (see Table 2). The proportion of positive agreement (PA) and negative agreement (NA) for emotional neglect was 75.2% and
89.5% respectively. For psychological abuse, the PA was 63.4% and the NA was 91.8%. PA was 57.1% and NA was 95.2% for physical abuse, while for sexual abuse PA was 79.1% and NA was 96.2%. Overall, the test-retest reliability seems good over time. However, looking at proportions of specific agreements, the negative 'no' answers are highly reliable while the positive 'yes' answers fluctuate more. Especially for psychological and physical abuse, there was less agreement on the positive answers.

**Predictors of reliability**

Three predictors of reliability were considered in our sample, namely age, cognition and depression, specifically for those participants that answered consistently 'yes' or answered inconsistently. We performed separate univariate comparisons for every type of childhood abuse (see Table 3-6). For all types of childhood abuse, we found no significant differences of age between the consistent and inconsistent groups. We found a significant difference on depression diagnosis and depression severity (Table 3) only for emotional neglect. Persons that gave a consistent 'yes' answer and persons that answered 'yes' on baseline and 'no' on follow-up more often had a depression diagnosis and scored significantly higher on depression severity (IDS) at baseline compared to the persons that answered 'no' on baseline and 'yes' on follow-up. Concerning cognition, we found no statistically significant differences, although for emotional neglect and sexual abuse the differences on cognitive measurements almost reached statistical significance.

**Table 2. Answer consistency on the childhood abuse questionnaire in our study sample (N=277)**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Baseline N=277</th>
<th>6-yr follow-up N=277</th>
<th>Int1*Int3 N=277</th>
<th>Kappa’s Cohen (k)</th>
<th>Proportion of specific agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Childhood abuse</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional neglect, N (% yes)</td>
<td>86 (31.0)</td>
<td>79 (28.5)</td>
<td>-</td>
<td>.65</td>
<td></td>
</tr>
<tr>
<td>Consistent yes-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>62 (22.4)</td>
<td>PA = 75.2%</td>
<td></td>
</tr>
<tr>
<td>Consistent no-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>174 (62.8)</td>
<td>NA = 89.5%</td>
<td></td>
</tr>
<tr>
<td>Inconsistent yes-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>24 (8.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inconsistent no-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>27 (6.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychological abuse, N (% yes)</td>
<td>57 (20.6)</td>
<td>44 (15.9)</td>
<td>-</td>
<td>.55</td>
<td></td>
</tr>
<tr>
<td>Consistent yes-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>32 (11.6)</td>
<td>PA = 63.4%</td>
<td></td>
</tr>
<tr>
<td>Consistent no-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>208 (75.1)</td>
<td>NA = 91.8%</td>
<td></td>
</tr>
<tr>
<td>Inconsistent yes-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>25 (9.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inconsistent no-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>12 (4.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical abuse, N (% yes)</td>
<td>26 (9.4)</td>
<td>30 (10.8)</td>
<td>-</td>
<td>.52</td>
<td></td>
</tr>
<tr>
<td>Consistent yes-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>16 (5.8)</td>
<td>PA = 57.1%</td>
<td></td>
</tr>
<tr>
<td>Consistent no-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>237 (85.6)</td>
<td>NA = 95.2%</td>
<td></td>
</tr>
<tr>
<td>Inconsistent yes-no, N (%)</td>
<td>-</td>
<td>-</td>
<td>10 (3.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inconsistent no-yes, N (%)</td>
<td>-</td>
<td>-</td>
<td>14 (5.1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 3. Characteristics of persons that gave a consistent ‘yes’-answer or an inconsistent answer concerning emotional neglect.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Consistent ‘yes’</th>
<th>Inconsistent ‘yes – no’</th>
<th>Inconsistent ‘no · yes’</th>
<th>Test value (df)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotional neglect</td>
<td>N= 62</td>
<td>N= 24</td>
<td>N= 17</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Baseline characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female, N (%)</td>
<td>43 (69.4)</td>
<td>19 (79.2)</td>
<td>13 (76.5)</td>
<td>X²= 0.98 (2)</td>
<td>.61</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>68.00 (6.44)</td>
<td>68.17 (6.93)</td>
<td>69.41 (6.66)</td>
<td>F(2,100) = 0.31</td>
<td>.73</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>11.48 (3.71)</td>
<td>10.33 (3.38)</td>
<td>11.71 (3.51)</td>
<td>F(2,100)= 1.04</td>
<td>.36</td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>60 (96.8)</td>
<td>21 (87.5)</td>
<td>10 (58.8)</td>
<td>X²= 18.69 (2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>30.24 (13.42)</td>
<td>29.26 (16.18)</td>
<td>19.47 (13.28)</td>
<td>F(2,99)= 4.00</td>
<td>.02</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.00 (2)</td>
<td>28.00 (2)</td>
<td>28.00 (2)</td>
<td>X²=0.47 (2)</td>
<td>.79</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>34.69 (6.71)</td>
<td>32.21 (6.08)</td>
<td>33.76 (6.01)</td>
<td>F(2,99) = 1.29</td>
<td>.28</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>75.23 (20.01)</td>
<td>75.36 (19.38)</td>
<td>68.02 (23.35)</td>
<td>F(2,100) = 0.89</td>
<td>.41</td>
</tr>
<tr>
<td><strong>Follow-up characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>18 (29.0)</td>
<td>7 (29.2)</td>
<td>5 (29.4)</td>
<td>X²= .001 (2)</td>
<td>1.00</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>19.37 (12.35)</td>
<td>19.78 (14.63)</td>
<td>16.24 (10.0)</td>
<td>F(2,97)= 0.48</td>
<td>.62</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>29.00 (3)</td>
<td>28.50 (3)</td>
<td>28.00 (2)</td>
<td>X²=2.34 (2)</td>
<td>.31</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.10 (7.60)</td>
<td>33.91 (6.93)</td>
<td>30.65 (10.59)</td>
<td>F(2,96)= 0.86</td>
<td>.43</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>76.02 (22.68)</td>
<td>66.26 (21.94)</td>
<td>62.74 (27.96)</td>
<td>F(2,95)= 2.82</td>
<td>.07</td>
</tr>
<tr>
<td><strong>Change scores (Int 1 – Int 3)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE</td>
<td>-0.05 (1.79)</td>
<td>-0.29 (2.05)</td>
<td>0.53 (1.23)</td>
<td>F(2,100) =1.10</td>
<td>.34</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>1.73 (5.65)</td>
<td>-1.50 (7.48)</td>
<td>3.12 (8.96)</td>
<td>F(2,96)= 2.65</td>
<td>.08</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>-0.04 (22.43)</td>
<td>11.11 (25.68)</td>
<td>5.28 (34.14)</td>
<td>F(2,95)=1.59</td>
<td>.21</td>
</tr>
</tbody>
</table>

*Continuous variables are analyzed using one-way ANOVA, categorical variables are analyzed using Chi-squared statics and non-normally distributed variables are analyzed using Kruskall Wallis Test.

Abbreviation: IDS = Inventory of Depressive Symptomatology; MMSE= Mini Mental State Examination; IQR = interquartile range; 10WT=10 Words Test.

Note: IDS scores range from 0-80; MMSE scores range from 0-30; 10WT retention score = score on 10WT recall / the maximum score on the 5 tests.
Table 4. Characteristics of persons that gave a consistent ‘yes’-answer or an inconsistent answer concerning psychological abuse.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Consistent ‘yes’</th>
<th>Inconsistent ‘yes – no’</th>
<th>Inconsistent ‘no - yes’</th>
<th>Test value¹ (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological abuse</td>
<td>N=32</td>
<td>N=25</td>
<td>N=12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female, N (%)</td>
<td>22 (68.8)</td>
<td>18 (72.0)</td>
<td>11 (91.7)</td>
<td>X²=2.45 (2)</td>
<td>.29</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>68.13 (5.78)</td>
<td>66.80 (5.81)</td>
<td>69.08 (8.02)</td>
<td>F(2,66)= 0.62</td>
<td>.54</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>11.53 (3.69)</td>
<td>11.68 (3.39)</td>
<td>10.08 (3.83)</td>
<td>F(2,66)= 0.84</td>
<td>.44</td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>30 (93.8)</td>
<td>20 (80.0)</td>
<td>10 (83.3)</td>
<td>X²=2.51 (2)</td>
<td>.29</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>31.44 (13.41)</td>
<td>27.42 (16.71)</td>
<td>22.58 (10.68)</td>
<td>F(2,65)=1.78</td>
<td>.18</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.00 (3)</td>
<td>28.00 (3)</td>
<td>28.00 (2)</td>
<td>X²=0.69 (2)</td>
<td>.71</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.38 (6.51)</td>
<td>34.40 (6.63)</td>
<td>34.00 (5.78)</td>
<td>F(2,66)= 0.17</td>
<td>.84</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>72.42 (18.61)</td>
<td>79.87 (24.46)</td>
<td>66.39 (27.62)</td>
<td>F(2,66)=1.61</td>
<td>.21</td>
</tr>
<tr>
<td>Follow-up characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>13 (30.4)</td>
<td>7 (28.0)</td>
<td>1 (8.3)</td>
<td>X²=4.41 (2)</td>
<td>.11</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>22.90 (15.21)</td>
<td>18.04 (13.75)</td>
<td>16.17 (6.99)</td>
<td>F(2,63)= 1.41</td>
<td>.25</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.00 (3)</td>
<td>29.00 (3)</td>
<td>29.00 (2)</td>
<td>X²=0.54 (2)</td>
<td>.76</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.23 (7.55)</td>
<td>34.79 (8.04)</td>
<td>30.75 (12.39)</td>
<td>F(2,63)= 0.86</td>
<td>.43</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>71.08 (24.25)</td>
<td>75.66 (15.99)</td>
<td>72.74 (34.66)</td>
<td>F(2,62)= 0.24</td>
<td>.79</td>
</tr>
<tr>
<td>Change scores (Int 1 – Int 3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE</td>
<td>0.03 (1.71)</td>
<td>-0.08 (2.12)</td>
<td>0.00 (1.65)</td>
<td>F(2,66)=0.01</td>
<td>.99</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>0.33 (5.59)</td>
<td>-0.42 (6.98)</td>
<td>3.25 (8.75)</td>
<td>F(2,63)=1.22</td>
<td>.30</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>1.08 (25.57)</td>
<td>6.53 (23.64)</td>
<td>-6.35 (40.76)</td>
<td>F(2,62)=0.83</td>
<td>.44</td>
</tr>
</tbody>
</table>

¹Continuous variables are analyzed using one-way ANOVA, categorical variables are analyzed using Chi-squared statics and non-normally distributed variables are analyzed using Kruskall Wallis Test.

Abbreviation: IDS = Inventory of Depressive Symptomatology; MMSE= Mini Mental State Examination; IQR = interquartile range; 10WT=10 Words Test.

Note: IDS scores range from 0-80; MMSE scores range from 0-30; 10WT retention score = score on 10WT recall / the maximum score on the 5 tests.
Table 5. Characteristics of persons that gave a consistent 'yes'-answer or an inconsistent answer concerning physical abuse.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Consistent 'yes'</th>
<th>Inconsistent 'yes – no'</th>
<th>Inconsistent 'no – yes'</th>
<th>Test value(1) (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical abuse</strong></td>
<td>N= 16</td>
<td>N=10</td>
<td>N=14</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Baseline characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female, N (%)</td>
<td>8 (50.0)</td>
<td>2 (20.0)</td>
<td>9 (64.3)</td>
<td>(X^2=4.65) (2)</td>
<td>.10</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>67.75 (5.83)</td>
<td>67.10 (7.45)</td>
<td>67.50 (4.80)</td>
<td>(F(2,37)= 0.04)</td>
<td>.96</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>12.06 (4.33)</td>
<td>11.60 (3.44)</td>
<td>12.43 (3.20)</td>
<td>(F(2,37)= 0.24)</td>
<td>.87</td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>15 (93.8)</td>
<td>9 (90.0)</td>
<td>13 (92.9)</td>
<td>(X^2=0.13) (2)</td>
<td>.94</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>31.13 (12.23)</td>
<td>27.60 (16.85)</td>
<td>25.50 (13.12)</td>
<td>(F(2,37)= 0.64)</td>
<td>.54</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.00 (3)</td>
<td>29.00 (2)</td>
<td>29.00 (1)</td>
<td>(X^2=1.68) (2)</td>
<td>.43</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.06 (6.80)</td>
<td>30.80 (5.53)</td>
<td>33.50 (6.35)</td>
<td>(F(2,37)= 0.58)</td>
<td>.76</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>74.25 (21.44)</td>
<td>65.37 (24.76)</td>
<td>70.02 (22.55)</td>
<td>(F(2,37)= 0.48)</td>
<td>.62</td>
</tr>
<tr>
<td><strong>Follow-up characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>6 (37.5)</td>
<td>2 (20.0)</td>
<td>4 (28.6)</td>
<td>(X^2=0.92) (2)</td>
<td>.63</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>24.00 (15.30)</td>
<td>15.90 (9.98)</td>
<td>22.00 (12.48)</td>
<td>(F(2,36)= 1.19)</td>
<td>.32</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>29.00 (3)</td>
<td>27.50 (5)</td>
<td>29.00 (3)</td>
<td>(X^2=1.54) (2)</td>
<td>.66</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.20 (6.78)</td>
<td>31.20 (7.63)</td>
<td>30.57 (7.51)</td>
<td>(F(2,36)= 0.51)</td>
<td>.60</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>68.59 (28.43)</td>
<td>67.82 (19.70)</td>
<td>74.21 (27.68)</td>
<td>(F(2,35)= 0.22)</td>
<td>.80</td>
</tr>
<tr>
<td><strong>Change scores (Int 1 – Int 3)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE</td>
<td>-0.56 (1.71)</td>
<td>0.50 (2.64)</td>
<td>0.21 (0.98)</td>
<td>(F(2,37)= 1.27)</td>
<td>.29</td>
</tr>
<tr>
<td>10 WT total score</td>
<td>-0.13 (5.28)</td>
<td>-0.40 (5.15)</td>
<td>2.93 (5.66)</td>
<td>(F(2,36)=1.56)</td>
<td>.22</td>
</tr>
<tr>
<td>10 WT retention</td>
<td>5.43 (33.24)</td>
<td>1.64 (28.64)</td>
<td>-4.19 (23.80)</td>
<td>(F(2,35)=0.40)</td>
<td>.67</td>
</tr>
</tbody>
</table>

\(^1\)Continuous variables are analyzed using one-way ANOVA, categorical variables are analyzed using Chi-squared statistics and non-normally distributed variables are analyzed using Kruskall Wallis Test.

**Abbreviation:** IDS = Inventory of Depressive Symptomatology; MMSE = Mini Mental State Examination; IQR = interquartile range; 10WT=10 Words Test.

**Note:** IDS scores range from 0-80; MMSE scores range from 0-30; 10WT retention score = score on 10WT recall / the maximum score on the 5 tests.
Table 6. Characteristics of persons that gave a consistent ‘yes’-answer or an inconsistent answer concerning sexual abuse.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Consistent ‘yes’</th>
<th>Inconsistent ‘yes – no’</th>
<th>Inconsistent ‘no – yes’</th>
<th>Test value¹ (df)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexual abuse</td>
<td>N=34</td>
<td>N=14</td>
<td>N=4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female, N (%)</td>
<td>28 (82.4)</td>
<td>12 (85.7)</td>
<td>3 (75.0)</td>
<td>X²= 0.26 (2)</td>
<td>.88</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>68.29 (5.44)</td>
<td>65.79 (3.58)</td>
<td>68.50 (10.02)</td>
<td>F(2,49)= 1.11</td>
<td>.24</td>
</tr>
<tr>
<td>Years of education, mean (SD)</td>
<td>10.53 (3.25)</td>
<td>10.07 (3.17)</td>
<td>10.00 (3.74)</td>
<td>F(2,49)= 0.23</td>
<td>.88</td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>29 (85.3)</td>
<td>13 (32.9)</td>
<td>4 (100.0)</td>
<td>X²= 1.12 (2)</td>
<td>.57</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>29.12 (14.53)</td>
<td>29.07 (14.30)</td>
<td>31.00 (18.89)</td>
<td>F(2,49)= 0.03</td>
<td>.97</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.00 (2)</td>
<td>29.00 (1)</td>
<td>29.00 (2)</td>
<td>X²= 5.45 (2)</td>
<td>.03</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>34.41 (6.26)</td>
<td>35.71 (3.99)</td>
<td>28.50 (5.80)</td>
<td>F(2,49)= 2.49</td>
<td>.09</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>75.85 (19.00)</td>
<td>75.75 (17.47)</td>
<td>79.17 (11.28)</td>
<td>F(2,49)= 0.06</td>
<td>.94</td>
</tr>
<tr>
<td>Follow-up characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis, N (%)</td>
<td>8 (23.5)</td>
<td>1 (7.1)</td>
<td>1 (25.0)</td>
<td>X²= 1.81 (2)</td>
<td>.41</td>
</tr>
<tr>
<td>Total scores on IDS, mean (SD)</td>
<td>21.58 (12.66)</td>
<td>17.00 (9.50)</td>
<td>14.50 (9.33)</td>
<td>F(2,48)= 1.29</td>
<td>.31</td>
</tr>
<tr>
<td>MMSE, median (IQR)</td>
<td>28.50 (4)</td>
<td>29.00 (1)</td>
<td>28.50 (3)</td>
<td>X²= 2.66 (2)</td>
<td>.26</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>33.19 (7.58)</td>
<td>35.29 (4.12)</td>
<td>35.00 (12.70)</td>
<td>F(2,47)= 0.45</td>
<td>.64</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>67.31 (30.94)</td>
<td>74.37 (19.37)</td>
<td>65.00 (24.23)</td>
<td>F(2,47)= 0.36</td>
<td>.70</td>
</tr>
<tr>
<td>Change scores (Int 1 – Int 3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE</td>
<td>0.32 (2.00)</td>
<td>0.21 (2.01)</td>
<td>0.50 (1.00)</td>
<td>F(2,49)= 0.04</td>
<td>.96</td>
</tr>
<tr>
<td>10 WT total score, mean (SD)</td>
<td>1.47 (6.03)</td>
<td>0.43 (4.93)</td>
<td>-6.50 (11.27)</td>
<td>F(2,47)= 2.91</td>
<td>.06</td>
</tr>
<tr>
<td>10 WT retention, mean (SD)</td>
<td>8.50 (31.62)</td>
<td>13.8 (12.70)</td>
<td>14.17 (28.73)</td>
<td>F(2,47)= 0.48</td>
<td>.63</td>
</tr>
</tbody>
</table>

¹Continuous variables are analyzed using one-way ANOVA, categorical variables are analyzed using Chi-squared statics and non-normally distributed variables are analyzed using Kruskall Wallis Test.

Abbreviation: IDS = Inventory of Depressive Symptomatology; MMSE= Mini Mental State Examination; IQR = interquartile range; 10WT=10 Words Test.

Note: IDS scores range from 0-80; MMSE scores range from 0-30; 10WT retention score = score on 10WT recall / the maximum score on the 5 tests.

Discussion

Controversy exists regarding the test-retest reliability of retrospective reports on childhood abuse. This is the first study in older adults that investigates the reliability and agreement of retrospective self-report measurements on childhood abuse and whether these are influenced by age, cognitive functioning or depression. We compared the answers from two measurements over time and found that the largest proportion of the answers was consistent (yes-yes or no-no) for every type of childhood abuse, varying from 85.2 to 93.5%. In particular, there was a large proportion of negative agreement (89.5-96.2%) but some variation in the proportion of positive agreement (57.1-79.1%). Sexual abuse was most consistently reported in two separate interviews. Next, we found that only the answer consistency on emotional neglect...
was significantly influenced by depression diagnosis and depression severity at baseline. There was no significant effect of age or cognition on the answer consistency.

Most importantly, the majority of people answered consistently over time, so retrospective self-reports on childhood abuse seem reliable. This is in line with some other studies in younger adults, such as Yancura et al. & Aldwin (2009), who found that retrospective reports on specific adverse childhood experiences were remarkably stable over a period of 5 years. Our results are not in line with those of Hardt et al. (2006), who concluded that physical and sexual abuse was only moderately reliably answered. However, like other studies (e.g. Shields et al., 2015), they used Cohen’s Kappa with skewed distribution, which makes it harder to interpret the results and is less useful for clinicians. Heterogeneity in methodology and statistical analyses is a major issue that contributes to the controversy (Hardt et al., 2006; Kottner et al., 2011; Shields et al., 2015; Yancura et al. & Aldwin, 2009). Since a large proportion of ‘no-no’ answers in our sample also led to skewed distributions, we calculated the proportion of specific agreement, which was a solution suggested by De Vet et al. (2013). This turned out to be useful and showed high agreement in answers when no childhood abuse was reported during the first interview (89.5-96.2%). The amount of positive agreement was 57-79%, meaning that they reported childhood abuse at the first interview but changed their reports in the second interview. These proportions showed that ‘no’ answers are highly stable and ‘yes’ answers fluctuate more, particularly for psychological and physical abuse, suggesting the need for more in-depth exploration in the case of a positive answer. Furthermore, it showed that especially childhood sexual abuse is answered consistently.

Next, we looked at factors that might influence the answer consistency. We found an effect of depression for emotional neglect, but not for psychological, physical and sexual abuse. It seems that only reporting emotional neglect might be coloured by depression. Comparison with other studies is difficult, since emotional neglect was not specifically investigated. Previous studies used different measurements and varied in the types of childhood experiences that were examined. Predominantly, these studies showed that retrospective reports were more strongly related to depression and other psychopathology (Susser & Widom, 2012); however, the inconsistency of reports and the association with depression has not been made very clear. One study in the general population found that the inconsistency between two retrospective reports with a 10-year interval was related to depression (Colman et al., 2016): the likelihood of reporting a new childhood adverse experience increased with development of depression and increased stress levels. In addition, Reuben et al. (2016) showed that retrospective self-reports were more related to subjective health measurements compared to objective health measurements. It might be that the examples given for emotional neglect, e.g. feeling unwelcome and getting no attention, are more related to depressive symptoms that were subjectively measured in our study. Susser & Widom (2012) concluded that adults with more psychological problems look back on and report more negatively about their childhood;
although it could also mean that (mentally) healthy adults are better at “forgiving and forgetting” (Reuben et al., 2016).

There was no significant effect of age and cognition. With regard to cognition, we found only a trend for emotional neglect and sexual abuse. One reason could be that older people with more cognitive problems dropped out of the study; however, there was no attrition difference for people with or without childhood abuse, except for physical abuse. Another possible explanation could be that inter-individual variability increases with age and influences the overall cognitive ageing effect (Drag et al., 2009& Bieliasauskas, 2010). So, positive and negative differences could be found on the individual level, possibly leading to non-significant differences on the group level.

Although most people will give reliable answers to concrete and specific questions (Hardt et al., 2006; Yancura & Aldwin, 2009), there will always be some doubt about the reliability of retrospective 'positive' reports on childhood abuse. No official records were used, but one could also argue that using official records would cause an underestimation of the actual number of childhood abuse cases. However, it is extremely difficult and expensive to design a prospective study on the effect of childhood abuse on mental health in older adults (Fergusson et al., 2000). In addition, the overall reliability was moderate to good, so one could argue that the inconsistent reports could be dependent on other factors, such as the context or the interviewer (Da Silva & Da Costa Maia, 2013) or it may be variable due to an implicit, grey area between abuse or no abuse.

Several strengths and limitations should be mentioned. A major strength is the availability of 6-year follow-up data in NESDO with two measurements on childhood abuse and the inclusion of measurements that were important for studying the answer consistency in older adults. Furthermore, both interviews contained the same concrete and specific questions concerning childhood abuse. Our study also has several limitations. First, we were not able to look at the duration of childhood abuse in this study, although a longer duration might lead to more consistent ‘yes’ answers. Although our study did contain reports on the frequency of childhood abuse, these were based on the initial question and did not specify the duration of childhood abuse. Additionally, using the frequency of the four types of childhood abuse would have led to groups that were too small for our statistical analyses. Furthermore, due to the sample of adults aged 60 years and older, a rather long period of time has passed since the actual childhood abuse, hence an impact of this long timeframe on recall of childhood abuse cannot be excluded. However, there was no significant effect of age on the answer consistency. Second, the attrition rate from baseline to the third interview, six years later, was fairly high (nearly 47%). Especially in the depressed group, attrition was due to mortality and mental reasons, such as cognitive impairment. This could mean that our findings were coloured by the attrition, and cognition might still be an important factor that influenced the test-retest reliability. Third, a large
number of univariate tests have been performed due to separate analyses for all types of abuse, which normally need to be corrected for multiple testing. However, we did not perform a Bonferroni correction, as this would have increased the chance of making a type-II error for all tests where we would not have detected a real effect (Gelman et al., 2012; Perneger, 1998). Fourth, since NESDO recruited particularly older adults within mental health care, the rate of reported childhood abuse might be higher than in the general population. However, it allowed us to examine the influence of mood, age and cognition, but this also limits the generalizability of our findings. Consequently, no conclusion can be drawn for the general population. Unfortunately, to our knowledge, research into the psychometric properties of the Childhood Abuse Inventory is lacking and also limits our interpretation of the reliability of this inventory. Finally, NESDO did not contain detailed information on specific treatments. It could have been interesting to examine whether the focus of treatment had an effect on the answer consistency of retrospective reports.

Clinical relevance
Acquiring information on adverse childhood experiences and retrospective self-reports on childhood abuse are of great value. Childhood abuse has a strong effect on psychopathology, both earlier and later in life. In late-life depression, childhood abuse is highly prevalent (Comijs et al., 2013). The co-occurrence of ageing, childhood abuse and depression provides information on important depression characteristics and the course of depression; for example, a history of childhood abuse as well as ageing are associated with more chronic depressive symptoms (Hovens et al., 2012; Schakx et al., 2018; Wieland et al., 2017). Predominantly, this means that retrospective reports on childhood abuse in older adults are a major clinically relevant diagnostic instrument. The current study shows that the large majority of older adults answer retrospective questions about childhood abuse consistently, especially ‘no’ answers. Positive ‘yes’ answers were slightly less reliable. Therefore, in the case of a positive ‘yes’ answer on childhood abuse questions, interviewers and clinicians need to ask more specific and concrete questions and ask for more in-depth information on their childhood to gain insight in possible childhood abuse.

Conclusion
In older adults, the test-retest reliability and agreement of retrospective self-reports on childhood abuse seems moderate to good over time and to some extent dependent on the type of childhood abuse. Furthermore, the consistency of the answers was not significantly influenced by age and cognition. Depression seemed to influence the answer consistency on emotional neglect, but not the other types of childhood abuse.

Acknowledgements:
We thank C. Terwee for her involvement with the statistical analyses and its considerations.
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The current study shows that the large majority of older adults answer childhood abuse consistently, especially ‘no’ answers. Therefore, if a positive ‘yes’ answer on retrospective questions about childhood abuse consistently, especially ‘no’ answers. Positive diagnostic instrument. The current study shows that the large majority of older adults answer childhood abuse as well as ageing are associated with more chronic depressive symptoms (Hovens et al., 2012; Schaaks et al., 2018; Wielard et al., 2017). Predominantly, this means that retrospective reports on childhood abuse in older adults are a major clinically relevant methodological Innovations, 9, 2059799116672875.


References


Susser, E., & Widom, C. S. (2012). Still searching for lost truths about the bitter sorrows of


Summary and general discussion
The aim of this thesis was to further disentangle the association between childhood abuse and late-life depression. We had four research questions. First, we looked at the association between childhood abuse and two possible underlying biological mechanisms, namely HPA-axis and telomere length. Next, we investigated the role of chronic somatic diseases, psychosocial and lifestyle factors in the association between childhood abuse and, respectively, late-life depression and functional disabilities. Third, we were interested in the association between childhood abuse and the course of late-life depression. Fourth, we examined the test-retest reliability of the retrospective self-reports concerning childhood abuse in older adults.

This chapter will give a short overview of the main findings and will discuss the results per research question. Next, it will reflect on methodological considerations and the strengths and limitations of the studies. Finally, clinical implications of the findings will be discussed and future research suggestions will be made.

**Findings per chapter**

In chapter 2 and 3, we studied the association between childhood abuse and two biological mechanisms, namely HPA-axis functioning and telomere length, and its link with late-life depression. In chapter 2, we examined the association between childhood abuse and the hypothalamic-pituitary-adrenal (HPA)-axis, which is a biological stress system that was measured by cortisol levels in saliva during the day. We found that childhood abuse was associated with lower basal cortisol levels at awakening, irrespective of depression state. In addition, a higher reactivity of the HPA axis during the hour after awakening (elevated AUCi) was found in non-depressed older adults only. In the depressed group, this elevated peak after awakening was not found. The age at onset of depression had no major influence on these associations. Our findings suggest that older adults with a history of childhood abuse might be more negatively affected by stress or stressful events, which is reflected by dysregulations of the HPA axis.

Another biological mechanism that might be associated with childhood abuse is cellular aging, reflected by telomere length. Some studies among younger adults have shown that psychosocial stressors and higher risks on mental and somatic health problems are linked to shortened telomere length. In chapter 3, we examined the association between childhood adversity (childhood abuse, childhood adverse events) and recent psychosocial stressors (recent negative life events and loneliness) and telomere length in older adults. In our study, we found that childhood abuse, recent negative life events and loneliness were unrelated to telomere length in both depressed and non-depressed older adults. There was a significant, but weak, negative association with telomere length for having experienced any childhood adverse event. We concluded that there is no clear, consistent association between psychosocial stressors (early and later in life) and shortened telomeres in older adults. We assumed that in older adults other telomere length-damaging factors, such as physical health problems, might blur the putative influence of psychosocial stressors on telomere length.
In chapter 4 and 5, our goal was to investigate the effect of childhood abuse on early- and late-onset depression, as well as functional disabilities, and the role of depression characteristics, psychosocial factors, chronic somatic diseases and lifestyle factors. Therefore in chapter 4, we studied whether psychosocial factors, specifically partner status, social network size and feelings of loneliness, mediate the association between childhood abuse and both early-onset and late-onset depression. In particular, we were interested whether the strength of the mediation effect differed between early- and late-onset depression. Overall, we found that childhood abuse was significantly associated with early- as well as late-onset depression. Only for early-onset depression, a smaller social network and loneliness partly mediated this association. Loneliness appeared the strongest mediator, leading to a reduction of almost 25% of the direct effect between childhood abuse and early-onset depression. The mediation effects were strongest for emotional neglect and psychological abuse. No psychosocial mediators were found for the association between childhood abuse and late-onset depression.

Childhood abuse as well as depression has an effect on functional disabilities, such as mobility, self-care, household activities and participation in society. The question remained whether childhood abuse is associated with functional disabilities later in life, and which factors mediate this association. In chapter 5, we therefore examined the association between childhood abuse and functional disabilities in older adults, and whether depression, chronic somatic diseases and life style factors mediate this association. The results showed that older adults reporting childhood abuse had more functional disabilities compared to those adults that did not report a history of childhood abuse, irrespective of depression state. Furthermore, this association was fully explained by having more depressive symptoms, a younger age at depression onset and more chronic diseases. Lifestyle factors were no mediators in the association between childhood abuse and functional disabilities.

Longitudinal studies showed that childhood abuse is associated with chronicity of depression in younger adults. Later in life, depression often has a chronic course. However, no consensus exists on the impact of childhood abuse and the factors that explain chronicity of late-life depression. So, in chapter 6, we studied the course of late-life depression over a period of two years in older adults that provided information on the childhood trauma questionnaire. In addition, we examined several mediating variables, namely severity of depression, age at onset of depression, neuroticism, number of chronic diseases, and psychosocial factors such as loneliness and social network size. Our study showed that childhood abuse is strongly associated with a poorer course of late-life depression. This poorer course was explained by having more depressive symptoms at baseline, younger age at depression onset, higher levels of neuroticism and loneliness and more chronic diseases. This also implicates that childhood abuse has predominantly an indirect effect on the course of depression. These findings strengthen the general idea that childhood abuse negatively influences the course of
depression, even in late life. Especially depression severity seems an important predictive course factor.

Last, but not least, we wanted to validate our findings by examining the reliability of retrospective self-reports on childhood abuse in older adults. This is described in chapter 7. Since the use of retrospective reports, controversy exists on the reliability of acquired information on childhood abuse obtained in adulthood. Although reasons for under- or overreporting might even be more prominent in old age, no studies have been done in older adults. We investigated the test-retest reliability and agreement of retrospective reports on childhood abuse and the role of age, cognitive functioning and depression. Our results showed that most answers were consistent (yes-yes or no-no) on two separate interviews, varying from 85 to 94%. This was most stable concerning the no-answers, whereas yes-answers were more fluctuating. The test-retest reliability was moderate to good over time, but slightly dependent on the type of abuse. Of all types of childhood abuse, sexual abuse was most reliably reported in both confirmative (positive) answers and denying (negative) answers, whereas the reliability of positive answers on physical abuse was lowest. The consistency of the answers was not significantly influenced by age or cognition. Depression seemed to influence the answer consistency on emotional neglect, but importantly not the other types of childhood abuse.

Discussion per research question (RQ)

RQ 1: What is the role of the HPA-axis and telomere length in the association between childhood abuse and late-life depression?

In older adults, childhood abuse was associated with the stress response, reflected by the cortisol awakening response. On the other hand, we did not find an association with telomere length. In younger adults, more consistent associations were found with these two biological mechanisms. In previous studies in older adults, findings are more diverse and inconsistent, which might be due an accumulation of other factors that come with age, such as somatic diseases, comorbidity, medications, or frailty. These other factors could confound the true relationship. In addition, large differences between studies in sample characteristics, measurements and methods are noticed (e.g. Strüber et al., 2014) and raise difficulties when comparing our findings on biological mechanisms with current literature. Furthermore, studies on HPA-axis functioning and telomere length in older persons with a history of childhood abuse are limited. Initially, we only found one study in older adults from a population-based sample showing an association between childhood adversity (including abuse) and lower morning cortisol (Gerritsen et al., 2010). Our findings were in line with this study, since we also found lower morning cortisol, independent of depression state. In addition, we found a higher reactivity during the hour after awakening (AUCi), only in non-depressed older adults with a history of childhood abuse, especially psychological and sexual abuse. The cortisol awakening response in childhood abused older adults has not been studied yet, and findings in younger
adults vary from greater reactivity to reduced reaction to stressors (Heim et al., 2010; Strüber et al., 2014). Accelerated cellular aging, which is measured by telomere length, was also predominantly studied in younger adults, showing that psychosocial stressors, mental and somatic health problems can be linked to shortened telomere length (e.g. Verhoeven et al., 2015; Verhoeven et al., 2014; Epel et al., 2004). Due to ageing and its associated physical changes, this might be different in older adults. However, in older adults from NESDO, depression was unrelated to telomere length (Schaakxs et al., 2015). In our study, we also concluded that psychosocial stressors (childhood abuse, recent negative life events and loneliness) were unrelated to telomere length. Our explanation is that in older adults other telomere length-damaging factors might blur the putative influence of psychosocial stressors on its length, such as physical health problems (Price et al., 2013) or alternatively, that NESDO contains the more ‘physically healthy’ depressed older adults.

Recently, Nemeroff (2016) wrote a review on neurobiological and clinical consequences of childhood abuse, particularly in children and younger adults. He concluded that findings on HPA-axis functioning after childhood abuse were divergent and conflicting, predominantly showing it is a complex area. The complexity is probably due to other (confounding) factors, such as type and timing of childhood abuse, presence of psychosocial support and more recent traumatic events, family history of psychiatric disorders and (epi)genetic factors. Looking at conflicting results on telomere length between younger and older adults one could suggest also a complex interaction of these and other factors that might have a combined negative effect on telomere length. Overall, studies among younger adults showed that childhood abuse has a negative effect on neurobiological mechanisms and this is intertwined with many factors. Comparing our findings with these studies seems to imply that age does differentially affect biological mechanisms, and need to be disentangled per age group. At old age, neurobiological mechanisms may still be influenced by childhood abuse but it seems particularly important to investigate explaining, mediating factors.

In addition, independent of the growing literature on childhood abuse consequences, there is still paucity concerning other biological mechanisms, such as certain hormones, neuroanatomical changes in certain brain areas and in brain functioning (Nemeroff, 2016). Teicher et al. (2016) reviewed neuroimaging studies and found that there is a clear association between childhood abuse and changes in the brain, but that the link between these changes and psychopathology is complicated. This complements our studies, showing that childhood abuse negatively affects other biological mechanisms and that particularly the link between neurobiology and late-life depression could be complicated. In two reviews (Teicher et al., 2016; Teicher & Samson, 2016), they concluded that childhood abuse probably was an unrecognized confounder in psychiatric imaging studies since it affects brain development, even in ‘healthy’ adults with a history of childhood abuse. This was also suggested by Heim et al. (2010; 2008; 2004) who showed that many biological changes were characteristic for childhood abuse. Apart from the impact of childhood abuse on psychopathology, the literature lacks a comprehensive
overview of the consequences of childhood abuse over the life course. The increasing number of meta-analyses and reviews published in the last years expands our understanding in younger age groups, but also shows the importance of continuity and persistence in research to disentangle the impact of childhood abuse on neurobiological and other mechanisms later in life. Especially since childhood abuse and its neurobiological alterations seems to create increased vulnerability for a poorer health and, hence, suggests a window of opportunities for a preventive approach (McCrory et al., 2017; Berens et al., 2017).

**Answer RQ1:** Later in life, childhood abuse is associated with dysregulation of the HPA-axis, but not with telomere length. The association between childhood abuse and dysregulation of the HPA-axis is independent from depression status.

**RQ 2:** What is the impact of childhood abuse on depression characteristics, psychosocial and lifestyle factors, chronic somatic diseases and functional disabilities later in life.

Cross-sectionally, we investigated the consequences of childhood abuse later in life on several important factors. Most clearly, childhood abuse has a strong influence on depression and its characteristics. Depression most often develops earlier in life and with more depressive symptoms, which has been predominantly found in younger adults (e.g. Bernet & Stein, 1999; Green et al., 2010; McLaughlin et al., 2012). Our studies were clearly in line with these findings. However, some people develop their first depression later in life, suggesting that they were able to stay mentally healthy until late life. We therefore wondered which factors mediate the association between childhood abuse and the onset of depression.

Adults with a history of childhood abuse more often have problems with psychosocial functioning (Horan & Widom, 2015; Pitzer & Fingerman, 2010), for instance their social networks are smaller and they more often feel lonely (Gibson & Hartshorne, 1996; Sperry & Widom, 2013; Wilson et al., 2006), and we hypothesized this could also be related to the onset of depression. We found that a smaller social network and feeling more lonely partly explained the association between childhood abuse and late-life depression; however, especially in early-onset and not in late-onset depression. So, psychosocial problems seem to be more important in depression with an onset at a younger age or a recurrent depressive episode. This might point at difficulties in developing social skills, long-lasting relationships and a strong social network. Therefore, early interventions should focus on developing better psychosocial functioning. Although results vary, even later in life interventions can improve psychosocial skills (Hagan et al., 2014; Masi et al., 2011).

Another factor that partly explained the onset of depression was the presence of chronic somatic diseases. Contrary to psychosocial factors it partly explained a late onset of depression (Comijs et al., 2013). In chapters 4 and 5, we showed that in older adults chronic somatic diseases often play a significant mediating role between childhood abuse and late-life depression, and functional disabilities. Chronic somatic diseases may be due to aging, but also
to long-term effects of an unhealthy lifestyle, which is also often associated with childhood abuse. Childhood abuse has been associated with lifestyle factors, such as smoking, obesity and risky behavior (Walker et al., 1999; Anda et al., 2006; Rehkopf et al., 2016). This seems particularly important at a younger age, since we did not find a mediation effect of lifestyle factors between childhood abuse and functional disabilities later in life. In our study, childhood abuse was not related to lifestyle factors. It might be that later in life the consequences of an unhealthy lifestyle become more important, such as more chronic somatic diseases, and explains why the presence of chronic somatic diseases was a significant mediator (chapters 4 and 5). At least we did find a significant association between certain lifestyle factors and functional disabilities later in life, meaning that it has an effect on daily life functioning. Furthermore, it could be that the sum of unhealthy lifestyle factors is more important than each separate lifestyle factor. Future longitudinal research is needed to explain the nature of this association.

As was earlier shown, childhood abuse as well as depression has an effect on functional disabilities (Walker et al., 1999; Chartier et al., 2007; Wegman and Stetler, 2009; van der Werff et al., 2010; Verhaak et al., 2014), particularly shown in younger adults. In older adults, we found that higher depression severity, younger age at depression onset and more chronic somatic diseases fully explained the association between childhood abuse and functional disabilities on domains such as cognition, mobility, household activities and participation in society (chapter 5). Functional disabilities are also related to depression and may increase the impact of childhood abuse on these two concepts, since the association between depression and functional disabilities seems reciprocal. Studies that have looked specifically into this association suggested a two-way association: depression has an effect on functional disabilities as well as the other way around (Ormel et al., 2002). This could explain why we found that depression was a significant mediator, and hints towards a vicious circle.

Answer RQ2: Childhood abuse is associated with an early age at depression onset and higher severity of late-life depression, an increased number of chronic somatic diseases and functional disabilities. Psychosocial factors are particularly associated with early-onset depression.

RQ 3: What effect has childhood abuse on the course of late-life depression?
In NESDO, the two-year course of late-life depression in older adults with a history of childhood abuse is significantly worse. This was fully explained by several factors, namely more depressive symptoms at baseline, younger age at depression onset, higher levels of loneliness and neuroticism and more chronic diseases (chapter 6). First of all, this indicates that childhood abuse particularly has an indirect, negative effect on the course of depression, even in late life. This is in line with other studies predominantly in younger adults (Wiersma et al., 2009a; Hovens et al., 2012; Rhebergen et al., 2011), leading to the conclusion that childhood abuse is
significantly associated with the course of depression, independent of age. Surprisingly, in contrast to previous studies, that have shown a dose-response relationship between frequency of childhood abuse and course of depression, especially in younger adults (Kessler et al., 1997; Green et al., 2010; Nanni et al., 2012; Norman et al., 2012), but also in older adults (Ege et al., 2014), we could not confirm this dose-response relationship. This could be due to the fact that just a few participants reported only a single childhood abuse event.

Most interesting were the mediating factors. Depression severity was the strongest factor associated with the course of depression in older adults with a history of childhood abuse. Also the age at depression onset was a significant mediator. A younger age at depression onset has been linked to more depressive symptoms, more depressive episodes and chronic depression (Bernet & Stein, 1999; Zisook et al., 2007; Rhebergen et al., 2012). So, this shows an important interplay between depression characteristics that are influenced by childhood abuse as well. Loneliness was also a significant explanatory factor for a poorer course of late-life depression. This concept consists of different aspects (e.g. van den Brink et al., 2018), such as having less contacts than one would prefer as well as feeling more emotionally isolated. At old age, it has been shown that childhood abuse more often led to smaller social networks and feeling more isolated (Wilson et al., 2006). In our study, only loneliness, not a smaller social network, influenced the course of depression and might be an important aspect to consider in treatment. Wilson et al. (2006) also found childhood abuse was related to neuroticism. We showed that neuroticism was another important mediating factor in the association between childhood abuse and a poor course of late-life depression. Other research in adults of 50 years and older also showed that neuroticism, particularly self-consciousness (shame and embarrassment), was more often reported by individuals with a history of childhood abuse and associated with more severe depressive symptoms (Gamble et al., 2006). Furthermore, this study showed that severe childhood abuse was related to higher levels of neuroticism and might explain why neuroticism only mediated between the association between the childhood abuse index and not the dichotomous childhood abuse variable, and the course of depression. In this study only the mediation effect of neuroticism was investigated. We did not consider other personality traits, particularly since earlier studies showed the strongest associations with neuroticism and diverse findings for extraversion, agreeableness, openness and conscientiousness (Moran et al., 2011; Hengartner et al., 2015). It has been suggested that cognitive styles (e.g. hopelessness) are important coping skills (e.g. Alloy et al., 1999). In younger adults, hopelessness was an important mediator between childhood abuse and a poor course of depression (Hovens et al., 2016). Neuroticism and cognitive styles have also been associated with each other (Hong & Paunonen, 2011). In old age, cognitive styles and its influence on coping skills and depression after childhood abuse could be interesting topics for future research. Lastly, a small, but significant, explanatory factor was the number of chronic somatic diseases. This is in line with earlier cross-sectional findings (chapters 4 and 5), and fits with a declining physical health later in life. Poor physical health was associated with a poor course of depression (Comijs et al., 2015) and has an effect on daily functioning (Ormel et al.,
Answer RQ3: Childhood abuse has a strong negative effect on the course of late-life depression, but this association was fully explained by depression characteristics, loneliness, neuroticism and chronic somatic diseases.

RQ 4: How consistent are retrospective self-reports concerning childhood abuse in older adults?

Based on our findings on test-retest reliability of retrospective reports on childhood abuse, we conclude that these reports were sufficiently reliable, also later in life. Although studies in younger adults were somewhat cautious and careful in their conclusion, most of them concluded the same (e.g. Da Silva & Da Costa Maia, 2013; Naicker et al., 2017). Especially negative (no) answers were highly reliable in our study, while positive (yes) answers were more fluctuating. So, positive answers require more in-depth questioning such as specific examples of the abuse experiences to ensure reliable reports. This is in line with earlier suggestions by other researchers (Yancura & Aldwin, 2009; Hardt et al., 2006). Furthermore, we found (small) differences per type of abuse, where childhood sexual abuse was reported most reliably. Surprisingly, the positive (yes) answers for physical abuse were more fluctuating over time although, at first glance, it seems a more objective type of abuse. Since it was not influenced by depression or cognition, it remains unclear why participants answered differently on two interviews. Only the retrospective reports on childhood emotional neglect were influenced by depression. In 2015, Humphreys & Zeannah argued that it would be useful to distinguish research in two different types of childhood abuse, namely neglect and abuse. Our findings also seem to correspond with their suggestion to split childhood abuse into what they call inadequate (neglect) and harmful (physical abuse) input, experiences. In earlier studies, we did compare emotional neglect and psychological abuse (inadequate input) with physical and sexual abuse (harmful input) and did find different associations with depression (chapter 4). However, this is also a difficult methodological issue since most people experienced more than one type of childhood abuse leading to overlapping categories and distorted conclusions. Other components of childhood adversity in the assessment were shown to be important and could be missing in our study, e.g. financial situation, substance abuse by parents/caregivers, parental mental illness, exposure to criminal behavior (Kessler et al., 2010). However, these components could also be interrelated with the reported childhood neglect and abuse. Furthermore, the duration of the childhood abuse might be important with respect to the reliability of the assessment. Future research can be helpful to further explore this framework.
**Answer RQ4:** Retrospective reports on childhood abuse are sufficient reliable and largely consistent. To enhance its test-retest reliability, it is important to ask concrete and specific questions that can be completed with examples.

**Overarching discussion points**

Overall, several remarks need to be made. First, protective personality characteristics, such as resilience, mastery and coping style, were not included in this thesis. Although we included some factors that could be viewed as the inverse of positive coping and resiliency, it would still be an interesting point of view for future research. In a review, Meng et al. (2018) found 85 studies of low to middle quality that investigated resilience and protective factors after childhood abuse. Although protective factors and resiliency were associated with better (health) outcomes, the authors accentuated that there was wide variation in measurements and outcomes which made it difficult to draw firm conclusions. In NESDO, unfortunately adequate measures on protective factors and resiliency are not available.

Next, previous research has shown that major depressive disorder can be a precursor (prelude, prodromal) for dementia. There is a strong association between late-life depression and dementia (Byers et al., 2011; Haigh et al., 2018). Since, older adults with a history of childhood abuse have a higher chance of developing a depression, their chances to develop dementia may also be higher. It is still unexplored territory, but would be an interesting topic. Attrition in NESDO was associated with the severity of depression and with worse cognition. This might indicate that the most severe cases fell out of the study. However, there was no difference in attrition rates between adults that reported childhood abuse and those who did not, except for physical abuse, which could also mean that a history of childhood abuse is not directly related to cognitive problems. At a younger age, childhood abuse seems to influence cognitive functioning (Sideli et al., 2014). In this study, they showed that childhood abuse was associated with poorer performance on tasks concerning working memory and executive functioning. In addition, childhood abuse is related to brain changes on several levels (Teicher et al., 2016), which might be related to cognitive deficits. So there might be an association between childhood abuse and cognitive function, also later in life. If childhood abuse is also associated with an increased risk on dementia remains to be investigated. Future (longitudinal) research could shed some light on this issue.

Finally, in the general population, the role of gender was an important factor that was suggested to cause differential risks for adult psychopathology after childhood trauma (Tiwari & Gonzalez, 2018). Indeed, Teicher et al. (2003) found gender differences due to childhood adverse experiences and suggested these could be related to the nature of these adverse experiences, and to brain and hormonal developments. Other studies also suggested sex differences related to a differential role of sex steroids in plasticity of brain regions (McEwen,
In a recent meta-analysis, Gallo et al. (2018) concluded that there were no statistically significant gender differences although the associations between childhood abuse and depression/anxiety were larger for women. In our studies, we did consider gender in the statistical analyses; however, odds ratios changed less than 10% in our sample. This suggests that in old age gender is less important in the association between childhood abuse and depression.

**Methodological strengths**
Concerning our studies, several strengths and limitations have to be considered. Major strength of all studies is the extensive assessment of childhood abuse that enquired information on four specific types of childhood abuse using concrete and specified operationalisations. This was shown to be important to get more veracious answers on this sensitive topic (e.g. Hardt et al., 2006; Yancura & Aldwin, 2009). Furthermore, it allowed us to use a categorical (‘yes-no’) as well as a dimensional variable considering childhood abuse and enabled a more thorough investigation. In addition, NESDO is a longitudinal cohort study, meaning that we had two-year and six-year follow-up data. Furthermore, NESDO consists of a large sample size of older adults with detailed information about formal DSM-IV diagnoses (APA, 2000) and related symptom questionnaires, as well as a wide array of (potential) explanatory factors and covariates. Consideration and possibility to incorporate multiple covariates in the statistical analyses allowed us to control for confounding effects, especially those that are important later in life.

**Methodological limitations**
Overall, there has been a lot of research concerning childhood abuse and its consequences in children, adolescents and younger adults (<60 years). It is however difficult to compare our results in older adults with those studies because there is a great variety of measurements and methods limiting the overarching conclusions. For example, some studies looked at one generic question on childhood abuse or adversity, while others considered specific types of childhood abuse or even broader childhood adversities (e.g. Gerritsen et al., 2010; Kessler et al., 2010). There were studies that also looked at the frequency or duration of childhood abuse (Schalinski et al., 2016). Some studies only considered retrospective reports, while others compared those with official records (e.g. Sperry & Widom, 2013). Although the general message is that childhood abuse has a large negative impact on mental health, uniformity in measures and methods seem essential to generate clear and unequivocal answers. Second, information was mostly gathered through retrospective reports in NESDO. Apart from our own study on test-retest reliability of childhood abuse, this concerns also reports about the age at onset. However, Toren et al. (2006) concluded in their study that age at disease onset was reliably and accurately reported with a 10-year interval in adults aged 18-60 years, indicating that we gathered reliable information. Third, studies showed that childhood abuse has also been related to post-traumatic stress disorder (PTSD); however, this information was not available in NESDO.
Furthermore, due to sample size and power issues we were not able to consider specific subtypes, course types or course trajectories of depression, such as atypical or melancholic depression (Lamers et al., 2010; Rhebergen et al., 2012). In addition, a mediation model applies stricter implicit rules to heighten its applicability (Preacher & Hayes, 2008) that, for instance, could lead to a reduced power by using smaller sample sizes. Sixth, in general, a larger amount of statistical tests could lead to type-I error. However, we did not perform a Bonferroni correction since this would have led to an increased chance of making a type-II error for all tests and would not have detected a real association (Perneger, 1998; Gelman et al., 2012). Seventh, the naturalistic design and the use of cross-sectional data in some studies in this thesis impede us to make causal inferences and study changes over time. Furthermore, detailed information on treatment was not available in NESDO. Therefore the impact of various treatment regimens on depressive symptoms in victims of childhood abuse could not be thoroughly examined. No firm conclusions can be drawn on important aspects concerning specific treatments. Last, our results came from a depressed sample of older adults, so no conclusions can be drawn for the general population. However, some results were independent of depression status, so it would be interesting to investigate the impact of childhood abuse in the general population.

Clinical implications: diagnostics
Childhood abuse affects daily life negatively, also later in life, so it requires clinical awareness. Most importantly, it requires skills of the clinician to enquire and detect childhood abuse. One might think only mental health clinicians need to be vigilant but it seems also important in other health areas (physical and public health). In addition, in older adults, it seems important to consider the overlapping symptoms of somatic health and depression, and its impact on functional disabilities. One aspect that needs to be emphasized is that when people answer positively (‘yes’) on questions concerning childhood abuse it requires more in-depth and concrete questioning to ensure the true nature of the childhood experiences. As was already suggested by Yancura & Aldwin (2009) and Hardt et al. (2006), specific and concrete questions heighten the reliability of the answers on personal, confidential issues. Also, independent of its reliability, the combination of late-life depression and positively answered questions about childhood abuse, could give some clues about the underlying causes for the diagnosis and the course of the (depressive) disorder. Childhood abuse is strongly associated with an early onset of depression and the symptoms are often more severe. It gives an indication of the prognosis and might help with treatment selection. However, childhood abuse is also associated with late onset depression, therefore in all cases, when older adults have a depression it is important to take childhood abuse into account during the diagnostic process and during the treatment.

Clinical implications: treatment
When someone has reported a history of childhood abuse, it is important to integrate this in treatment of current psychopathology. Nemeroff (2016) discussed that a history of childhood
abuse often leads to poorer response to (conventional) treatment, suggesting a distinct biological endophenotype for depression and childhood abuse, and should be taken into account to increase treatment effectiveness. In late-life, the treatment selection might be driven by several factors. First of all, it is and should be based on evidence-based guidelines for depression, suggesting pharmacotherapy and/or psychotherapy. A review showed that psychological interventions were effective treatments in late-life depression, although there were fewer studies on pharmacotherapy or combined treatment to draw firm conclusion (Cuijpers et al., 2014). Second, during treatment of depression childhood abuse needs to be taken into account. A study of Williams et al. (2016) showed that there was a poorer response to commonly prescribed antidepressants in adults with a history of childhood abuse, which also seems to depend on the timing of the abuse. This needs to be considered in the treatment selection. From our findings several other factors can be considered, such as psychosocial factors. As was described in chapter 4, we found that social network size and loneliness were partly explanatory of early-onset depression. The focus of treatment in this specific group could be on ways to reduce feelings of loneliness and increase the size of their social network. Furthermore and in line with Tunvirachaisakul et al. (2018), we found several baseline depression characteristics that have a prognostic value, such as age at depression onset and depression severity. A younger age at onset and more severe depression explained a poor course of depression in older adults with a history of childhood abuse. These factors can be assessed at the first contact and might be used as an indication for a more specialized, individualized treatment. It might even be considered to choose upfront for a treatment that has been effective in case of chronic depression such as Cognitive Behavioral Analysis System of Psychotherapy (CBASP; Wiersma et al., 2009b; McCullough, 2003). Next, in older adults, it seems important to consider chronic somatic diseases and its overlap with depression. In chapter 5, we also showed that depression as well as chronic somatic diseases explain increased functional disabilities in childhood abused older adults. Combined treatment for depression as well as chronic somatic diseases might be useful to reduce both diseases as well as functional disabilities in daily life. Availability and access to treatment, for example videoconferencing or blended treatments, can also be considered promising (Renn & Aréan, 2017; Hagan et al., 2014). Furthermore, if childhood abuse becomes the focus of treatment, it could be that the therapeutic relationship and specific relational experiences are important (Parry & Simpson, 2016). It would also be interesting to consider different treatments for childhood neglect versus childhood abuse. Humphreys & Zeanah (2015) suggested to split these forms of abuse based on the way of ‘input’, where neglect is considered as the absence of certain behaviour while abuse is related to the presence of (traumatic) behaviour. Studies on post-traumatic stress disorder (PTSD) considered different treatments for neglect versus abuse childhood experiences (Cloitre et al., 2012; Arntz et al., 2012), and might be interesting to consider in late-life depression as well.
Clinical implications: prevention

Childhood abuse is a major public health issue (e.g. Cuijpers et al., 2011), since it has a large negative impact over the life course. Predominantly, prevention strategies need to focus on children and their development (Garner et al., 2015). After childhood, other preventive strategies can be considered. As was mentioned earlier, childhood abuse is associated with a younger age at depression onset, and psychosocial functioning partly explained the earlier age of depression onset (chapter 4). So, prevention of late-life depression could focus on social skills, social network and loneliness. Creating new or maintaining existing social contacts can be helpful later in life. In addition, effective strategies have been found to decrease feelings of loneliness such as structured group interventions, experimental interventions using new technologies, focusing on new maladaptive social cognitions (Lang, 2001; Hagan et al., 2014; Masi et al., 2011).

Future research

Qualitative studies could be very informative and enhance knowledge of important but less quantifiable factors. This could consist of in-depth interviews on childhood abuse, coping strategies, mental health issues and resiliency. As was mentioned earlier, resiliency was missing in our study, but seems quite important (Meng et al., 2018). Qualitative studies might be particularly interesting for the mentally healthy adults with a history of childhood abuse. Related to that, a study in the general population could be helpful and might give more clues on differentiating factors between childhood abused adults with and without health issues. Currently, our sample consisted predominantly of depressed older adults, but it would have been interesting to study the effects of childhood abuse, independent of depression, e.g. study the association between childhood abuse and cognitive problems. Especially since some reviews suggest that current findings in psychiatry might have been confounded by the independent effect of childhood abuse (e.g. Teicher et al., 2016; Heim et al., 2008). This could mean that studies need to adjust for childhood abuse, but particularly implicates that more research is needed on the specific effects of childhood abuse. Furthermore, long-term prospective studies are needed to enable reliable life-course data, particularly life course developments and enabling comparison of younger and older adults. Although many studies focused on depression, Haigh et al. (2018) already showed that there is insufficient evidence of depressive symptom profiles over the life course; let alone, the impact of childhood abuse. It would be valuable to specify long-term course trajectories, as was already done in younger adults (Lamers et al., 2010; Rhebergen et al., 2012). In addition, as a future direction, it would be interesting to see which treatment is most effective for late-life depression in adults with a history of childhood abuse and what should be the primary focus of the treatment. So, evaluating the effectiveness of medication for this specific group of patients or a randomized-controlled trial comparing psychological treatments for depression with and without a primary focus on traumatic events would be interesting.
Concluding remarks

Childhood abuse affects a person’s life with an enormous burden, also later in life: a higher risk of depression, a poorer course of depression, chronic somatic diseases and functional disabilities. As became clear in this thesis, it is truly important to enquire childhood adversities independent of timing and nature of the problem, especially when someone is presenting his or herself with mental health problems. In-depth questioning and empathic listening are key components when enquiring something so important and disastrous.

Mrs. de Jonge, 75 years old, was chronically depressed and got treatment from mental health care for a long period of time. Recently, she told a mental health care practitioner for the first time that she was sexually abused during her childhood. She wanted to know whether she could get some help with childhood memories that became more lively with increased care for her grandson. It took her a long time to be able to speak about her childhood experiences. Mrs. de Jonge had difficulty trusting people, and hence found it difficult to start talking about this topic. She was grateful that someone actively explored her childhood experiences with her.
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Nederlandse Samenvatting
Negatieve jeugdervaringen als verwaarlozing en mishandeling kunnen ernstige en langdurige consequenties hebben, waaronder psychische problemen. Al vele jaren blijft het aantal kinderen dat slachtoffer is van mishandeling en/of verwaarlozing in Nederland gelijk, en wordt geschat op 118.000 kinderen per jaar. Er is weinig bekend over de effecten van negatieve jeugdervaringen op de psychische gezondheid op latere leeftijd. Ook in de klinische praktijk van de ouderenpsychiatrie wordt er tot nog toe weinig aandacht aan de vroege jeugdervaringen besteed.

**Jeugdtrauma: verwaarlozing en mishandeling in de jeugd**


Jeugdtrauma’s kunnen negatieve consequenties hebben op vele levensgebieden. Er zijn veel studies gedaan bij uiteenlopende leeftijdsgroepen. Kort gezegd kwam hieruit naar voren dat jeugdtrauma’s negatieve effecten hebben op ons brein, op ons stresssysteem en op andere neurobiologische processen. Op lange termijn zijn er ook allerlei andere gevolgen, zoals een ongezondere leefstijl, meer problemen met lichamelijke gezondheid en meer problemen op sociaal en interpersoonlijk vlak. Bovendien hebben verwaarlozing en mishandeling in de jeugd een sterke invloed op de geestelijke gezondheid en is het geassocieerd met een breed scala aan psychopathologie, zoals post-traumatische stress stoornis, depressie en bepaalde persoonlijkheidsproblematiek. De ernst van het jeugdtrauma (duur, frequentie en meerdere typen) versterkt de associatie met psychopathologie en leidt vaak tot co-morbiditeit. Tot nu toe is veel onderzoek gedaan bij kinderen, adolescenten en jongere volwassenen. Echter de verklarende, mediërende factoren tussen jeugdtrauma en de gevolgen op latere leeftijd zijn nog grotendeels onbekend.

**Depressieve stoornis**


Doelstellingen en onderzoeksvragen
Volwassenen met een jeugdtrauma ontwikkelen vaker een depressie dan volwassenen zonder jeugdtrauma. Meestal ontstaat de depressie al op jonge volwassen leeftijd, echter sommige mensen krijgen hun eerste depressie pas op latere leeftijd. Jeugdtrauma vergroot dus ook bij ouderen de kans op een slechte(re) geestelijke gezondheid. Mogelijk spelen bij ouderen onderliggende factoren die samenhangen met veroudering een rol bij het ontwikkelen van een depressie.

Het hoofddoel van dit proefschrift was dan ook om de associatie tussen jeugdtrauma en depressie bij ouderen verder te ontrafelen. Tevens was het doel om de betrouwbaarheid van retrospectieve zelfrapportages over jeugdtrauma te onderzoeken. We hadden vier onderzoeksvragen. Eerst hebben we gekeken naar het verband tussen jeugdtrauma en twee mogelijke onderliggende biologische mechanismen, namelijk de HPA-as en telomeerlengte. Vervolgens onderzochten we de rol van chronische somatische ziekten, psychosociale - en leefstijl factoren in de associatie tussen jeugdtrauma en respectievelijk depressie en functionele beperkingen. Ten derde waren we geïnteresseerd in de associatie tussen jeugdtrauma en het beloop van depressie op latere leeftijd. Ten vierde hebben we de betrouwbaarheid van zelfrapportages over jeugdtrauma's bij ouderen volwassenen onderzocht. De jeugdtrauma vragenlijst vraagt terug te kijken op gebeurtenissen die lang geleden hebben plaatsgevonden, de vraag is hoe betrouwbaar die herinneringen zijn. Doordat de vragenlijst tweemaal is afgenomen, kon de betrouwbaarheid van deze meting worden onderzocht.

Onderzoekspopulatie
De Nederlandse Studie naar Depressie bij Ouderen (NESDO) is een longitudinale cohortstudie die in 2007 is begonnen met de werving van oudere volwassenen bij huisartsen en GGZ-instituties in vijf regio's in Nederland. Bij aanvang waren er 510 deelnemers, 387 depressieve

Nederlandse Samenvatting | 169
en 132 niet-depressieve oudere volwassenen van 60 jaar en ouder (gemiddeld 70,6 jaar). Twee jaar later werden alle deelnemers opnieuw uitgenodigd, waarna 83,4% (401 volwassenen) meedenen aan het tweede interview. Zes jaar na de basismeting deden nog 299 van de 510 deelnemers mee aan het derde interview. Uitval was voornamelijk te wijten aan overlijden en mentale problemen, met name cognitieve stoornissen, en weigering (geen interesse of geen tijd). Elk face-to-face interview omvatte diagnostische instrumenten voor depressie, namelijk een gestructureerd klinisch interview en een vragenlijst om de ernst van de depressie in kaart te brengen. Met een interval van zes jaar, werd de deelnemers gevraagd naar mogelijke jeugdtrauma’s, waarbij de eerder beschreven vier typen jeugdtrauma’s werden uitgevraagd. Daarnaast omvatte het interview een scala aan vragenlijsten en tests waardoor een goede indruk verkregen kon worden van de lichamelijke en psychische gezondheid en het dagelijks functioneren.

Onderzoeksresultaten

Onderzoeksvraag 1: Welke rol spelen biologische mechanismen, zoals telomeerlengte en de hypothalamus-hypofyse-bijnier (HPA-)as, in de associatie tussen jeugdtrauma en depressie op latere leeftijd?

In hoofdstuk 2 is gekeken naar de invloed van psychosociale stress op biologische veroudering bij oudere volwassenen. Psychosociale stress omvat in ons onderzoek jeugdtrauma, maar ook recente negatieve gebeurtenissen en eenzaamheid, en kan leiden tot mentale en lichamelijke gezondheidsproblemen. Studies uitgevoerd bij jongere volwassenen laten zien dat psychosociale stress er voor kan zorgen dat het menselijk lichaam sneller verouderd. Deze biologische veroudering wordt in kaart gebracht door naar de lengte van telomeren te kijken. Telomeren bevinden zich aan de uiteinden van onze chromosomen en beschermen ons DNA. Wanneer men ouder wordt, worden deze telomeren vanzelf korter, maar er zijn factoren die het verouderingsproces kunnen versnellen. Wanneer telomeren te kort worden, sterven cellen in ons lichaam af en kunnen gezondheidsproblemen optreden. Binnen NESDO hebben wij onderzocht of ouderen die stressvolle psychosociale gebeurtenissen hebben meegemaakt, kortere telomeren hebben. Het recent of in de jeugd ervaren van stressvolle psychosociale gebeurtenissen blijkt geen verband te hebben met telomeerlengte op latere leeftijd. Met andere woorden, ouderen die stressvolle psychosociale gebeurtenissen hebben meegemaakt, hebben vergelijkbare telomeerlengte als ouderen die deze gebeurtenissen niet hebben meegemaakt. Bij jongere volwassenen werd in eerder onderzoek wel een verband gevonden tussen stressvolle psychosociale gebeurtenissen en telomeerlengte. Mogelijk zijn ouderen gedurende hun leven blootgesteld geweest aan een verscheidenheid van factoren die (gezamenlijk) invloed hebben op telomeerlengte (zoals een ongezonde leefstijl of ziekte), waardoor het effect van stressvolle psychosociale gebeurtenissen niet terug te zien is.

In hoofdstuk 3 hebben we gekeken of het stresshormoon cortisol een rol speelt in de samenhang tussen jeugdtrauma en depressie op latere leeftijd. Cortisol is een hormoon dat een
rol speelt in de HPA-as en dat we nodig hebben om goed op stress te kunnen reageren. Het is
altijd in ons lichaam aanwezig. ‘s Morgens, bij het wakker worden en opstaan is de concentratie
van dit hormoon het hoogst en een uur na ontwaken neemt het weer af (dit noemen we de
cortisol ochtendcurve). Studies bij jongere volwassenen hebben al aangetoond dat bij mensen
die te maken hebben gehad met verwaarlozing en mishandeling in hun jeugd, dit patroon er
anders uit ziet, en ons biologische stress systeem dus anders werkt. Bij depressieve ouderen is
dit nog niet eerder onderzocht. Uit ons onderzoek blijkt dat ouderen met jeugdtrauma een
lagere cortisol waarde hebben bij het wakker worden, of ze nu wel of niet depressief zijn. Dit
zou kunnen komen doordat mensen die als kind mishandeld zijn, gevoeliger zijn geworden voor
teveel cortisol en deze hormoonproductie sneller stopt, zodat er minder cortisol in het lichaam
komt. Bij ouderen die als kind mishandeld zijn zou het lichaam zich zo hebben aangepast dat ze
hier gevoeliger voor zijn geworden, wat er toe leidt dat ze juist minder cortisol aanmaken bij het
wakker worden. Ook vonden we in dit onderzoek dat er grotere verschillen waren over tijd in de
cortisol ochtendcurve als er sprake was van psychologische en/of seksuele mishandeling, maar
enkel bij deelnemers die niet depressief waren. Dit zou erop kunnen wijzen dat depressie het
effect van jeugdtrauma’s op het stresshormoon cortisol modificeert. Oudere volwassenen die
jeugdtrauma rapporteren zouden dus negatiever beïnvloed kunnen worden door stress en
stressvolle gebeurtenissen dan ouderen zonder jeugdtrauma. Dit moet echter nog verder
onderzocht worden. Deze uitkomsten helpen om beter te begrijpen hoe jeugdtrauma bijdraagt
aan de grotere kans op een depressie op latere leeftijd.

Antwoord: Op latere leeftijd is jeugdtrauma geassocieerd met een dysregulatie van de HPA-as,
maar niet met telomere lengte. De associatie tussen jeugdtrauma en dysregulatie van de HPA-as is
onafhankelijk van depressie.

Onderzoeksvraag 2: Wat is de impact van jeugdtrauma op psychosociale en leefstijl factoren,
chronisch somatische ziekten, depressiekaracteristieken en functionele beperkingen?

In hoofdstuk 4 hebben we gekekken of psychosociale factoren, met name partnerstatus, de
omvang van het sociale netwerk en gevoelens van eenzaamheid, de associatie tussen
jeugdtrauma en zowel vroeg- als laat-ontstane depressies verklaren. In het bijzonder waren we
génteresseerd of de sterkte van het effect verschilde voor vroeg- en laat-ontstane depressie.
Over het algemeen vonden we dat jeugdtrauma significant geassocieerd is met zowel vroeg- als
laat-ontstane depressie. Een kleiner sociaal netwerk en meer gevoelens van eenzaamheid
verklaarden een deel van het verband tussen jeugdtrauma en depressie met een jongere
ontstaansleeftijd. Dit effect was het grootst voor emotionele verwaarlozing en psychologische
mishandeling. Eenzaamheid bleek de sterkst verklarende factor. Er werden geen psychosociale
verklarende factoren gevonden voor de associatie tussen jeugdtrauma en depressie met een
latere ontstaansleeftijd.

**Antwoord:** Jeugdtrauma is geassocieerd met een vroeg(er) ontstaan van de depressie, ernstiger depressieve symptomen, meer chronische somatische ziekten en meer functionele beperkingen in het dagelijks leven. Psychosociale factoren waren voornamelijk geassocieerd met een vroeg ontstane depressie.

**Onderzoeksvraag 3: Welk effect heeft jeugdtrauma op het beloop van depressie op latere leeftijd?**

In hoofdstuk 6 hebben we gekeken naar het effect van jeugdtrauma op het beloop van depressie bij oudere volwassenen. Uit eerder onderzoek weten we dat een depressie bij jongere volwassenen (<65 jaar) die jeugdtrauma hebben meegemaakt vaker een chronisch beloop heeft. Dit betekent dat de depressieve klachten langer duren. Er is echter weinig bekend over de impact van jeugdtrauma op het beloop van depressie bij ouderen en over de factoren die de chroniciteit van depressie kunnen verklaren. Eerder in het NESDO-onderzoek is gevonden dat verwaarlozing en mishandeling in de jeugd vaker voorkomt bij ouderen met depressie dan bij ouderen zonder depressie. Wij hebben nu onderzocht hoe het twee-jaars beloop is van depressie bij ouderen die te maken hebben gehad met jeugdtrauma en welke factoren dit beloop kunnen verklaren. Wij vonden dat deze groep een grotere kans heeft om na twee jaar nog steeds of opnieuw depressief te zijn. En dat dit verklaard zou kunnen worden doordat zij vaak meer depressieve symptomen hadden op meting één, dat zij vaak jonger waren ten tijde van de eerste depressieve episode, neurotischer waren, zich vaak eenzamer voelden en meer chronische ziekten hadden. Vooral de ernst van de depressie lijkt een belangrijke verklarende factor te zijn voor een slechter beloop. Uit deze studie blijkt dus dat jeugdtrauma, in combinatie met deze verklarende factoren, een belangrijke rol spelen in een meer chronisch beloop van depressie. Daarom is het belangrijk dat in de praktijk enerzijds expliciet gevraagd wordt naar ervaringen uit de jeugd, en anderzijds gekeken wordt naar de aanwezigheid van genoemde andere verklarende factoren, omdat deze combinatie het beloop van een depressie lijken te beïnvloeden.
Antwoord: Jeugdtrauma heeft een sterk negatief verband met het beloop van depressie op latere leeftijd. Dit verband werd volledig verklaard door kenmerken van de depressie, eenzaamheid, neuroticisme en chronisch somatische ziekten.

Onderzoeks vraag 4: Hoe consistent zijn retrospectieve zelfrapportages over jeugdtrauma bij oudere volwassenen?

In hoofdstuk 7 wilden we onze bevindingen valideren door de betrouwbaarheid van retrospectieve zelfrapportages over jeugdtrauma’s bij oudere volwassenen te onderzoeken. Er bestaat al jarenlang discussie over de betrouwbaarheid en validiteit van verkregen informatie rond verwaarlozing en mishandeling in de jeugd. Hoewel de redenen voor onder- of overrapportage, zoals een slechter geheugen, op latere leeftijd prominenter kunnen zijn, zijn daar geen studies naar verricht. Wij keken naar de consistentie van retrospectieve zelfrapportages van twee interviews en de rol van leeftijd, het geheugen en depressie. Onze resultaten lieten zien dat de meeste antwoorden consistent waren (ja-ja of nee-nee) op twee afzonderlijke interviews, variërend van 85 tot 94%. Dit was het meest stabiel met betrekking tot de nee-antwoorden, terwijl meer fluctuaties te zien waren op ja-antwoorden. De betrouwbaarheid was matig tot goed, maar enigszins afhankelijk van het type jeugdtrauma. Van alle soorten jeugdtrauma werd seksueel misbruik het meest betrouwbaar gerapporteerd in zowel bevestigende (ja-) antwoorden als ontkennende (nee-) antwoorden, terwijl de betrouwbaarheid van ja-antwoorden op fysiek misbruik het laagst was. De consistentie van de antwoorden werd niet significant beïnvloed door leeftijd of cognitie. Depressie leek de consistentie van antwoorden op enkel emotionele verwaarlozing te beïnvloeden, maar niet op andere vormen van jeugdtrauma.

Antwoord: Retrospectieve zelfrapportages over jeugdtrauma zijn voldoende betrouwbaar en grotendeels consistent. Om de betrouwbaarheid te vergroten is het belangrijk om concrete en specifieke vragen te stelen die aangevuld kunnen worden met voorbeelden.

Klinische implicaties

De resultaten van dit onderzoek hebben implicaties voor de diagnostiek, behandeling en preventie van depressie bij ouderen. Allereerst is het nodig dat men zich binnen (maar ook buiten) de geestelijke gezondheidszorg bewust wordt van de langdurige gevolgen van jeugdtrauma. Dit vraagt vaardigheden van de clinicus om jeugdtrauma’s te detecteren en uit te vragen. Als men ‘ja’ antwoordt op, bij voorkeur concrete en specifieke vragen naar jeugdtrauma’s, dan is het van belang om door te vragen en voorbeelden te horen om meer helderheid te krijgen over de jeugdervaringen. Bij oudere volwassenen lijkt het bovendien van belang om rekening te houden met overlappende symptomen van lichamelijke gezondheid en depressie, en de impact ervan op het dagelijks functioneren. Onafhankelijk van de betrouwbaarheid van antwoorden, kunnen positief beantwoorde vragen over jeugdtrauma’s en
depressie op latere leeftijd aanwijzingen geven over onderliggende factoren en het beloop van de depressieve stoornis. Daarnaast is het belangrijk om kennis over jeugdtrauma te integreren in de behandeling van de huidige psychopathologie. Uit de literatuur blijkt vaak dat aanwezigheid van jeugdtrauma een slechtere respons geeft op conventionele behandeling, waarmee het dus vraagt om behandeling die rekening houdt met of meer gericht is op jeugdervaringen. Toekomstig onderzoek zou hier meer licht op kunnen werpen. Omdat verwaarlozing en mishandeling in de jeugd en de effecten ervan, naast een persoonlijke negatieve impact, een belangrijk probleem is in de huidige maatschappij, blijft het van belang om aandacht te hebben voor preventieve aanpak.

**Tot slot**

Jeugdtrauma geeft veel lijdensdruk gedurende het leven, ook op latere leeftijd: een hoger risico op depressie, een slechter beloop van depressie, chronische somatische ziekten en meer functionele beperkingen. Zoals in dit proefschrift duidelijk wordt, is het belangrijk om aandacht te hebben voor jeugdtrauma’s, onafhankelijk van timing en aard van het probleem, en vooral wanneer iemand geconfronteerd wordt met psychische problemen.
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Dankwoord
Het is af! Wie had dat gedacht, het is afgerond met of ondanks een brede interesse, vele werkzaamheden en onverwachte gebeurtenissen. Dit dankwoord geeft een fantastische ruimte om mijn dankbaarheid te uiten, hoe lastig en eigenlijk ontoereikend woorden ook kunnen zijn, want een proefschrift schrijven doe je niet in je eentje. Dankbaarheid kun je leren – gek genoeg vooral als het anders loopt dan verwacht - geeft nieuwe energie en is iets wat mij bij zal blijven. Dankbaar ben ik voor alle mensen die om mij heen stonden, mij geholpen hebben als het tegenzat en plezier gaven in ieder onderdeel en op vele momenten in dit proces. Een aantal groepen en individuen wil ik hier met aandacht noemen.

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Met mijn liefsten wil ik natuurlijk het leukste vieren! Mijn dankbaarheid is groot en niet in genoeg woorden uit te drukken!
Curriculum Vitae

Ilse Wielaard was born on September 17th, 1987 in Vlaardingen, the Netherlands. She graduated from high school in 2005 at the Accent College Reviusplein in Maassluis. She studied Psychology at the Utrecht University, where she got her bachelor’s degree in Neuropsychology in 2008. She continued her education with the research master ‘Neuroscience and Cognition’ and combined this in 2009 with a master Neuropsychology. During this period she did a scientific internship at the department of neurology at the UMC Utrecht and a second internship at the psychiatry department of the AMC Amsterdam. At the same time she did her clinical internship at Aveant, which is an organization for nursing homes, where she also started working as a psychologist. In 2011 she finished her master ‘Neuroscience and Cognition’, and finished her master Neuropsychology cum laude. After her graduation, she worked one year as a psychologist at stichting Humanitas in Rotterdam. In December 2012 she started at GGZ inGeest at the research department as a PhD-student and at the department for old age psychiatry as a psychologist. At the same time she started her training to become a mental health care psychologist (GZ-psycholoog), which she finished in 2015. As PhD-student she examined the role of childhood trauma in late-life depression using data from the Netherlands Study of Depression in Older Persons (NESDO), which resulted in this dissertation. Currently she is working at the department for Anxiety and Mood Disorders from GGZ inGeest as a mental health care psychologist, trained to become a specialist (clinical psychologist), which she started in January 2018.
List of publications

Published


Submitted for publication


Other publications


Dissertation series

Department of Psychiatry, VU University Medical Center


UITNODIGING

Voor het bijwonen van de 
openbare verdediging van het 
proefschrift:

Childhood Abuse and 
Late-Life Depression

Vrijdag 14 december 2018
om 11.45 uur in de aula van 
de Vrije Universiteit,
De Boelelaan 1105

te Amsterdam

Na afloop van de 
verdediging bent u
van harte welkom

op de receptie

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