The effect of trauma onset and frequency on PTSD-associated symptoms

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ABSTRACT

Background: Different trauma characteristics have been suggested to lead to distinct symptom profiles. This study investigates the effect of two trauma characteristics, age of onset and frequency, on PTSD symptom profiles.

Methods: Trauma characteristics (childhood versus adulthood trauma and single versus multiple trauma), psychiatric diagnosis, PTSD severity, depressive symptoms, dissociation, guilt, shame, anger, and interpersonal sensitivity were assessed in 110 PTSD outpatients.

Results: Single versus multiple trauma and childhood versus adulthood trauma groups did not differ in depressive symptom and co-morbidity. Multiple trauma patients reported more dissociation, guilt, shame, and interpersonal sensitivity than those that experienced single trauma. Anger of multiple trauma patients was more often directed towards themselves, whereas anger in single trauma patients was more often directed towards others. Childhood trauma patients reported more dissociation and state anger than adulthood trauma patients. However, with the exception of multiple trauma patients having more dissociation and shame than those with single trauma, all differences disappeared after controlling for PTSD severity.

Limitations: This study is a first step in unraveling the impact of different trauma characteristics. Causal inferences are limited, though, because of the cross-sectional design.

Conclusions: The results suggest that experiencing trauma at young age or multiple times may lead to different symptom profiles but these are, with the exception of dissociation and shame, dependent on PTSD severity. These results support the proposed DSM-V criteria in which these symptoms appear as part of the disorder, and stress the importance of early treatment.

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1. The effect of trauma onset and frequency on PTSD symptom profiles

Trauma has been associated with a variety of psychiatric problems, with posttraumatic stress disorder (PTSD) being the most common one. Patients with PTSD all suffer from approximately the same symptoms that are described in the DSM-IV (APA, 2000): reexperiences of the traumatic event, avoidance and emotional numbness, and hyperarousal symptoms. This may suggest that PTSD forms a uniform psychiatric disorder. However, some researchers posited that some trauma characteristics (like childhood trauma and multiple traumatization) may lead to different symptom profiles, such as simple and complex PTSD. The present study explores this hypothesis by investigating whether specific trauma characteristics (single versus multiple and childhood versus adulthood trauma) are associated with differences in symptoms that are associated with PTSD but not included in the DSM-IV PTSD criteria: dissociation, guilt, shame, anger, interpersonal sensitivity.

The DSM-IV (APA, 2000) constellation of PTSD symptom clusters is quite consistently found in studies using factor analysis. Mostly, a 3 or 4 cluster solution delineating re-experiencing, avoidance (emotional numbing), and hyper-arousal symptoms, seems to be the best fit (Admundson et al., 2004). However, in addition to the DSM-IV symptom clusters, some other symptoms are thought to be strongly associated
with PTSD. These are described under “associated features” in the DSM-IV, and resemble symptoms that are sometimes referred to as “complex PTSD” (Herman, 1992). In addition to DSM-IV PTSD symptoms, complex PTSD also includes disturbances in affective and interpersonal self-regulatory abilities, such as alterations in consciousness (e.g., dissociation), self-perception (e.g., guilt and shame), affect regulation (e.g., anger modulation), and relationships with others (distrust and interpersonal sensitivity) (Roth et al., 1997). A complex symptom presentation is considered to result from sustained or multiple traumas, especially if these occur during childhood (Cloitre et al., 2009).

Studies on childhood trauma often concern repeated or sustained trauma. Therefore, it is difficult to clarify whether the distinct consequences are due to the chronic character of the trauma or to the young age of the victim. Animal studies have found different effects for single versus multiple trauma. For example, multiple stressful events (shock) were associated with changes in fear learning, whereas one event was not (Rau and Fanselow, 2009). Enhanced startle was found for adult rats that were exposed to stress in both juvenility and adulthood, compared to those exposed in juvenility or adulthood only (Avital and Richter-Levin, 2005). Human studies have also suggested distinct consequences for single versus multiple trauma, such as differences in self-confidence (Allen and Lauterbach, 2007), coping abilities (i.e., denial and disengagement; Dale et al., 2009), vagal regulation (Dale et al., 2009) or automatic freezing-like responses (Hagenaaars et al., 2011).

Childhood trauma is thought to be of great influence because it impairs the developmental processes like emotion regulation and interpersonal behaviors (e.g., Shipman et al., 2005). Early life stress would more easily result in changes in ones neurobiological profile, like long-term alternations in the amygdala (Tsoory et al., 2008) or increased sensitization of the neuroendocrine stress response, which may serve as a vulnerability factor for later psychopathology (Heim and Nemeroff, 2001). Schore and Schore (2008) posit that the early formation of secure attachment mediates stress regulation, central and autonomic nervous system. Chronic childhood trauma would disrupt this process. Animal studies have found that early sustained stress affected adult fear-related responses (freezing), suggesting long-lasting changes in the animals defense response system (e.g., Sanders and Knoepfler, 2008). Human studies also suggest different symptom profiles for childhood versus adulthood trauma. For example, childhood trauma (age <13 years) was associated with an elevated risk for depression (Maercker et al., 2002). Childhood trauma victims also had different baseline and reactive cortisol levels relative to adulthood trauma victims (Santa Ana et al., 2006). As stated before, it is not clear whether this effect is the result of the sustained character of the trauma or the young age of the subjects, or possibly an interaction between these two.

In sum, age of onset and trauma frequency are suggested to play an important part in the development of impairments in affect regulation and interpersonal functioning. As most studies included victims of childhood abuse, it is not clear which aspect is responsible for the development of a complex symptom profile. Furthermore, although dissociation has been studied relatively frequently (although mainly in chronic childhood versus single adulthood trauma), other aspects of complex PTSD have not or scarcely been investigated. The present study therefore aimed to investigate whether the age of onset and trauma frequency are associated with specific PTSD-associated symptoms. More specifically, we tested whether single versus multiple trauma and childhood versus adulthood trauma differed in terms of dissociation, guilt and shame, anger intensity and direction, and interpersonal behavior style. Secondly, it was tested whether these differences were independent or could be explained in terms of PTSD severity.

2. Method

2.1. Participants

One-hundred-and-ten patients with PTSD were referred to an outpatient clinic specialized in the treatment of anxiety disorders. Patients were included if they met the DSM-IV criteria for PTSD according to the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). Mean age of the sample was 35.05 (SD 11.38; range 18 to 63). Twenty-four patients (22%) were male, 86 (78%) were female. Level of education was low in 28 patients (25%), medium in 47 patients (43%), and high in 35 patients (32%). The patients suffered from mixed traumas: sexual violence (n = 23, 21%), physical violence (n = 31, 28%), both sexual and nonsexual violence (n = 17, 16%), emotional abuse or neglect (n = 10, 9%), accidents (n = 12, 11%), and other (n = 17, 16%). The mean time since the traumatic event had taken place was 10 years and 7 months (SD = 11.10 years, range 6 months to 52 years). Twenty-eight patients (25%) were diagnosed with co-morbid panic disorder with agoraphobia, 18 (16%) with social phobia, 14 (13%) with generalized anxiety disorder, 3 (3%) with obsessive compulsive disorder, 31 (31%) with a current depressive disorder, 8 (7%) with somatoform disorder, 6 (5%) with eating disorder, 5 (5%) with substance abuse, and 1 (1%) with psychotic disorder. In addition, 15 patients (14%) were diagnosed with cluster C personality disorder, 1 (1%) with cluster A personality disorder, and 7 (6%) with personality disorder NOS. In total, 73 patients (66%) had at least one co-morbid axis I or II DSM-IV disorder.

2.2. Measures

2.2.1. Mini-International Neuropsychiatric Interview (MINI)

The MINI (Sheehan et al., 1998a) is a structured interview that was used to establish DSM-IV axis I psychiatric diagnoses. The MINI consists of closed questions that are based on DSM-IV and ICD-10. Interrater reliability of the MINI is good with kappa values of all diagnostic subscales above .75 (Sheehan et al., 1998b). In addition, comparison of the MINI with the SCID-P shows that in general, MINI-diagnosis were characterized by good or very good kappa values (except for current drug dependence with a kappa below .50; kappa = .78 for PTSD), and very good operating characteristics (Sheehan et al., 1998b).

2.2.2. Structured Clinical Interview for DSM-III-R (SCID-II)

SCID-II is a standardized, semi structured, diagnostic interview for diagnosing DSM-IV axis I personality disorders (First et al., 1997). The reliability of the SCID-II showed to be good. Kappa's ranged from .77 for obsessive–compulsive personality disorder to .82 for avoidant personality disorder in an outpatient population. Weighted kappa for all
personality disorders was .80 (Arntz et al., 1992). The Intraclass Correlation Coefficient (ICC) indicated fair to excellent agreement (range .41 to .88), except for the dependent personality disorder (ICC< .40; Weertman et al., 2003).

2.2.3. Clinician-Administered PTSD Scale (CAPS)

The CAPS is a structured interview designed to test for the presence of the 17 DSM-IV-TR criteria for PTSD and to establish PTSD severity in the previous month (Blake et al., 1995). Each symptom is scored on two dimensions, i.e., frequency and intensity, using 5-point scales. The interrater diagnostic agreement proved excellent (Blake et al., 1990), and test-retest reliability for the three symptom clusters (r = .77 to .96) and total scale (r = .90 to .98) was good (Blake et al., 1995). The internal consistency for all CAPS items proved to be high (α=.94; Blake et al., 1995) and the concurrent validity adequate (correlation with Mississippi Scale for Combat-related PTSD: r = .70 to .91, correlation with MMPI PTSD subscale r = .77 to .84; Blake et al., 1990; Blake et al., 1995).

2.2.4. Beck Depression Inventory (BDI)

The BDI (Beck et al., 1961) is a self-report questionnaire that measures the severity of depressive symptoms. It consists of 21 items that can be rated from 0 to 3. The internal consistency is high for both psychiatric and nonpsychiatric samples (α coefficients range from .76 to .95 and .73 to .92 respectively). The concurrent validity is high in both psychiatric and nonpsychiatric samples (correlation with clinical ratings: r = .55 to .96, correlation with Hamilton Rating Scale for Depression: r = .61 to .86, correlation with Zung: r = .57 to .86, correlation with MMPI-D: r = .41 to .75; Beck et al., 1988).

2.2.5. Trait dissociation

Was measured by the Dissociative Experiences Scale (DES; Bernstein and Putnam, 1986; Carlson and Putnam, 1993). The DES is a self-rating scale that measures the tendency to experience dissociative experiences in daily life. The scale consists of 28 items and the total range is from 0 to 100. For each item the person has to state how often a specific dissociative symptom occurs. The convergent validity compared to other instruments that measure dissociation showed to be very good (overall Cohen’s d = 1.82). Reliability of the DES also showed to be good (mean alpha reliability = .93; Van IJzendoorn and Schuengel, 1996).

2.2.6. Guilt

Guilt was assessed by a composite score that was calculated by adding item 26 (frequency and intensity of guilt about behavior during the event) and item 27 (frequency and intensity of guilt about surviving the event) of the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). This new composite score had a range from 0 to 16.

2.2.7. Shame

Shame was assessed using one item addressing any experiences of shame in the previous week. This item ranged from 0 (not at all) to 3 (all the time).

2.2.8. Anger intensity and anger direction

State and trait anger were assessed using the State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988). The STAXI is a 20-item self-report questionnaire that measures state anger (10 items) and trait anger (10 items) using a 4-point Likert scale. Trait anger is defined as a stable tendency to react with anger, rage or frustration. State anger is defined as a temporary emotional state. Cronbach’s α of the trait items was found to be above .8 (Bishop and Quah, 1998) and the scale was shown to have satisfactory validity and good reliability (Evers et al., 1996). Anger direction was assessed using one item with 5 answering options indicating that the anger was directed only or mainly towards others, only or mainly towards oneself, or not relevant as they were not angry. Patients that scored “not relevant” were excluded from the anger direction-analyses, leaving a total of 91 patients for this specific research question.

2.2.9. Interpersonal sensitivity

The Symptom Check List 90 (SCL-90; Derogatis et al., 1973) is a widely used measure of current psychopathological symptom severity. The Interpersonal Sensitivity (SCL-IS) subscale of the SCL-90 was used in the present study. This 18 item subscale refers to interpersonal sensitivity and distrust, including for example “being critical towards others”. Items are scored on a 5-point Likert scale.

2.3. Procedure

Participants cooperated on a voluntary basis. Before the treatment started they were screened using the MINI, SCID-II, and CAPS to establish diagnosis and co-morbidity. Age of onset and trauma frequency were determined in an open interview, asking the patients what traumas they had experienced, how many times each of these traumas had happened and at what age it had happened. DSM-IV criteria A1 and A2 were used to define trauma. A trauma was categorized as childhood trauma if it had taken place before the age of 16; a trauma was categorized as multiple if the same event happened more than once or if several different events had happened. Patients then completed all questionnaires.

3. Results

3.1. Single versus multiple trauma

Single (n = 51) and multiple (n = 59) trauma groups were first compared on gender, age, depressive symptoms and co-morbid psychopathology to make sure that these variables would not be responsible for any of the group-differences. There were no group differences in gender (χ²(1, N = 110) = 1.77, p = .18), age (t(108) = .92, p = .36), or BDI (t(108) = −.59, p = .56). Neither did the single and multiple trauma groups differ in the number of co-morbid axis I disorders (t(108) = −.58, p = .56) or the number of personality disorders (t(108) = −.33, p = .74). Means and SDs are listed in Table 1. As none of these variables differed between groups, all following analyses were done using t-tests for independent groups.

Independent t-tests showed that those who experienced multiple trauma had higher DES scores than their single trauma counterparts (t(108) = −3.24, p = .002; Fig. 1), see Table 2 for
mean scores. Patients with multiple trauma also had more guilt ($t(108) = -2.31, p = .02$) and shame ($t(108) = -2.91, p = .004$) than those who experienced single trauma (Fig. 1).

Single and multiple trauma patients did not differ on STAXI-trait ($t(108) = .96, p = .33$). STAXI-trait tended to be higher in patients with multiple versus single trauma ($t(108) = -1.81, p = .07$). Anger was directed more towards oneself in the multiple trauma patients and more towards others in the single trauma patients ($\chi^2 (3, N = 91) = 8.02, p = .046$). SCL-IS was significantly different for single and multiple trauma patients ($t(108) = -2.10, p = .04$), indicating more sensitivity and distrust in interpersonal relations in multiple trauma patients.

Next, we examined whether the group differences were independent of PTSD severity. To this end, all previously significant analyses were rerun, but this time with the CAPS entered as a covariate, using univariate analysis of variance (ANCOVA). Differences between single and multiple trauma patients were still significant for DES ($F(1, 109) = 5.07, p = .03$) and shame ($F(1, 109) = 4.27, p = .04$), indicating these variables were associated with single-multiple group allocation independent of PTSD severity. Single and multiple trauma groups no longer differed in guilt ($F(1, 109) = 2.66, p = .11$), STAXI-trait ($F(1, 109) = .48, p = .49$), and SCL-IS ($F(1, 109) = .98, p = .33$) after controlling for CAPS, indicating these factors were PTSD-related. The CAPS was significant in all analyses (all $p < .001$). Binary logistic regression analyses with CAPS entered as covariate showed that anger direction still tended to be associated with single or multiple (anger direction: $p = .08$, CAPS: $p = .01$).

3.2. Childhood versus adulthood trauma

Childhood ($n = 38$) and adulthood ($n = 72$) trauma groups were also first compared on gender, age, depressive symptoms and co-morbid psychopathology. There were no group differences in gender, ($\chi^2 (1, N = 110) = 1.24, p = .27$), BDI ($t(108) = -1.59, p = .12$), the number of co-morbid axis I disorders ($t(108) = .24, p = .81$) or the number of personality disorders ($t(108) = - .95, p = .35$). Patients with childhood trauma were younger than those with adulthood trauma ($t(108) = -3.31, p = .001$). Therefore, univariate analyses were done with age entered as a covariate for all childhood-adulthood comparisons.

ANCOVA analyses showed that those who experienced childhood trauma had higher DES scores than their adulthood trauma counterparts ($F(1, 109) = 5.40, p = .02$). There were no differences in guilt ($F(1, 109) = .27, p = .61$) between patients with childhood versus adulthood trauma, but childhood trauma patients tended to experience more shame ($F(1, 109) = 3.72, p = .06$).

MANCOVA analyses yielded higher STAXI-state scores for patients with childhood trauma than those with adulthood trauma ($F(1, 109) = 3.94, p = .05$) and a trend in the same direction for STAXI-trait ($F(1, 109) = 3.02, p = .09$). Anger was not differently directed for patients with childhood versus adulthood trauma ($\chi^2 (3, N = 91) = 1.83, p = .61$). SCL-IS was not different for childhood versus adulthood trauma ($F(1, 109) = 1.91, p = .17$).

The CAPS was entered as another covariate in all previously significant analyses to examine whether the observed differences would be dependent on PTSD severity. Childhood and adulthood trauma groups were also compared on gender, age, depressive symptoms and co-morbid psychopathology. There were no group differences in gender, ($\chi^2 (1, N = 110) = 1.24, p = .27$), BDI ($t(108) = -1.59, p = .12$), the number of co-morbid axis I disorders ($t(108) = .24, p = .81$) or the number of personality disorders ($t(108) = - .95, p = .35$). Patients with childhood trauma were younger than those with adulthood trauma ($t(108) = -3.31, p = .001$). Therefore, univariate analyses were done with age entered as a covariate for all childhood-adulthood comparisons.

ANCOVA analyses showed that those who experienced childhood trauma had higher DES scores than their adulthood trauma counterparts ($F(1, 109) = 5.40, p = .02$). There were no differences in guilt ($F(1, 109) = .27, p = .61$) between

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>Single trauma (n=51)</th>
<th>Multiple trauma (n=59)</th>
<th>Adulthood trauma (n=72)</th>
<th>Childhood trauma (n=38)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (in years)</strong></td>
<td>36.12 (11.92)</td>
<td>34.12 (10.90)</td>
<td>37.54 (10.82)</td>
<td>30.32 (11.02)</td>
</tr>
<tr>
<td>PTSD symptoms (CAPS)</td>
<td>61.89 (19.81)</td>
<td>71.59 (17.64)</td>
<td>63.17 (18.26)</td>
<td>74.53 (19.00)</td>
</tr>
<tr>
<td>Depressive symptoms (BDI)</td>
<td>19.43 (11.40)</td>
<td>22.77 (10.55)</td>
<td>19.72 (11.33)</td>
<td>23.64 (9.83)</td>
</tr>
<tr>
<td>Number of co-morbid DSM-IV axis I disorders</td>
<td>1.16 (1.26)</td>
<td>1.29 (1.07)</td>
<td>1.21 (1.20)</td>
<td>1.26 (1.08)</td>
</tr>
<tr>
<td>Number of DSM-IV personality disorders</td>
<td>.24 (.62)</td>
<td>.27 (.52)</td>
<td>.29 (.64)</td>
<td>.18 (.39)</td>
</tr>
</tbody>
</table>

Note. CAPS = Clinical Administered PTSD Scale, BDI = Beck Depression Inventory.

* $p < .01$.

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>Single trauma (n=51)</th>
<th>Multiple trauma (n=59)</th>
<th>Adulthood trauma (n=72)</th>
<th>Childhood trauma (n=38)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trait dissociation (DES)</strong></td>
<td>13.48 (11.36)</td>
<td>21.21 (13.38)</td>
<td>15.57 (12.85)</td>
<td>21.53 (12.58)</td>
</tr>
<tr>
<td><strong>Guilt (CAPS)</strong></td>
<td>1.78 (2.97)</td>
<td>2.33 (2.76)</td>
<td>2.33 (2.66)</td>
<td>2.58 (2.87)</td>
</tr>
<tr>
<td><strong>Shame</strong></td>
<td>.60 (.79)</td>
<td>1.10 (.98)</td>
<td>.74 (.87)</td>
<td>1.11 (.99)</td>
</tr>
<tr>
<td><strong>Anger Intensity-State (STAXI)</strong></td>
<td>16.10 (7.41)</td>
<td>16.03 (7.46)</td>
<td>15.04 (6.72)</td>
<td>18.00 (6.72)</td>
</tr>
<tr>
<td><strong>Anger Intensity-Trait (STAXI)</strong></td>
<td>18.72 (6.35)</td>
<td>21.12 (7.44)</td>
<td>19.15 (6.38)</td>
<td>21.64 (7.95)</td>
</tr>
<tr>
<td><strong>Anger direction (PSS-SR)</strong></td>
<td>46.2% (25.0%)</td>
<td>25.0% (37.3%)</td>
<td>28.8% (28.1%)</td>
<td>28.1% (28.1%)</td>
</tr>
<tr>
<td>Towards others only</td>
<td>25.6% (30.8%)</td>
<td>28.8% (28.1%)</td>
<td>34.4% (34.4%)</td>
<td>34.4% (34.4%)</td>
</tr>
<tr>
<td>Towards myself mainly</td>
<td>20.5% (30.8%)</td>
<td>22.0% (34.4%)</td>
<td>9.4% (9.4%)</td>
<td>9.4% (9.4%)</td>
</tr>
<tr>
<td>Towards myself only</td>
<td>7.7% (13.5%)</td>
<td>11.9% (22.0%)</td>
<td>20.5% (30.8%)</td>
<td>20.5% (30.8%)</td>
</tr>
<tr>
<td><strong>Interpersonal sensitivity (SCL-IP)</strong></td>
<td>39.46 (16.38)</td>
<td>45.87 (15.61)</td>
<td>40.88 (16.57)</td>
<td>46.73 (15.01)</td>
</tr>
</tbody>
</table>

Note. CAPS = Clinician Administered PTSD Scale, DES = Dissociative Experiences Scale, STAXI = State-Trait Anger, SCL-IP = Symptom Check List-90, subscale Interpersonal Sensitivity.

*** $p < .01$.

** $p < .05$.

* $p < .10$. 
adulthood trauma patients. This approach yielded similar results as the ones reported here.

In sum, multiple trauma was associated with elevated levels of dissociation, guilt, shame, interpersonal sensitivity, and self-directed anger, relative to single trauma. Childhood trauma was associated with elevated levels of dissociation and state anger, relative to adulthood trauma. As all patients were categorized on both dimensions, it is possible that either trauma type was not equally distributed in the four cells. We therefore tested whether this as a final check. A chi-square test revealed that childhood trauma patients experienced multiple trauma more often (68.4%) than adulthood trauma patients (40.3%; $\chi^2(1, N = 110) = 7.88, p = .005$).1

1 The data were also analyzed with all trauma types separated (thus: single childhood, multiple childhood, single adulthood and multiple adulthood trauma used as independent variable). This approach yielded similar results as the ones reported here.

4. Discussion

The present study was set up to investigate the effects of trauma onset and trauma frequency on several PTSD-associated symptoms. An accurate picture of symptom profiles is especially important as traditional definitions of PTSD receive growing criticism (Bodkin et al., 2007; McNally, 2007) and concepts such as "complex PTSD" emerge more frequently. Interestingly, groups (single versus multiple trauma and childhood versus adulthood trauma) did not differ in depressive symptoms, and axis I and II psychiatric comorbidity, possibly stretching the validity of PTSD as a syndrome.

Although groups did not differ in depressive symptoms and psychiatric co-morbidity, they did show differences on symptoms that are associated with PTSD but not included in the DSM-IV PTSD diagnosis (dissociative tendencies, guilt, shame, state anger, anger direction and interpersonal sensitivity). First, multiple trauma and childhood trauma patients had a higher tendency to experience dissociation. Dissociation is considered to be a developmentally sensitive and learned psychobiological defense mechanism that is used in response to overwhelming stress (Terr, 1991). Its association with complex traumas like childhood abuse (Briere and Spinazzola, 2005) may indicate that children are more prone to respond with dissociation or lack adult coping strategies to deal with overwhelming events. Remarkably, adult multiple trauma victims also showed increased dissociative tendencies in our study. Possibly, cumulative trauma adds to the negative expectations about the world and promotes avoidance-related strategies such as dissociation. Our data suggest that both young age and trauma frequency may contribute to the development of trait dissociation.

Constructs like self-blame have been associated with PTSD (Foa et al., 1999), but very few studies have investigated guilt and shame in relation to specific trauma characteristics. The present study found that feelings of guilt and shame were associated with trauma frequency, with multiple trauma patients experiencing more guilt and shame than single trauma patients. Age of trauma onset did not affect current guilt and shame feelings. This may seem contradictory with other studies reporting that childhood sexual abuse is associated with increased levels of shame (e.g., Feiring et al., 2002). However, childhood sexual abuse often includes both young age and multiple trauma, and it may be that the chronic component, and not age of onset, is responsible for these earlier findings. Another explanation could be that multiple traumas often involve a perpetrator. This may more easily lead to feelings of shame relative to traumas that do not involve interpersonal contact. That is, shame reflects a recognition of ones own inferior status within the hierarchical interpersonal dynamics with a perpetrator (Budden, 2009).

In line with this, interpersonal sensitivity was increased for multiple versus single trauma patients, but no such difference was found for childhood versus adulthood trauma. The majority of patients in this study experienced interpersonal trauma such as sexual and physical assault (74%). Such overwhelming social threat triggers feelings of humiliation and shame, which are powerful motivators of interpersonal behavior (Budden, 2009). Future research should verify this.
by comparing victims of multiple interpersonal versus multiple non-personal trauma types (like several accidents) on interpersonal coping styles such as interpersonal sensitivity. In general, victims of frequent traumatization might also have incorporated distrust as a general attitude, as they have learned that the world, including other people, is uncontrollable and unpredictable and therefore dangerous and not to be trusted (Foa et al., 1992).

In addition, two of the proposed PTSD symptoms for the DSM-V seem to address interpersonal sensitization, namely persistent negative expectations about the world and detachment from others. These are listed in the “negative alterations in cognitions” cluster, suggesting a link between negative cognitions and interpersonal functioning. Our data suggest a dose-response effect with multiple trauma victims showing more impairment in interpersonal behavior.

Anger was directed more often towards one-self after multiple trauma and more towards others after single trauma. This finding may be related to the increased shame and guilt levels in the multiple trauma group. Shame, guilt and anger towards one-self seem to indicate negative evaluations of one-self, which would be enhanced after multiple “proof” (i.e., repeated trauma). Also, multiple trauma victims may have learned that anger is safer to turn inwards as it can increase danger levels during trauma and also trigger a new event by attracting unwanted attention of the perpetrator. Again, note the new symptom cluster is proposed for the DSM-V: negative alterations in cognitions. Our findings may indicate that this cluster is especially present in multiple trauma victims. Like shame, the chronic character of childhood abuse, and not young age, may be responsible for previous associations between childhood and anger towards one-self (Andrews et al., 2000).

The only symptom that differed between the onset and not the frequency groups was state anger, with childhood trauma patients experiencing more state anger than those with adulthood trauma. This finding seems even more surprising as trait anger levels and interpersonal sensitivity did not differ between these groups. Speculatively, dysfunctional emotion regulation abilities may cause these patients to experience elevated levels of anger without being overall sensitive. However, replication is merited before a solid interpretation is possible, especially as childhood and adulthood trauma groups no longer differed on state anger after controlling for PTSD severity. These findings do not seem to be in line with Miller and Resick’s (2007) distinction between a simple and externalizing PTSD cluster with the latter being associated with increased disinhibition and anger. However, the aim of that study, and thereby the approach was different: the authors tested whether different PTSD profiles could be detected in a PTSD sample, whereas the present study was set up to examine whether a priori formulated categories (trauma onset and frequency) would differ in symptomatology. Therefore, their externalization profile probably does not overlap with the childhood-adulthood trauma distinction in our study and the results may thus not be contradictory. Finally, our data suggest that childhood trauma victims may be at risk for other reasons than their age. In our sample, they were more often exposed to multiple than to single trauma relative to adulthood trauma victims, amongst whom single and multiple trauma was equally distributed. Thus, indirectly, childhood trauma victims may be at risk because they more often have experienced multiple trauma, which was associated with additional PTSD-associated symptoms such as dissociation, guilt, shame and interpersonal sensitivity.

The fact that all differences no longer reached significance – except for dissociation and shame – may suggest that these symptoms are actually part of the PTSD syndrome. This could underscore the validity of the DSM-V PTSD criteria, in which symptoms like guilt and interpersonal sensitivity are included. The distinct PTSD symptom profiles found in the present study would then simply suggest a more severe PTSD profile. Dissociative tendencies and shame were the only independent variables in our study, suggesting that these may develop after (multiple) trauma independent of the development of PTSD symptoms. Indeed, dissociation seems a broader feature of distress that is associated with trauma but also with psychiatric illness in general, not just PTSD. Shame may be a similar independent factor, but this merits further investigation.

The present study included many sorts of trauma which optimizes generalisability. However, specific traumas types, such as victims of war, were not present in our sample and future research should therefore replicate the findings in different trauma populations. In the same line, we chose childhood-adulthood and single-multiple trauma as trauma dimensions for investigation. Other trauma characteristics may also be associated with differences in later symptom profiles, though. For example, future research should test traumas with and without a perpetrator or traumas with a known or unknown perpetrator, as these dimensions are likely to have an effect on symptoms such as interpersonal sensitivity. Also, social support was found to protect against PTSD development (Ozer et al., 2003). It would be interesting to investigate whether it also affects PTSD associated symptoms. Our study was limited by assessments of shame and guilt with a self-composed item and two CAPS items respectively. Our findings are intriguing and should be replicated with specific shame and guilt assessment instruments. Finally, the trauma type dimensions in our study overlapped. Although testing the four cells (single childhood, multiple childhood, single adulthood, and multiple adulthood trauma) resulted in similar findings, future research should further delineate trauma onset and frequency by selecting non-overlapping groups.

Practically, it has to be stated that differences in PTSD-associated symptom profiles do not necessarily indicate different treatment strategies. In our study, most symptoms were no longer significant after controlling for PTSD severity, suggesting they may be part of the PTSD syndrome or caused by PTSD symptoms. Therefore, evidence-based PTSD treatments may be effective in all groups. Indeed, prediction studies failed to consistently find an effect of trauma type or severe co-occurring symptoms such as dissociation on treatment effect (Hagenaars et al., 2010; Van Minnen et al., 2002). It is possible, though, that in complex cases the treatment would take more sessions or additional treatment strategies may be required (Levitt and Cloitre, 2005). Our data may indicate that especially dissociation and shame should be targeted, as these were associated with trauma independent of PTSD severity, and may therefore not automatically diminish after PTSD treatment. In any case, our data underscore the importance of treatment after the first trauma, as multiple traumatization may lead to a broader
spectrum of symptoms. Note however, that it may be important to evaluate ongoing traumatization, as actual threat may not allow disconfirmation of the basic negative cognitions associated with the fear memory, in which case treatment may not be effective (Foa and Kozak, 1986).

In sum, the present study found no differences in depressive symptoms or psychiatric co-morbidity between childhood versus adulthood trauma patients and single versus multiple trauma patients. However, single versus multiple groups differed in associated PTSD features (dissociation, guilt, shame, anger direction and interpersonal sensitivity), whereas childhood trauma patients had higher tendencies to dissociate and higher levels of state anger than those who had experienced adulthood trauma. However, except for multiple trauma patients experiencing more dissociation and shame, all effects disappeared after controlling for PTSD severity. These results seem to support the proposed criteria for DSM-V in which additional impairments in cognitions, affect regulation and interpersonal behavior are included in addition to the three DSM-IV PTSD symptom clusters. In any case, the findings are generally in line with animal research and suggest a cumulative effect of multiple trauma.

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Conflict of interest
None of the authors have any actual or potential conflict of interest.

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