Aus dem Institut für Psychiatrische und Psychosomatische Psychotherapie
des Zentralinstituts für Seelische Gesundheit
der Medizinischen Fakultät Mannheim
der Universität Heidelberg
(Direktor: Prof. Dr. med. Martin Bohus)

Psychopathological and Psychophysiological Sequelae of Childhood Abuse in Women without Mental Disorders

Inauguraldissertation
zur Erlangung des Doctor scientiarum humanarum (Dr. sc. hum.)
der
Medizinischen Fakultät Mannheim
der Ruprecht-Karls-Universität
zu
Heidelberg

vorgelegt von
Sophie Rausch
aus
Seeheim-Jugenheim
2018

Dekan: Prof. Dr. med. Sergij Goerdt

Referent: Prof. Dr. med. Martin Bohus

Table of Contents

Abbreviations

AAQ-II	Acceptance and Action Questionnaire-II
AM	Arithmetic mean
ANOVA	Analysis of variance
ANS	Autonomic nervous system
BDI-II	Beck Depression Inventory II
BIS	Barratt Impulsiveness Scale
BPD	Borderline personality disorder
BSI	Brief Symptom Inventory
CA	Childhood abuse
CAPS	Clinician Administered PTSD Scale
CEA	Childhood emotional abuse
CIMH	Central Institute of Mental Health
CPA	Childhood physical abuse
CSA	Childhood sexual abuse
CTQ	Childhood Trauma Questionnaire
DSM	Diagnostic and Statistical Manual of Mental Disorders
DTS	Davidson Trauma Scale
e.g.	Example gratia, for example
FDS	Fragebogen zu Dissoziativen Symptomen
GAF	Global Assessment of Functioning Scale
HC	Healthy controls
HPA	Hypothalamic-pituitary-adrenal
HR	Heart rate
HRQOL	Health-related quality of life
HRV	Heart rate variability
HTEW	Healthy trauma-exposed women
ICD	International Classification of Diseases
i.e.	Id est, that is

IPDE	International Personality Disorder Examination
MDD	Major depressive disorder
MMST	Mannheim Multicomponent Stress Test
OR	Odds Ratio
PD	Personality Disorder
PNS	Parasympathetic nervous system
PTE	Potentially traumatic event(s)
PTSD	Posttraumatic stress disorder
QoL	Quality of life
RCT	Randomized controlled trial
TRGI	Trauma related guilt inventory
RS	Resilience Scale
RSA	Respiratory sinus arrhythmia
RSP	Resources in Sexuality and Partnership
SCID	Structured Clinical Interview for DSM-IV
SD	Standard deviation
SES	Self-esteem Scale
SIA	Stress-induced analgesia
SIH	Stress-induced hyperalgesia
SNS	Sympathetic nervous system
SWLS	Satisfaction With Life Scale
TRGI	Trauma Related Guilt Inventory
TSA	Thermal Sensory Analyzer
VAS	Visual analog scale
WHO	World Health Organization

1 Theoretical Background

This dissertation aims to examine relevant psychopathological and psychophysiological sequelae of childhood abuse in women without mental disorders in order to increase our knowledge in this field by disentangling the effects of trauma and psychopathology. First, a definition of childhood abuse (CA), its components, and the respective prevalence rates will be provided. In the following, this dissertation focuses on a vast selection of relevant psychopathological and psychophysiological effects of CA, its implications on physical health, stress responsiveness and Quality of Life (QoL). Finally, resilient coping with CA will be discussed. In the last part of this chapter, research questions and aims of the present work will be derived with respect to the presented studies in chapter 2 and 3.

1.1 Prevalence of Childhood Abuse

Over the past decades, the prevalence and consequences of child maltreatment have received more and more scientific and public attention. According to the U.S. Department of Health and Human Services, child maltreatment subsumes any act of a parent or caregiver that results in harm, potential harm, or threat of harm to a child such as childhood sexual abuse (CSA), childhood physical abuse (CPA), childhood emotional/psychological abuse (CEA), and child neglect. This U.S. Department in cooperation with the National Center for Injury, Prevention and Control (2008) has furthermore defined a set of recommendations to promote consistent terminology for the different forms of child maltreatment. In this set, sexual abuse is defined as “any completed or attempted sexual act, sexual contact with, or exploitation of a child by a caregiver.” (p.14). Physical abuse is defined as “the intentional use of physical force against a child that results in, or has the potential to result in, physical injury.” (p.14). Emotional/psychological abuse is defined as “intentional caregiver behavior that conveys to a child that he/she is worthless, flawed, unloved, unwanted, endangered, or valued only in meeting another’s needs.” (p.16) and child neglect is defined as “failure of a caregiver to meet a child’s basic physical, emotional, medical, or educational needs.” Notwithstanding these recommended definitions of different types of child maltreatment, controversies exist as to the terminology of the overall term of child maltreatment varying from childhood interpersonal trauma to adverse

childhood experiences, childhood interpersonal violence, and childhood abuse. In this dissertation, the overall term childhood abuse was chosen as this term has most commonly been used in previous research (e.g., Vonderlin et al., 2018). In the studies presented within this thesis all participants were exposed to CA, as intake criterion encompassed exposure to CSA and CPA as assessed by the Childhood Trauma Questionnaire (D. P. Bernstein, Ahluvalia, Pogge, & Handelsman, 1997).

CSA and CPA are a historical constant and occur in all cultures and societies (Stoltenborgh, van IJzendoorn, Euser, & Bakermans-Kranenburg, 2011; Walker, Bonner, & Kaufman, 1988). However, controversies exist as to the overall prevalence rates. Global prevalence rates of CSA vary widely and range from as low as 0.1% (Mackenzie, Blaney, Chivers, & Vincent, 1993) to as high as 71% (Everill & Waller, 1995). Possible reasons for these discrepancies will be discussed below. More recent studies have found prevalence rates of CSA in the general adult population ranging from 4.0% to 21.4% (Coker et al., 2002; Dube et al., 2005; Häuser, Schmutzer, Brähler, & Glaesmer, 2011; Molnar, Berkman, & Buka, 2001; Romans, Gendall, Martin, & Mullen, 2001). In 2002, according to the world report on violence and health by the world health organization (WHO), 20% of women and 5-10% of men reported having been sexually abused as children (Krug, Mercy, Dahlberg, & Zwi, 2002). Conforming to this, two meta-analyses found similar prevalence rates in both women and men: According to Pereda and colleagues (2009), covering 65 studies from 22 countries, 19.7% of women and 7.9% of men reported a history of CSA. The highest prevalence rates were found in Africa and the lowest in Europe. Stoltenborgh and colleagues (2011), examining 217 publications from Australia, America, Africa and Europe, found a combined prevalence rate of studies using self-reported CSA and studies based on official records of CSA ranging between 11.3-21.5% in women and 4.1 to 19.3% in men. For women, the highest prevalence rates were found in Australia and the lowest in Asia. For the Federal Republic of Germany, the Criminological Research Institute of Lower Saxony conducted an extensive survey to examine the prevalence rate of CSA in men and women aged between 16 and 29 years. In 1996, 7.3% of men and 18.1% of women aged between 16 and 29 years reported experiences of CSA (Wetzels, 1997).

Compared to prevalence rates of CSA, much less attention has been given to assessing prevalence rates of CPA. In 1990, the Ontario Ministry of Health conducted a large survey involving 7016 participants in order to gather information on

physical health of residents in the Canadian province Ontario. The respective survey found a prevalence rate of CPA in women of 21.2% (MacMillan et al., 2001). In the U.S., a large epidemiology study by Kessler and colleagues (1995) found that 4.8% of the 3065 surveyed women reported a history of CPA. Similar numbers have been found in a more recent national U.S. survey by Finkelhor and colleagues (2015). Of the 4000 examined children and adolescents aged between 0 and 17 years, the one-year prevalence rate of CPA by a caregiver was 5%. For high-income countries, one year prevalence rates of CPA range between 4 and 16% (Gilbert et al., 2009). For the Federal Republic of Germany, an extensive study conducted by the Criminological Research Institute of Lower Saxony found prevalence rates for physical abuse in 10% of the 15 to 16 years old 9th and 10th graders (Pfeiffer, Wetzels, & Enzmann, 1999) indicating higher prevalence rates of CSA than CPA in women in Germany.

Possible reasons for this massive variation in prevalence rates of CA might be differing definitions of CSA and CPA (Wyatt & Peters, 1986). While some researchers used a less restrictive definition of CSA (e.g. kissed on the mouth), others used more restrictive inclusion criteria (e.g., touched in sensitive body parts). Discrepancies in prevalence rates might also be a result of differences in cultural beliefs and values. For example, some traumas are likely to be underreported due to the fear of embarrassment or other culturally sensitive issues. For example, in some Asian cultures protecting the family from the shame that is associated with reported abuse might be more important than protecting the abused individual (Schaeffer, 2000). Furthermore, methodological issues might contribute to the extensive variation in prevalence rates. Previous research has shown that CA prevalence rates were underestimated in studies employing official statistics or official records of CA. For example, Mac Millan and colleagues (2003) showed that only 5% of children who suffered from CPA and 8% of children who suffered from a history of CSA contacted child protection services. Statistics in these studies are likely to be underestimated due to frequent underreporting of CSA (Kogan, 2004; G. Priebe & Svedin, 2008). Another reason for the underreporting of experiences of CSA might be that people forget about the abuse. In a prospective longitudinal study by Williams and colleagues (1994), 129 women with previously documented histories of CSA were asked about abuse that took place 17 years ago. Of these women, 38% did not remember the abuse. In line with that, similar conservative lifetime prevalence

estimates of trauma exposures due to memory errors were found in a more recent study by Belli and colleagues (Belli, 2014). However, the opposite might also occur, with an over reporting of experiences of CSA. This bias towards overestimating prevalence rates has especially been observed in studies using retrospective self-report to gather prevalence rates (Andrews, Corry, Slade, Issakidis, & Swanston, 2004; Finkelhor, 2008; Ghate, Creighton, & Field, 2002; van IJzendoorn et al., 2007).

To sum up, CA is a wide-spread phenomenon affecting an extensive number of people in different cultures. Although CA is a serious problem that affects both men and women, prevalence rates for females are much higher as compared to mens' (Kessler et al., 2017; Krahé & Berger, 2013; Maikovich-Fong & Jaffee, 2010; Mills, Kisely, Alati, Strathearn, & Najman, 2016).

1.2 Sequelae of Childhood Abuse

Adjustments to traumatic and stressful events, so-called potentially traumatic events (PTE) including CA vary widely between persons. Responses and reactions are diverse, and there is no particular disorder or symptom pattern that typically follows experiences of CA (Hecht & Hansen, 2001; Kendall-Tackett, Williams, & Finkelhor, 1993). Interindividually different reactions to experiences of CA encompass a broad range of adjustments from developing mental and somatic disorders, developing psychopathological or somatic subthreshold symptomatology, to resilient coping without suffering from psychopathological or somatic impairments and possibly even positive psychological changes after facing adversities (Valle & Silovsky, 2002; Webster, 2001). Due to its widespread range of adjustment, investigating its sequelae is a major challenge in mental health research. The most valid method to assess consequences and effects of CA is by means of prospective longitudinal studies. However, those studies are rare, as they are expensive and time-consuming.

In a large prospective longitudinal study by Cutajar and colleagues (2010), the use of public mental health services of 2759 sexually abused participants was compared to that of a matched control cohort of 2677 participants without a history of CSA. CSA cases were identified using forensic medical records; the comparison group was drawn from the general population of Victoria, Australia and matched regarding age and gender. As reported by the authors, 23.3% of those affected by CSA had a lifetime record of using public mental health services as compared to 7.7% of controls. Accordingly, the rate of contacting public mental health services among the

CSA cohort was 3.65 times higher than in the control cohort. Although exposure to sexual abuse appeared to increase the use of public mental health services, the small number of 23.3% is still very surprising and might suggest that one can experience child trauma without suffering from symptoms or impairment. However, a limitation of this study lies in the fact that contacts with private mental health services or other services were not considered. Additionally, the unreported cases who did not contact mental health services are not covered in the above mentioned numbers.

Even though the Cutajar study suggests a low percentage of people contacting public mental health services after CSA, developmental consequences of CA are widespread and profound. As compared to the general population, people with experiences of CA have an elevated risk of developing psychopathology (Cutajar et al., 2010). An extensive body of literature has shown that experiences of CA can have significant ramifications on psychological and physical well-being.

1.2.1 Psychopathological impairments

Participants affected by CA more often exhibit various mental disorders and subthreshold symptomatology than participants without such experiences. According to a study by Fergusson et al (2008), people exposed to CSA had a 2.4 times heightened risk for the development of psychopathological symptoms as compared to individuals who were not exposed to CSA. For CPA, this factor was estimated around 1.5. In the above mentioned Australian prospective longitudinal study by Cutajar and colleagues (2010), forensic medical records of sexually abused children were linked with a public psychiatric database between 12 and 43 years later. In this study period, participants with official CSA records were 3.01 times more likely to meet criteria for any axis-I disorder (CSA: 18.4% versus Controls: 7%) and 5.47 times more likely to meet criteria for any axis-II personality disorder (CSA: 3.6% versus Controls: 0.7%) than controls without experiences of CSA. The most pronounced discrepancies between CSA victims and controls were found for posttraumatic stress disorder (PTSD), substance abuse and personality disorders. When examining only female CSA victims, the same pattern emerged (PTSD Odds Ratio (OR): 7.25; alcohol abuse OR: 8.96; drug abuse OR: 8.82; personality disorder OR: 7.89, especially borderline personality disorder (BPD) OR: 8.45). Overall, Cutajar and colleagues showed that participants with a history of CSA suffered three times the burden of psychopathological impairment compared with controls without

experiences of CSA. Similarly, a meta-analysis by Chen and colleagues (2010) systematically assessed evidence for an association between experiences of CA and mental disorders and found an increased risk of a lifetime diagnosis of multiple mental disorders with OR ranging from 2.34 for PTSD to 16.17 for sleeping disorders. In line with that, further studies showed an elevated risk for multiple mental disorders such as psychotic disorders, affective disorders, organic disorders, PTSD, other anxiety disorders, alcohol misuse, drug misuse, somatoform disorders, personality disorders (mainly BPD and antisocial personality disorder [PD]), and eating disorders in participants with experiences of CA, with OR ranging between 1.1 and 16.2 (Andrews et al., 2004; Chen et al., 2010; Cutajar et al., 2010; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Kessler et al., 2017; MacMillan et al., 2001; McCauley et al., 1997; Molnar, Buka, & Kessler, 2001; Rorty, Yager, & Rossotto, 1994; Wise, Zierler, Krieger, & Harlow, 2001). Among those mental disorders associated with experiences of CA, previous research focused on PTSD, BPD, and depression. Therefore, findings from these studies will be summarized in the following. Additionally, besides the elevated risk of fulfilling diagnostic criteria of mental disorders, trauma-exposed individuals may also show subthreshold psychopathological symptoms of mental disorders, or exhibit a broad range of further psychopathological symptoms. The following chapter will also introduce current research on psychopathological symptoms that are related to PTE, namely intrusions and dissociation.

1.2.1.1 Posttraumatic stress disorder

Concerning PTSD, a large U.S. National Comorbidity Survey with 5877 participants showed that 39.1% of women who experienced CSA developed PTSD, whereas only 5.7% of women without experiences of CSA developed PTSD. In Germany, epidemiologic studies have found conditional probabilities of developing PTSD after CSA in women to be ranging from 28.8% (Perkonigg, Kessler, Storz, & Wittchen, 2000) to 35.3% (Maercker, Forstmeier, Wagner, Glaesmer, & Brähler, 2008). In the U.S. National Comorbidity Survey by Kessler and colleagues (1995), 45.9% of women who reported experiences of CSA developed PTSD. In Kessler's study, the conditional probability of developing PTSD after experiencing CPA was as high as 48.5% for women. However, besides meeting full criteria for PTSD, partial PTSD is also highly prevalent after experiencing CSA or CPA (McLeer, Deblinger, Atkins, Foa, & Ralphe, 1988). In a study by Stein and colleagues (1988) examining a

community sample, 2.7% of women met full criteria of PTSD and another 3.4% met partial PTSD after experiencing rape and physical abuse among other PTE. According to a recent WHO world mental health survey (Kessler et al., 2017), experiences of interpersonal trauma, especially those early in life, carry the highest risk of a subsequent PTSD as compared to other traumatic experiences.

1.2.1.2 Borderline personality disorder

Likewise, BPD also appears to be associated with a history of CA. According to Zanarini and colleagues (2000), experiences of CA, especially CSA, have been reported in 40% to 76% of BPD patients. This percentage is significantly higher than among participant groups of other PDs. However, although experiences of CA might play an important role in the pathogenesis of BPD, they are neither specific nor sufficient for the development of BPD (Gunderson & Sabo, 1993; Paris, 1994; Scheiderer, Wood, & Trull, 2015). According to an epidemiologic study by Grant and colleagues (2008), BPD frequently coexists with comorbid PTSD, with around 38% of women meeting criteria for both disorders. As the symptom clusters of PTSD and disturbances in self-organization (as in BPD) frequently co-occur following repeated traumatic events such as CA, the WHO proposed to supplement the diagnosis of PTSD with the diagnosis of complex PTSD (cPTSD) for the upcoming ICD-11 (Chris R. Brewin et al., 2017). This complex form of PTSD, first described by Judith Herman in 1992, identifies a distinct group of patients who have experienced prolonged and repeated (interpersonal) trauma and greater functional impairment more often than those with PTSD. The ICD-11 cPTSD diagnosis is grounded on the three PTSD symptom clusters (re-experiencing, avoidance, sense of threat) plus the additional symptom clusters of affect dysregulation, negative self-concept, and difficulties in relationships. This symptom cluster was derived largely from a review of the empirical literature (Chris R. Brewin et al., 2017). Although cPTSD and BPD often share etiological risk factors (e.g. CA) as well as overlapping symptoms (e.g., problems in emotion regulation), they are also distinguishable with regard to other symptom domains (for further information see Cloitre, Garvert, Weiss, Carlson, & Bryant, 2014; Knefel, Tran, & Lueger-Schuster, 2016; Shevlin et al., 2017). Although personality disorders are often supposed to be structural and a stable diagnosis, Zanarini and colleagues (2016) conducted a longitudinal study over 16 years and showed that BPD is a rather unstable diagnosis. After two years of follow-up, 93% of participants showed a partial symptomatic remission that led them out of the diagnosis criteria for

BPD. Further dimensions of BPD such as anger, impulsivity and suicidal attempts were shown to be less stable (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008; Roberts, Walton, & Viechtbauer, 2006; Zanarini et al., 2016). Interestingly, all these behaviors are strongly associated with stress. Accordingly, one could discuss whether BPD could also be summarized within the category of stress-related disorders rather than the category of personality disorders.

1.2.1.3 Depression

Concerning depression, a number of studies have shown a relation between depression and CA (Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996; Fergusson & Lynskey, 1997; Flisher et al., 1997; Molnar, Buka, et al., 2001). In an American prospective longitudinal study, Brown and colleagues (1999) assessed the prevalence of CSA, CPA, and childhood neglect on adult depression. The authors examined 639 participants with retrospectively self-reported and/or official abuse records. Young adults with a history of childhood maltreatment were three to four times more likely to meet criteria for major depression as compared to individuals without such a history. In the respective study, the OR for developing major depression after experiencing childhood maltreatment were as follows: CSA: 3.17; CPA: 2.37; childhood neglect: 2.49. Various other studies have found a moderate to strong relationship between depressive symptoms and a history of CSA (Andrews et al., 2004; Calam, Horne, Glasgow, & Cox, 1998; Dube et al., 2005) with OR for reported CSA ranging from 2.1 to 7.0 in participants with depression (Mullen & Fergusson, 1999).

To summarize, traumatic events can result in various forms of psychopathology. Therefore, one central challenge in research with trauma-exposed individuals is to disentangle the effects of trauma experience per se versus the effects of (other) mental disorders (e.g., PTSD) in the sequel of the traumatic events. Studies I and II of this dissertation are aimed at a differentiation between effects of trauma and effects of psychopathology by including a mentally healthy traumatized control group.

1.2.1.4 Intrusions

One of the core features of PTSD encompasses spontaneous and emotionally laden intrusions. However, having intrusive memories is not unique to PTSD, but rather common in the initial months after experiencing PTE. Previous research has tried to answer the question whether intrusive memories differ between PTE-experienced

participants with or without PTSD regarding frequency or distress. For example, Kleim and colleagues (2013) were able to show that participants with PTSD reported only marginally more intrusive memories as compared to those without PTSD. However, significant differences between groups occurred when assessing the distress that was caused by the intrusions, with participants without PTSD showing less distress than PTSD-patients. These results indicate that even when PTE-experienced participants do not develop PTSD they might still suffer from intrusions. In the study by Kleim and colleagues, intrusions did not only occur in the initial months after experiencing PTE, but persisted as the assessment took place on average 63 months after the PTE.

1.2.1.5 Dissociation

Another central psychopathological symptom that is associated with experiences of PTE and accordingly regarded as a symptom of PTSD in DSM-5 (American Psychiatric Association, 2013b) is dissociation. The term dissociation was first introduced into psychiatry in the late 19th century (Janet, 1889). Dissociation describes the "disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior" (American Psychiatric Association, 2013a, p. 291). It was described as a developmentally sensitive and learned psychobiological defense mechanism that is usually used by an organism in response to overwhelming stress (Terr, 2003). The occurrence of dissociative symptoms in situations of acute stress may either serve to reduce emotional distress caused by the current situation, or may be an attempt to reduce the emotional stress elicited by triggered traumatic memories (Briere, 2002). Therefore, it is not surprising that dissociation is prevalent in patients with PTSD, both under baseline conditions (for meta-analysis see Lyssenko et al., 2017) as well as in response to experimentally induced stress (Lanius et al., 2002). Furthermore, dissociation in therapy sessions has been associated with attenuated therapy outcome in BPD and PTSD (Kleindienst et al., 2011; Kleindienst et al., 2016).

According to Steiner and colleagues (2003), the tendency to dissociate usually has its peak in childhood. Furthermore, a strong association between traumatic experiences during childhood and high levels of dissociation later on in life has been found in college students as well as in hospitalized adolescents in a study by

Sanders and colleagues (1991). In this cross-sectional study, the authors conclude that dissociation represents a reaction to CA, although this conclusion has to be interpreted with caution, since correlation does not necessarily imply causation. Importantly, it was not taken into account whether participants met diagnostic criteria of mental disorders. In an editorial on acute and chronic responses to PTE, Bremner (1999) wrote about research implications resulting from several longitudinal studies and suggested that a history of CA might serve as a risk factor for a dissociative subtype of PTSD. Supporting this assumption, dissociation seems to be very prevalent in CA-exposed participants with PTSD (for review see Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012; Wolf et al., 2012).

Taken together, previous research has shown elevated levels of dissociation in CA-exposed participants as well as those with PTSD. To disentangle the effects of a mere exposure to CA and the effects of a PTSD-diagnosis, Lanius and colleagues (2002) tested whether the level of dissociation differed between trauma-exposed individuals with and without PTSD. In a laboratory study, the authors examined female participants with CA-related PTSD, and compared this group with controls with a history of CA or motor vehicle accidents who did not meet criteria of PTSD. Both under baseline conditions as well as in response to an imagery-rescripting task PTSD-patients showed significantly higher levels of dissociation as compared to traumatized participants without PTSD. Ginzburg and colleagues (2006) examined CA-experienced treatment-seeking women regarding their levels of dissociation. The authors report that 77% of women with high dissociation levels had PTSD according to a self-report measure, whereas the other 23% of women with high dissociation levels were part of the non-PTSD group. Results of these two studies point to a rather strong relation between PTSD and elevated levels of dissociation. However, one cannot neglect that a mere history of CA, independent of PTSD, appears to be associated with dissociation, albeit to a lesser degree. However, it has to be noted that the authors used the classical trauma control design, assessing participants with and without PTSD, but not controlling for mental disorders other than PTSD, which might also influence the occurrence of dissociative symptomatology (i.e. dissociative disorder, BPD, somatic disorders, affective disorders; for meta-analysis see Lyssenko et al., 2017). Additionally, in the study by Lanius and colleagues (2002), not all participants in the trauma control group had experienced CA, as some participants were included based on a history of motor vehicle accidents. Ginzburg

and colleagues (2006) assigned women to the PTSD and non-PTSD groups according to the results of a self-report measure. Although this self-report measure is directly adapted from the DSM-IV PTSD criteria and provides good validity and reliability, it does not replace a structured clinical interview to diagnose PTSD.

1.2.1.5 Suicidality

Beside a broad range of impaired psychopathology discussed above, experiences of CA also seem to have a major impact on suicidality. In a 17-years longitudinal cohort study including 776 participants, Brown and colleagues (1999) found that experiences of CA were also accountable for a three to four times increased risk of becoming suicidal. These results suggest that people affected by CA more often try to escape from the burden associated with a history of CA or with the potential consequential psychopathology clarifying the magnitude of a possible impairment.

1.2.1.6 Impaired general functioning

Finally, psychopathological sequelae of CA are not limited to the development of mental disorders or psychopathology, but may well be reflected in impaired general functioning. The term general functioning encompasses the social, occupational/academic, and psychological functioning of an individual. More colloquially, it encompasses whether one is meeting problems in activities of daily life. In previous studies, various methods and instruments have been employed to assess general functioning. Among others, these measures include instruments to assess overall psychopathological symptomatology and levels of functioning, such as the Brief Symptom Inventory, BSI (Derogatis, 1993) or the Global Assessment of Functioning Scale, GAF (American Psychiatric Association, 2000), as well as measures of self-esteem.

In a prospective longitudinal study by Bolger and Patterson (2003), participants affected by CA were assessed over a period of five years regarding their level of functioning (e.g., self-esteem, aggressive behavior, externalizing problems). Of all these participants, only 5% were functioning well (good self-esteem, few externalizing problems and little aggressive behavior) over the study period of five years. Notably, women with a history of CA tend to show more overall psychopathological symptomatology, as reflected in elevated scores in the BSI (Braver, Bumberry, Green, & Rawson, 1992), more self-blame and guilt cognitions (Whiffen & MacIntosh, 2005), as well as significantly lower self-esteem when

compared to women without such experiences (Griffin & Amodeo, 2010; Jackson, Calhoun, Amick, Maddever, & Habif, 1990; Mullen, Martin, Anderson, Romans, & Herbison, 1996). However, with the exception of the study by Griffin and colleagues, all women in the respective studies met criteria of PTSD. Therefore, it is unclear whether these symptoms can be interpreted as an effect of the CA-exposure per se, or as an effect of the development of PTSD in the aftermaths of the CA-exposure.

Previous studies trying to disentangle the effects of mere trauma exposure versus a diagnosis of PTSD on general functioning have showed significantly lower GAF scores in participants with PTSD as compared to traumatized participants without PTSD (e.g., Agorastos et al., 2013; Wolf et al., 2012). However, these studies encompassed male combat veterans, and not participants with a history of CA.

1.2.2 Implications for subjectively perceived quality of life and satisfaction with sexuality

According to a vast body of research, experiences of CA also have an effect on the subjectively perceived Quality of Life (QoL), resulting in reduced levels of QoL (Afifi et al., 2007; Biggs, Aziz, Tomenson, & Creed, 2004; G. R. Brown, McBride, Bauer, Williford, & Team, 2005; Dickinson, 1999). The term QoL represents a broad multidimensional concept that includes subjective evaluations of all positive and negative aspects of life (The WHOQOL Group, 1998). The World Health Organization Quality of Life (WHOQOL) Group further describes QoL as “individuals’ perception of their position in life…. a broad ranging concept affected in a complex way by the person’s physical health, psychological state, level of independence, social relationships and their relationships to salient features of their environment” (World Health Organization, 1995). According to the WHO, it is important to know the subjective evaluation of all important aspects in the individual’s life that either bother or satisfy the individual, as this has significant effects on the physical and mental well-being. A more specified description of QoL is represented by the term health-related Quality of Life (HRQOL). The concept of HRQOL has evolved since the 1980s and encompasses all those aspects of QoL that can clearly be related to physical or mental health (Centers for Disease Control Prevention, 2000; McHorney, 1999). However, the terms QoL and HRQOL are often used interchangeably.

Research suggests an association of experiences of CA and affected QoL, as indicated by significantly lower levels of QoL in female participants with a history of

CA in comparison to female participants without a history of CA (Griffin & Amodeo, 2010; A. J. Lang, Stein, Kennedy, & Foy, 2004; Surís, 2007). However, participants in the respective studies encompassed female veterans, female victims of intimate partner violence, and US community women who were not assessed with regard to mental disorders, which prevents us from disentangling the effects of PTSD versus trauma exposure. First approaches in disentangling these effects have been conducted by Cloitre and colleagues (2001), who examined the relative contributions of PTSD versus trauma exposure as factors contributing to current subjectively perceived physical health-status of women with a history of CA either with or without PTSD. In this study, women with CA-related PTSD showed significantly lower physical well-being as compared to women with experiences of CA without PTSD, indicating that PTSD might be a better predictor of lower QoL levels including physical well-being than trauma exposure per se.

According to previous research, one component that might have beneficial implications for good psychological well-being, as indicated by good overall QoL ratings, is sexual well-being (for review see Laumann et al., 2006; Rosen & Bachmann, 2008). Sexual well-being, as defined by the World Health Organization (1987) can be seen as ”the recognition and acceptance of the individuality and variability of human sexual experiences and needs, but emphasized the individual’s right to be free from sexual discrimination, exploitation and abuse”. On the other hand, impaired sexual well-being is defined as a “wide-ranging set of problems associated with an impaired ability to respond sexually or to experience sexual pleasure” (DSM-5, American Psychiatric Association, 2013, p. 423). Among other aspects, these impairments include reduced satisfaction with sexuality as well as reduced sexual activity (i.e., due to unwanted or intrusive thoughts and feelings; Öberg, Fugl-Meyer, & Fugl-Meyer, 2002; Rosen & Bachmann, 2008). Prevalence estimates of sexual impairment in the female general population range from 40-45% (R. W. Lewis et al., 2010), and increase to approximately 60% in CSA-exposed women (Berthelot, Godbout, Hébert, Goulet, & Bergeron, 2014; Öberg, Fugl-Meyer, & Fugl-Meyer, 2004; Van Berlo, 2000). Whenever sexual abuse occurs in childhood or early adolescence, it generally constitutes the person’s first experiences with sexuality. Due to the involuntarily nature of the acts, these sexual experiences are often paired with conflicting or confusing messages: to cover the abuse, perpetrators

often talk their victims into believing that they enjoy what they are doing while at the same time they feel pain, fear, shame or disgust.

Consequently, it is not surprising that CSA-exposed women experience lower sexual satisfaction (Leonard & Follette, 2002; Meston, Rellini, & Heiman, 2006; Rellini & Meston, 2007; Stephenson, Pulverman, & Meston, 2014), and show a decrease in the frequency of sexual contacts and activities (Vaillancourt-Morel et al., 2015; Van Berlo, 2000), as compared to women who were not exposed to CSA. Limiting these findings, however, is the fact that in most studies regarding sexual satisfaction and frequency of sexual contacts in women with a history of CSA, mental disorders such as PTSD, the most marked mental disorder following CSA as compared to controls (Cutajar et al., 2010), were not assessed or not taken into account as a mediating variable (for meta-analysis see O'driscoll & Flanagan, 2016).

Accordingly, it is unknown whether PTSD itself entails a higher prevalence of sexual problems, or whether sexual problems can be attributed to comorbid diagnoses, as PTSD has a high prevalence of comorbidity (Bleich & Solomon, 2004). Likewise, it is unknown whether individuals with PTSD carry a higher risk of sexual problems, or whether the exposure to PTE (in this case CSA) per se may be the most important risk factor. Studies trying to disentangle this question are scarce, and were mostly conducted on veterans or other participants with experience of PTE other than CA. Results of studies on participants with war-related trauma point to the assumption that PTSD, rather than the exposure to sexual violence per se, is crucial for the impairment in sexual-well-being (Arbanas, 2010; Letourneau, Resnick, Kilpatrick, Saunders, & Best, 1996; Tran, Dunckel, & Teng, 2015). To the best of my knowledge, besides one recent study by Bornefeld and colleagues (Bornefeld-Ettmann et al., 2018) based on data of this dissertation, study I of this dissertation is one of the the first attempts to investigate the influence of sexual trauma exposure and PTSD on various aspects of QoL, including sexual functioning and sexual satisfaction in women with CA-related trauma.

1.2.3 Physical health problems

Most studies on consequences of CA are based on clinical populations with mental disorders. These circumstances could lead to an overestimation of psychological sequelae, and an underestimation of somatic consequences, such as physical health problems. However, various studies have shown that experiences of CA also predict

medical problems and poorer overall health (Dickinson, 1999; Sachs-Ericsson, Blazer, Plant, & Arnow, 2005). For example, high rates of CA have been found in patients with a variety of medical problems such as headache, fibromyalgia, chronic pain, obesity, cardiovascular disorders, respiratory diseases, metabolic disorders, immune-related disorders, chronic fatigue syndrome, gastrointestinal disorders, and cancer (Anda et al., 2006; Anda, Tietjen, Schulman, Felitti, & Croft, 2010; Drossman, Talley, Leserman, Olden, & Barreiro, 1995; Fagundes, Glaser, & Kiecolt-Glaser, 2013; Felitti et al., 1998; Fuller-Thomson, Brennenstuhl, & Frank, 2010; Golding, 1999; Gustafson & Sarwer, 2004; Shields et al., 2016; Van Houdenhove et al., 2001). Additionally, increased rates of health risk behaviors such as smoking, drinking and drug use were found in women with a history of CA as compared to non-abused women (Springs & Friedrich, 1992). These effects of CA might, among other reasons, potentially be traced back to an increased stress responsiveness, a dysfunctional regulation of the hypothalamic-pituitary-adrenal (HPA) axis (Cicchetti & Rogosch, 2001; Heim et al., 2000; Hulme, 2010; Pace & Heim, 2011) and to a dysregulation in central serotonergic responses (Rinne, Westenberg, den Boer, & van den Brink, 2000).

1.2.4 Psychophysiological impairment and altered stress responsiveness

Altered baseline stress levels as well as altered stress responsiveness can be common consequences of early stressors such as CA. Stress, as defined by Hans Selye, is a “nonspecific response of the body to any demand made upon it” (Selye, 1973). Stress can emerge in different types with regard to duration (acute stress, episodic acute stress, chronic stress) and type of stressor (e.g., physical, emotional, social). Stress response was further described as a biological or psychological response when a subject is confronted with a threat that one feels one does not have the resources to deal with (Lazarus, 1991; Rom & Reznick, 2016). Common methods to assess baseline stress levels as well as stress responsiveness involve subjective ratings as well as endocrinological (i.e., cortisol levels) or psychophysiological functioning (i.e., heart rate [HR], heart rate variability [HRV]). As the focus of this dissertation is set on subjective and psychophysiological functioning, the association of baseline stress levels and stress-responses in the metrics of subjective ratings, HR and HRV and exposure to CA and PTSD will be summarized in the following.

A vast body of research has examined baseline stress levels as well as stress-responsiveness, both on a subjective and on a psychophysiological level in individuals with PTSD related to CA or other kinds of PTE. Correspondingly, various studies have analyzed these variables in individuals exposed to CA. However, most studies did not assess or did not control for mental disorders, or CA-exposed individuals with PTSD were not compared to CA-exposed individuals without PTSD. Accordingly, it is difficult to differentiate conclusively between altering effects of CA and altering effects of PTSD on subjective and psychophysiological stress markers, both at baseline as well as in response to stress.

1.2.3.1 Subjective stress responses

On a subjective level, patients with CA-related PTSD showed elevated subjective stress ratings in confrontation with experimentally induced stress as compared to healthy controls with neither PTSD nor experiences of CA (Pierrehumbert et al., 2009). As the authors compared CA-related PTSD-patients with non-trauma-exposed healthy controls (HC), it remains unknown whether elevated subjective stress ratings in response to stress are driven by a mere exposure to CA or by meeting diagnostic criteria of PTSD. In a study by Carpenter and colleagues (2007), healthy individuals without diagnosable psychopathology with or without experiences of CA were studied in response to experimentally induced stress. Subjective stress ratings did not differ between healthy adults with and without experiences of CA. These results contest the idea of a mere effect of trauma exposure on impaired stress-response at the subjective level.

1.2.3.2 Psychophysiological HR baseline levels and responses to stress

On a psychophysiological level, altered functioning was proposedly following from early trauma. Threat and trauma, especially early in life, are thought to affect the autonomic nervous system (ANS; Buckley & Kaloupek, 2001). The ANS covers two branches that act complementarily, namely the sympathetic (SNS) and the parasympathetic nervous system (PNS). While the SNS is associated with energy mobilization, the PNS is associated with restorative and vegetative functioning. In a resting state, the SNS is dominated by the PNS, while challenges in the environment lead to an engagement of the SNS. When confronted with a regular stressor, the SNS prompts autonomic arousal responses (e.g., change in HR) that soon return to

resting equilibrium after removing the stressor. However, when confronted with a traumatic or trauma-related stressor, the SNS response is often elevated (Elzinga, Schmahl, Vermetten, van Dyck, & Bremner, 2003). Early confrontation with PTE can compromise the long-term ability to modulate SNS and PNS responses to subsequent stress, resulting in elevated baseline HR levels (for meta-analysis see Buckley & Kaloupek, 2001), as well as heightened psychophysiological stress responsiveness (for an overview see Heim, 2009; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). More specifically, this compromised functioning can lead to a pronounced imbalance of the autonomic nervous system, characterized by a hypoactive PNS and a hyperactive SNS, which has been found in women with a history of CSA (Rellini & Meston, 2006).

A broad range of studies have tried to disentangle the effects of trauma and psychopathology (e.g., PTSD) on elevated baseline HR levels as well as on impaired stress responsiveness. Based on the results of a meta-analysis by Buckley and Kaloupek (2001), basal HR seemed to be increased in participants with PTSD in comparison to controls who have or have not been exposed to a previous trauma, respectively, indicating a positive association between PTSD and elevated basal HR levels. With respect to stress response, Orr and colleagues (2003) examined monozygotic twins who were discordant with respect to trauma exposure. Half the group of combat veteran twins met diagnostic criteria of PTSD whereas the other half did not. Veterans with PTSD showed significantly higher HR responses as compared to the non-PTSD veterans as well as compared to their monozygotic twins without combat exposure in response to stressful tones. These results speak against the hypothesis that increased HR solely originates from a susceptibility of PTSD-patients to react to an unfamiliar environment (i.e., a familial vulnerability factor), and it seems to be an acquired sign of PTSD rather than the consequence of a trauma.

In a study by Adenauer and colleagues (2010), war and torture-experienced participants with PTSD reacted with an almost immediate increase in HR toward unpleasant stimuli, whereas HC participants reacted with a typical orienting response. In contrast, war and torture-experienced participants without PTSD showed an indiscriminate orienting response to all stimuli, irrespective of the valence. These results point towards a faster fight/flight response to aversive stimuli in PTSD, whereas trauma-exposed participants without PTSD seem to exhibit a permanent alertness toward a broad range of cues.

When specifically trying to disentangle the effects of trauma and psychopathology on impaired HR in female participants with a history of CA, existing research is scarce and has significant limitations. In a study by Heim and colleagues (2000), the authors found that women with experiences of CA showed increased HR in response to psychosocial stress as compared to women without experiences of CA. More specifically, when controlling for mental disorders (i.e. major depressive disorder, MDD), women with experiences of CA and MDD showed highest HR levels, followed by women with MDD and without experiences of CA as well as women with experiences of CA and no MDD. Control participants with neither a history of CA nor MDD showed the lowest HR levels. Unfortunately, the authors did not control for PTSD in these specific groups, which also affects HR levels, as described above.

1.2.3.2 Psychophysiological HRV baseline levels and responses to stress

One psychophysiological marker that is proposed to reflect the organisms' ability to flexibly adjust physiological arousal (i.e., state of the ANS) in response to environmental challenges is heart rate variability (HRV; Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012; Thayer & Brosschot, 2005). HRV measures the vagal tone and displays the degree of variability of beat-to-beat intervals across time. Flexible adaptation of the HR results in high levels of HRV, which are a sign of a healthy cardiac system and are also associated with enhanced physical and mental health (Stauss, 2003; Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011; Thayer et al., 2012). In contrast, a less flexible adaptation of HR results in low levels of HRV, which is in turn associated with a vulnerable cardiac system and psychopathology (Liddell et al., 2016). Based on the results of a previous meta-analysis including 36 studies, reduced baseline HRV seems to be very prevalent in PTSD-patients (Chalmers, Quintana, Abbott, & Kemp, 2014). In the same study, HRV was significantly lower in participants with PTSD than in controls not meeting the criteria of PTSD. This is in line with results of a systematic review by Sammito and colleagues (2015) revealing that patients with PTSD related to all kinds of PTE (i.e. firemen, veterans, people who experienced sexual abuse in adulthood) show reduced baseline HRV as compared to trauma exposed controls without PTSD. Results of both studies point to the conclusion that reduced baseline HRV seems to be an acquired sign of PTSD rather than the consequence of mere trauma-exposure. In line with that, Shah and colleagues (2013) conducted a large study on monozygotic and dizygotic twins

including 459 male twins either concordant or discordant for combat-exposure as well as either concordant or discordant for full or remitted PTSD. Summing up, results of all comparisons within that study favor the interpretation that reduced baseline HRV is a manifestation of PTSD itself rather than a reaction to trauma-exposure or a genetic predisposition.

With respect to HRV in response to distressing tasks, various studies have been conducted in order to disentangle the effect of trauma and PTSD in participants with and without PTSD after various traumatic experiences (e.g., urban violence, invasive medical procedure, witnessed domestic violence, motor vehicle accident). Results point to a significantly reduced HRV in participants with PTSD as compared to traumatized participants without PTSD (Norte et al., 2013; Scheeringa, Zeanah, Myers, & Putnam, 2004). Although there is a broad range of studies trying to disentangle the effects of PTSD and trauma-exposure in samples including individuals with experiences of all kinds of PTE, to the best of my knowledge, research on impaired baseline HRV as well as impaired stress responsiveness in CA-exposed participants, especially females, is scarce.

With regard to basal HRV levels in CSA-exposed females, one study was conducted by Rellini and colleagues (2006). The authors examined the effects of experiences of CSA on the baseline sympathetic nervous system activation while being sexually aroused. They included women with experiences of CSA not meeting diagnostic criteria of PTSD (n=8), women with CSA and a diagnosis of PTSD (n=10) as well as HC without a history of CSA (n=10). Both women with a history of CSA as well as women with a history of CSA and PTSD showed heightened SNS activation as compared to HC, indicating an effect of mere trauma exposure on altered baseline HRV levels.

With respect to HRV responses to stress, women with a history of CSA demonstrated reduced HRV as compared to women without a history of CSA while writing a distressing essay (Lorenz, Harte, & Meston, 2015). However, in the group of CSA exposed participants, the authors did not control for mental disorders. Accordingly, it remains unknown whether reduced HRV in response to this distressing task is associated with a diagnosis of PTSD, or rather the trauma-exposure per se.

In synopsis of the existing literature, baseline stress levels and stress-responses in the metrics of subjective ratings, HR and HRV seem to be impaired in participants

with a history of CA as well as in patients with PTSD. While there is beginning evidence that, especially in male veterans, impaired psychophysiological functioning in form of elevated HR levels and reduced HRV at baseline as well as reduced HRV in response to stress seem to be associated with PTSD rather than trauma exposure per se, the picture is less clear in CA-exposed individuals. To date, it is difficult to conclusively differentiate between effects of CA and effects of PTSD on subjective and psychophysiological stress markers, both at baseline as well as in response to stress, which is due to methodological issues. First, different studies encompassed different inclusion criteria such as participants having experienced traumatic events from different trauma categories, resulting in a vast heterogeneity in study samples. Second, mental disorders (i.e. depression) were either not assessed or not controlled for which might have significant ramifications on the results (for review see A. D. Brown, Barton, & Lambert, 2009). Third, the studies by Norte and colleagues and Lorenz and colleagues encompassed very small sample sizes (mean sample size=12).

1.2.3.3 Pain processing

Besides altered baseline stress levels as well as altered stress responsiveness discussed above, pain sensitivity constitutes another stress-related variable which seems to be impaired as a consequence to early stress. Pain sensitivity has been repeatedly investigated in PTSD in experimental settings, both at baseline as well as in response to stress. Experiencing pain is characterized by a vast interindividual variability and a multicausal etiology (Turk & Gatchel, 2013). According to the biopsychosocial model, the heterogeneity of pain perception is explained by biological (genetic factors), psychological (personality traits, cognitive and affective processes) and socio-cultural (cultural distinctions) variables. Furthermore, pain perception and pain sensitivity are influenced by many other factors, e.g., gender (Berkley, 1997), hormones (Craft, Mogil, & Aloisi, 2004), age (Gibson & Helme, 2001), or strong current emotions (Beecher, 1946; Meagher, Arnau, & Rhudy, 2001). However, as pointed out above, an additional factor modulating pain sensitivity is stress (Dufton et al., 2008; Girdler et al., 2005). With respect to pain sensitivity in response to stress, previous research has shown that one stressor can cause interindividual variability in pain perception (Coghill, McHaffie, & Yen, 2003), and that the impact of stress on pain sensitivity varies relative to the different types of stress and the magnitude of its appraisal. Social stress, for example, has been shown to

elicit strong stress reactions. In a study by Geva and colleagues (2014), the authors found a linear correlation between the level of perceived social stress and the degree of impairment in dealing with pain: the higher the perceived social stress level, the more impaired the ability to deal with pain. Although an extensive body of research has found stress to be a modulating factor of pain sensitivity, the different studies on traumatized populations provide inconsistent results regarding the effect of experimental stress on pain sensitivity. On the one hand, there is evidence for stress-induced analgesia (SIA) in PTSD-patients (e.g., as compared to HC), which is defined as a temporary reduction of pain thresholds that is caused by experimentally induced stress (Asmundson & Katz, 2008; Bandura, Cioffi, Taylor, & Brouillard, 1988; Defrin et al., 2008; Frid, Singer, Oei, & Rana, 1981; Kraus et al., 2009; Ludäscher et al., 2009; Strigo et al., 2010). On the other hand, there is evidence for stress-induced hyperalgesia (SIH) which describes increased pain sensitivity as indicated by increased pain thresholds as a reaction to stress (Orr et al., 2000). And yet other authors have not found alterations in PTSD-patients as compared to HC in pain sensitivity neither before nor after stress induction (Schmahl et al., 2010). Although there are contradictory findings suggesting no differences or even stress-induced hyperalgesia in PTSD-patients as compared to HC, the literature predominantly suggests stress-induced analgesia in PTSD-patients (for review see Moeller-Bertram, Keltner, & Strigo, 2012). When comparing pain sensitivity in PTSD-patients with both healthy and clinical controls, various pain inducing methods such as a thermode to examine subjective ratings of pain sensitivity (Geuze et al., 2007) or the cold pressor test to assess thresholds of pain sensitivity (Mostoufi et al., 2014) have revealed that participants with PTSD showed significantly lower subjective pain ratings as well as higher pain thresholds than the two comparison groups.

To answer the question whether experiences of PTE per se elicit alterations in baseline pain sensitivity independent of developing mental disorders, previous research provides contradictory findings. For example, Geuze and colleagues (2007) examined veterans either with or without PTSD regarding their pain sensitivity (i.e., subjective rating) under baseline conditions. In that study, veterans with PTSD showed significantly reduced pain sensitivity as compared to veterans without PTSD at baseline. In contrast, Gómez-Pérez and colleagues (2013) examined pain tolerance via cold pressor test in trauma-exposed women with PTSD, trauma-exposed women without PTSD and HC without traumatic experiences. Trauma-

exposed women with PTSD reported significantly lower pain tolerance than HC, but did not differ significantly from trauma-exposed women without PTSD, suggesting that trauma exposure per se and not the development of PTSD in the aftermath seems to be associated with altered pain tolerance. As a limiting factor, PTSD was assessed only by means of a self-report questionnaire in this study (Davidson Trauma Scale; Davidson et al., 1997). Accordingly, the authors assessed PTSD symptomatology, but did not assess whether participants met diagnostic criteria of PTSD. Furthermore, the two studies assessed participants with different trauma categories (veterans versus participants with all kinds of traumatic events), which limits the comparability of the results.

With respect to subjective pain sensitivity ratings in response to experimentally induced stress, Pitman and colleagues (1990) examined veterans with PTSD as well as veterans without PTSD or any other current axis-I disorder. After confronting the participants with stressful stimuli, the veterans with PTSD showed an average decrease of 30% in reported pain intensity ratings of standardized heat stimuli whereas the traumatized veterans without PTSD did not show a decrease in pain intensity ratings. Results of the Pitman study indicate that the PTSD diagnosis affects pain processing in response to experimentally induced stress rather than trauma-exposure per se.

When examining pain sensitivity in individuals with experiences of CA, previous research has drawn a more homogenous picture with existing evidence pointing to reduced pain sensitivity in individuals with a history of CA, both at baseline as well as in reaction to experimentally induced stress. More specifically, women with a history of CA showed lower baseline pain thresholds as compared to women without a history of CA (Alexander, 2014; Fillingim & Edwards, 2005; Scarinci, McDonald-Haile, Bradley, & Richter, 1994). This pattern holds true when examining pain sensitivity (i.e., subjective rating) in reaction to experimentally induced stress. Here, Fillingim and colleagues (2005) showed that women with a history of CA reported significantly reduced subjective pain intensity in response to stress as compared to women without a history of CA. But, as a major limitation, neither of these studies assessed or reported whether participants met diagnostic criteria for mental disorders or controlled for the assessed disorders, which prevents us from disentangling the effects of a history of CA per se against the effects of diagnosed mental disorders such as PTSD. To the best of my knowledge, no study has yet examined pain

sensitivity at baseline as well as in response to experimentally induced stress in a sample with CA-exposed participants either with or without PTSD. Accordingly, study II aims to clarify whether experiences of CA per se may produce psychobiological changes that result in decreased pain sensitivity.

To sum up, studies trying to disentangle the effects of PTSD versus trauma exposure (especially CA), on stress-related parameters such as subjective ratings, HR, HRV and pain sensitivity are inconsistent. There are possible factors explaining these inconsistencies. First, inclusion and/or comparison of different trauma categories, i.e., motor vehicle accidents (e.g., Lanius et al., 2002; Scheeringa et al., 2004) or veterans (e.g., Bremner, Staib, et al., 1999; Norte et al., 2013), which are known to be related to different probabilities in setting off PTSD and psychopathology in trauma response (for summary see Briere & Spinazzola, 2005; Rausch et al., 2016). Second, usage of different pain induction paradigms (thermode versus cold pressor test) and different dependent variables (pain threshold, subjective appraisal of pain, pain tolerance) - as pain is a complex phenomenon associating an emotional (appraisal) as well as an perceptive processing (threshold) of stimuli (Bonnot, Anderson, Cohen, Willer, & Tordjman, 2009). Third, negligence of the impact of mental disorders other than PTSD on pain sensitivity and psychophysiology (Geuze et al., 2007; Gómez-Pérez & López-Martínez, 2013; Heim et al., 2000; Lorenz et al., 2015).

While some studies on CA-exposed individuals suggest an effect of trauma exposure on altered stress responsiveness (i.e. in pain sensitivity, Gómez-Pérez & López-Martínez, 2013), other studies also point to an effect of PTSD on altered stress responsiveness (e.g. in dissociation, Lanius et al., 2002). However, the majority of studies described above included participants with a history of CA with and without PTSD, the classical trauma control design, not controlling for mental disorders other than PTSD. Therefore, results might be affected by ramifications of other mental disorders such as major depression, which is known to be related to impaired autonomic regulation characterized by increased sympathetic and/or reduced vagal modulation (for review see A. D. Brown et al., 2009). Consequently, more research is needed to clarify whether altered subjective stress ratings, HR, HRV, and pain sensitivity might be seen as diagnostic correlates of CA-related PTSD rather than being related to experiences of CA per se.

1.3 Resilient Coping

Bearing in mind all the above mentioned potential sequelae of CA, experiences of CA can be seen as a substantial risk factor for the development of subsequent impairment in mental and physical health. However, when examining only those individuals with experiences of PTE who suffer from subsequent mental disorders, one is negligent of those who seem to withstand those difficulties unscathed. Since the 1970s, research on those individuals who withstand PTE unscarred has become more and more popular. One concept frequently associated with this field of research is the concept of resilience.

The term resilience descends from the Latin word “resilire” (=rebound, recoil). It initially originated in the field of physics and materials science and was characterized as a material’s characteristic to quickly regain its original form after experiencing outer impact (Campbell, 2008). In the field of psychology, medicine and social studies, the concept of resilience was implemented after longitudinal studies showed that children can grow into mentally healthy adults despite living under very challenging and risky circumstances (Werner, 1993). Yet, there is no consensus on an accepted definition and conceptualization of the term resilience in psychology (Fletcher & Sarkar, 2013). One of the most common definitions is presented by the American Psychological Association, and defines resilience as “the process of adapting well in the face of adversity, trauma, tragedy, threats or even significant sources of threat” (American Psychiatric Association, 2014, para. 4). A more detailed definition is offered by Masten and Obradovic: “Individual resilience refers to the processes of, capacity for, or patterns of positive adaptation during or following exposure to adverse experiences that have the potential to disrupt or destroy the successful functioning or development of a person (2008, p. 2)“. Today, in the respective field of research, resilience can be seen as a maintenance of mental health in the face of adversities or chronic stress (Fletcher & Sarkar, 2013).

Factors that help promote mental healthcare are called protective factors. These protective factors of mental health have been examined on various levels, including environmental variables, neurobiology, epigenetics as well as behavioral, cognitive and emotional coping strategies (Davydov, Stewart, Ritchie, & Chaudieu, 2010). It has to be noted that these factors might interact on various levels, may change dynamically over time (Bengel & Lyssenko, 2012), and may modify, alter or

ameliorate an individual's response to PTE, for example experiences of CA (Rutter, 1985). In a systematic review of 37 primary studies with individuals with a history of CSA by Domhardt and colleagues (2014), the authors found interpersonal and emotional competence, education, optimism, attachment, external attribution of responsibility, family support and extra-family social support to be the most important protective factors for mental health. In a narrative review by Herrman and colleagues (2011) including studies on various PTEs, the personality traits extraversion, agreeableness, and openness as well as high self-esteem were reported to be of importance in withstanding adversities unscathed. In the following, several additional factors that are specifically related to resilience after CA, are introduced.

1.3.1 Self-esteem

First, self-esteem is conceptualized as an affective and evaluative component of self-perception. Various studies have shown that low levels of self-esteem are associated with experiences of CA (Chan, Brownridge, Yan, Fong, & Tiwari, 2011; Soler, Kirchner, Paretilla, & Forns, 2013; Turner, Finkelhor, & Ormrod, 2010). Additionally, low levels of self-esteem seem to be positively correlated with psychopathology in general in clinical populations (Bungert et al., 2015; Shirk, Burwell, & Harter, 2003). In reverse conclusion, according to a review by Herrman and colleagues (2011), high self-esteem may enhance resilience, which in turn might potentially attenuate the impact of CA.

1.3.2 Attribution of responsibility

Second, the attribution of responsibility (i.e. guilt cognitions) is also relevant with regard to resilience after CA. Research shows that people with a history of CA are more likely to blame themselves for negative events in general (Feiring, Taska, & Lewis, 2002). Furthermore, having guilt cognitions and self-blame concerning the PTE seems to be related to poor adjustment and pathology in participants with experiences of CA (Aakvaag et al., 2016; Ginzburg et al., 2009; McMillen & Zuravin, 1997). Conforming to this, sexually abused teenagers with PTSD, as assessed by a symptom checklist, showed significantly higher guilt regarding the abuse than sexually abused teenagers without PTSD (Wolfe, Sas, & Wekerle, 1994). Having guilt cognitions and self-blame seems to be more prominent in women than in men (Aakvaag et al., 2016).

1.3.3 Disclosing and social support

The third factor that seems to play a role in the development of psychopathology in the aftermaths of PTE is the victim's disclosure of the experience. Almost two decades ago, Pennebaker and Seagal (1999) already stressed the importance of disclosing experiences of PTE. Forming a narrative, be it in a written or spoken way, converts emotions and images into words. This process changes the way people organize and think about the potentially traumatic experiences and can be seen as a phenomenon that might lead to recovery. Previous research has assessed various barriers of disclosing experiences of CA. Among others, these barriers encompass the following: a) strong feelings of shame or guilt, b) the perpetrators' threats, c) an anxiety of frightened and irritated parents or friends by disclosing, d) the fear of not being believed or being blamed, e) the intention to protect the perpetrator, f) the lack of a trustworthy person to disclose and g) fears of social stigmatization (Münzer et al., 2016).

Finally, social reactions and support as well as social acknowledgement as a victim are related to resilience (Maercker & Mehr, 2006; Maercker & Müller, 2004; Mueller, Moergeli, & Maercker, 2008; Sippel, Pietrzak, Charney, Mayes, & Southwick, 2015) and have frequently been described as personal protective factors. Apparently, the self-perceived social support serves in a more protective way as the actual social support (Bengel & Lyssenko, 2012; Sippel et al., 2015).

1.4 Research Questions

To date, research with participants who have experienced CA and did not develop PTSD or other mental disorders is scarce. Those studies that did assess CA-exposed participants without PTSD (e.g., Dannlowski et al., 2012; Lanius et al., 2005; Wolfe et al., 1994) did not assess whether participants met other mental disorders apart from PTSD. Therefore, results might have been affected by ramifications of other axis-I disorders. Accordingly, the overall aim of this dissertation is to provide groundwork for a better understanding of the psychopathological and psychophysiological effects of CA in affected women who did not develop mental disorders. In order to increase our knowledge in this field, the two studies presented in this thesis focus on relevant psychopathological and psychophysiological aspects that are known to be impaired after experiencing CA and in patients with PTSD related to CA. Increased knowledge in this field could help disentangling the effects

of trauma experience per se and the effects of mental disorders (i.e., axis-I disorders) as a consequence of traumatic events. This, in turn, could possibly help identifying novel diagnostic markers for pathologic trauma sequelae, identify indices utilized for early detection of mental changes, or prevention of mental disorders as well as resilience factors.

In order to clarify whether healthy participants with a history of CA develop psychopathological or psychophysiological (subthreshold) symptomatology apart from meeting criteria of axis-I disorders, these studies were conducted with a traumatized but healthy participant sample. To prevent the dependent variables from ramifications by other mental disorders, this group encompassed only mentally healthy trauma-exposed women (HTEW) who did not meet criteria of any lifetime axis-I disorder or BPD, never took psychotropic medication, and never attended psychotherapy sessions. As reasoned above, prevalence rates of CA for females are much higher than men's. Therefore, only female participants were included into the following studies.

Study I examines whether healthy trauma-exposed women who did not meet criteria of any mental disorder (HTEW) differ from a) healthy controls who have never experienced PTE nor meet the diagnosis of a mental disorder and b) participants with PTSD related to experiences of CSA/CPA on a broad range of domains associated with psychopathology, and with respect to QoL. More specifically, this encompassed measures of general and PTSD-specific psychopathology, global functioning, as well as quality of life, and satisfaction with sexuality.

Study II focuses on elucidating whether elevated stress levels, as well as heightened stress responsiveness, is related to the diagnosis of CA-related PTSD, or rather to experiences of CA per se. Accordingly, self-ratings and psychophysiological assessment of stress-related variables of HTEW were compared both at baseline and in response to an established stress-induction paradigm to (a) women with PTSD related to CA and (b) mentally healthy participants without experiences of any PTE. Stress-related variables in this study included subjective stress ratings and dissociation, heart rate, heart rate variability, and pain sensitivity. Based on the existing literature, it was hypothesized that HTEW in comparison to PTSD-patients, but not HC, show lower baseline stress levels as well as lower stress reactivity with regard to subjective indicators of stress. With regard to psychophysiological

indicators of stress, it was expect that HTEW show larger stress reactivity than HC, but a lower reactivity than PTSD-patients.

2 STUDY I – Women with exposure to childhood abuse without mental disorders show no signs of impairment in general functioning, quality of life and sexuality

Published as: Rausch, S., Herzog, J., Thome, J., Ludäscher, P., Müller-Engelmann, M., Steil, R., Priebe, K., Fydrich, T., & Kleindienst, N. (2016). Women with exposure to childhood interpersonal violence without mental disorders show no signs of impairment in general functioning, quality of life and sexuality. *Borderline Personality Disorder and Emotion Dysregulation*, 3(1), 13. doi:10.1186/s40479-016-0048-y

2.1 Abstract

Childhood abuse is a major risk factor for developing posttraumatic stress disorder (PTSD), other axis-I disorders or borderline personality disorder (BPD). Individuals with a history of childhood sexual abuse (CSA) and childhood physical abuse (CPA) who meet the criteria of any axis-I disorder usually also exhibit general psychopathologic symptoms and impairments in quality of life and sexuality. The present study investigates whether women with a history of potentially traumatic CSA/CPA without any axis-I disorder or BPD show subthreshold symptoms of PTSD-specific and general psychopathology and impairments in global functioning, quality of life, and sexuality. Data were obtained from N=92 female participants: n=31 participants with a history of potentially traumatic CSA/CPA (defined as fulfilling PTSD criterion A) without any axis-I disorder or BPD; n=31 participants with PTSD related to CSA/CPA; and n=30 healthy controls without any traumatic experiences. All three groups were matched for age and education. Those with a history of CSA/CPA with and without PTSD were further matched with regard to severity of physical and sexual abuse. While women with a history of potentially traumatic CSA/CPA without axis-I disorder or BPD clearly differed from the PTSD-group in the collected measures, they did not differ from healthy controls (e.g., GAF:87, BSI:0.3, BDI-II:4.5). They showed neither PTSD-specific nor general subthreshold symptoms nor any measurable restrictions in quality of life or sexual satisfaction. Women with a history of potentially traumatic childhood abuse without axis-I disorder or BPD show a high level of functioning and a low level of pathological impairment that are comparable to the level of healthy controls. Further studies are needed to identify what helped these women survive these potentially traumatic experiences (PTE) without developing any mental disorders.

2.2 Theoretical Background

The quest for protective factors that prevent people from developing diagnosable psychopathology in the aftermath of potentially traumatic events (PTE) has recently gained much attention in research. The term “PTE” subsumes different events such as natural disasters, motor vehicle accidents, serious injury, or childhood abuse (Bonanno & Diminich, 2013). Among those affected by PTE, only a minority develops posttraumatic stress disorder (PTSD). Some develop partial PTSD (high symptom levels that do not meet full diagnostic criteria; M. B. Stein, Walker, Hazen, & Forde,

1997), and some do not develop any manifest axis-I or axis-II disorder. Previous studies have examined the epidemiology of psychopathology (e.g., PTSD) as a reaction to PTE. A large national comorbidity survey in the U.S. estimated the conditional probabilities of developing PTSD after PTE to be around 20.4% in women (lifetime prevalence rate; Molnar, Buka, et al., 2001). Maercker et al. have found conditional probabilities of developing PTSD after PTE in 12.0% and of developing partial PTSD in another 12.8% of male and female participants in a German representative epidemiologic study (Maercker et al., 2008). Estimates of PTSD have varied depending on the different types of traumatic events. In a study by Ehlers et al., 16.5% of survivors of motor accidents developed PTSD, but the vast majority did not meet the criteria, with participants showing only 3.3 PTSD symptoms on average (Ehlers, Mayou, & Bryant, 1998). When looking at incidence of PTSD-diagnoses in veterans, 12.5% of the examined Gulf War veterans developed PTSD and 25% suffered from partial PTSD, while 62.5% had no psychological impairments 1 year after returning to the United States (Sutker, Davis, Uddo, & Ditta, 1995). A similar pattern was found for Manhattan residents who survived the 9/11 terrorist attacks in New York: 7.5% developed PTSD, 17.4% developed partial PTSD, and the rest did not report a single PTSD-symptom (Galea et al., 2002). However, when experiences of childhood abuse are considered, the rates of subsequent PTSD are substantially higher, with estimates of PTSD in childhood or adolescence ranging between 20-90% (Nurcombe, 2000) and increased ORs for developing other axis-I disorders in childhood or adulthood (Affective Disorders OR: 1.75–3.57; Anxiety Disorders: OR: 1.69–3.21; Substance Abuse Disorders OR: 1.0–4.14; Cutajar et al., 2010; Fergusson, McLeod, & Horwood, 2013; MacMillan et al., 2001; Molnar, Buka, et al., 2001; Spataro, Mullen, Burgess, Wells, & Moss, 2004; Tolin & Foa, 2006). In a large U.S. study (Molnar, Buka, et al., 2001), 39.1% of women with a history of childhood sexual abuse (CSA) developed PTSD. In line with these numbers, German epidemiologic studies have found the conditional probability of developing PTSD after CSA in women ranges between 28.8 (Perkonigg et al., 2000) and 35.3% (Maercker et al., 2008).

Aside from PTSD, psychopathological impairments after experiences of childhood abuse cover a broad range of symptomatology. A few prospective longitudinal studies have examined the impact of CSA or CPA on the level of functioning or help-seeking behavior in public mental health services. With regard to the level of

functioning, Bolger and Patterson assessed 107 abused children in a prospective longitudinal study over a period of 5 years. Of those affected by childhood abuse, fewer than 5% were functioning well over the study period of 5 years (from grade 2 until grade 5; Bolger & Patterson, 2003). With regard to help-seeking behaviors, Cutajar et al. (2010) followed 2759 registered CSA cases for a period of 43 years in Australia. Of those 2759 affected by CSA, only 23.3% had a lifetime record of using public mental health services. This number is surprisingly small. However, it has to be kept in mind that this study only captured contacts with public mental health services and did not register contacts with private health services, counseling groups or support groups. The study also did not cover the estimated number of unknown cases with mental health impairments who did not contact mental health services.

Those who developed PTSD in the aftermath of CSA not only show the hallmark symptoms of PTSD such as intrusions, avoidance, numbing and hyperarousal, but usually also a range of further symptoms. Previous cross-sectional studies with participants with a history of CSA with PTSD have found significantly higher depression scores, lower self-esteem, higher general psychopathology, more dissociative symptoms, more intense guilt cognitions and more impulsiveness compared to their non-abused counterparts (Braver et al., 1992; Jackson et al., 1990; Möhler et al., 2009; Molnar, Buka, et al., 2001; Mullen et al., 1996; Neumann, Houskamp, Pollock, & Briere, 1996; Sanders, 1991; van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). Additionally, experiences of childhood physical abuse (CPA) and CSA are often associated with reduced quality of life (QoL; Afifi et al., 2007; Dickinson, 1999). Previous studies have identified further risk factors for poorer QoL such as more pronounced PTSD and depressive symptomatology (d'Ardenne, Capuzzo, Fakhoury, Jankovic-Gavrilovic, & Priebe, 2005; Rapaport, 2005).

However, psychopathological impairments and reduced QoL are not limited to those who meet full criteria of PTSD. People suffering from partial PTSD may as well have clinically significant symptoms that affect their mental and physical health and social relationships. Previous studies suggest that a diagnostic approach with a binary classification into present or absent PTSD diagnosis might not be sufficient to describe the impact of impairment in survivors of traumatic events. For example Stein et al. (1997) found that survivors of PTE (e.g., rape, physical abuse, combat, natural disaster, etc.) show serious functional impairment even when full PTSD diagnostic criteria are not met. Although survivors with full PTSD reported significantly more

impairment in work or school functioning than persons with partial PTSD, the latter still reported more impairment in work or school functioning than traumatized persons with fewer PTSD symptoms and non-traumatized persons. In terms of impaired home and social functioning, survivors with full and partial PTSD did not differ, but both groups experienced more impairment than survivors without PTSD and non-traumatized individuals. This subthreshold symptomatology is particularly common in participants with a history of CSA (McLeer et al., 1988). However, it has to be kept in mind that none of these studies controlled for co-occurring diagnoses. Therefore, the results could have been affected by ramifications of other psychopathologies such as depression or anxiety disorders. Furthermore, Stein et al.'s study provides only indirect evidence when examining the influence of childhood abuse because it included survivors of all kinds of PTE. To our knowledge, no study has assessed partial PTSD in a sample of participants who had solely been affected by CSA/CPA.

Besides general psychopathological impairments, those affected by CSA frequently reported difficulties concerning their sexual relationships in adulthood (Van Berlo, 2000). Laumann et al. (1994) examined the prevalence of adult sexual dissatisfaction and disturbances in CSA victims. Among those affected by CSA, 40% lacked interest in sex, and 32% reported that sex was not pleasurable. Several other studies examining victims of CSA replicated these findings (Jackson et al., 1990; Leonard, Iverson, & Follette, 2008; Öberg et al., 2002). However, none of these studies on sexual satisfaction assessed PTSD or other axis-I diagnoses and therefore did not distinguish between participants with and without psychopathology.

Besides studies on psychopathological impairments in participants with a history of CSA/CPA with full and partial PTSD, few studies have examined participants with a history of CSA/CPA without PTSD. Kleim et al. (2013) showed that trauma survivors without PTSD experience only marginally less intrusions than those with PTSD, but they experience them less vividly. Concerning dissociation, Lanius et al. (2005) showed that trauma survivors without PTSD experience significantly less dissociative symptoms as compared to trauma survivors with PTSD under experimentally induced distress in a laboratory setting. But these studies by Lanius and Kleim examined trauma survivors of all kinds of traumatic events (e.g., combat, assault, motor vehicle accidents, or CSA) and not only those affected by childhood abuse. Therefore, these results provide only indirect evidence of the influence of childhood abuse on dissociation and intrusive re-experiencing. In addition, all these studies on trauma

survivors without PTSD included survivors who possibly met other axis-I or axis-II diagnoses. Thus, effects of pathologies other than PTSD were not considered.

In sum, previous research has shown that participants with a history of childhood abuse with full and partial PTSD show significant impairments in general functioning, quality of life, and sexuality. Research on trauma survivors without PTSD is scant and has significant limitations. Limitations emerge from the fact that different groups are only distinguished by means of the dichotomy into presence or absence of PTSD-diagnosis, but axis-I or axis-II disorders other than PTSD are not considered. Additionally, previous research has not attempted to minimize the impact of differences in severity of experienced CSA or CPA in those with and without PTSD when examining psychopathologic responses to traumatic events, which poses a threat to the internal validity of the results. To our knowledge, all these studies included participants without PTSD who potentially met other axis-I diagnoses. Hence, it remains unclear whether participants with no axis-I or axis-II disorders plus no experiences of psychotherapeutic interventions or intake of psychotropic medication with a history of potentially traumatic CSA/CPA suffer from subthreshold symptoms and exhibit at least unspecific restrictions in psychological well-being.

The present study examines whether healthy participants with a history of potentially traumatic CSA or CPA without any axis-I disorder or borderline personality disorder (BPD; healthy trauma-exposed women; HTEW) differ from 1) healthy controls (HC) who have never experienced a trauma nor meet the diagnosis of a mental disorder and 2) participants with PTSD related to experiences of CSA/CPA (PTSD-patients), in the following domains: dissociation, depression, global functioning, impulsivity, self-esteem, PTSD-specific psychopathology, satisfaction with mental and physical aspects of quality of life and sexuality. Furthermore, we were interested in current resilience scores of those three groups, as measured with a widely used resilience scale (RS; Wagnild & Young, 1993). To avoid the potentially confounding influences of severity effects of CSA or CPA in individuals in the HTEW and PTSD groups, we controlled for severity by matching the individuals of the HTEW and the PTSD-patient group with regard to their Childhood Trauma Questionnaire (CTQ; D. P. Bernstein & Fink, 1998) scores for the subscales of “Physical Abuse” and “Sexual Abuse”.

2.3 Methods

2.3.1 Sample

A total of 92 women participated in this study: 31 women with a history of potentially traumatic CSA/CPA and no axis-I diagnosis or BPD (HTEW), 31 patients meeting criteria for PTSD related to experiences of CSA/CPA, and 30 healthy controls without experiences of childhood abuse or other traumatic events (HC; see Fig. 1).

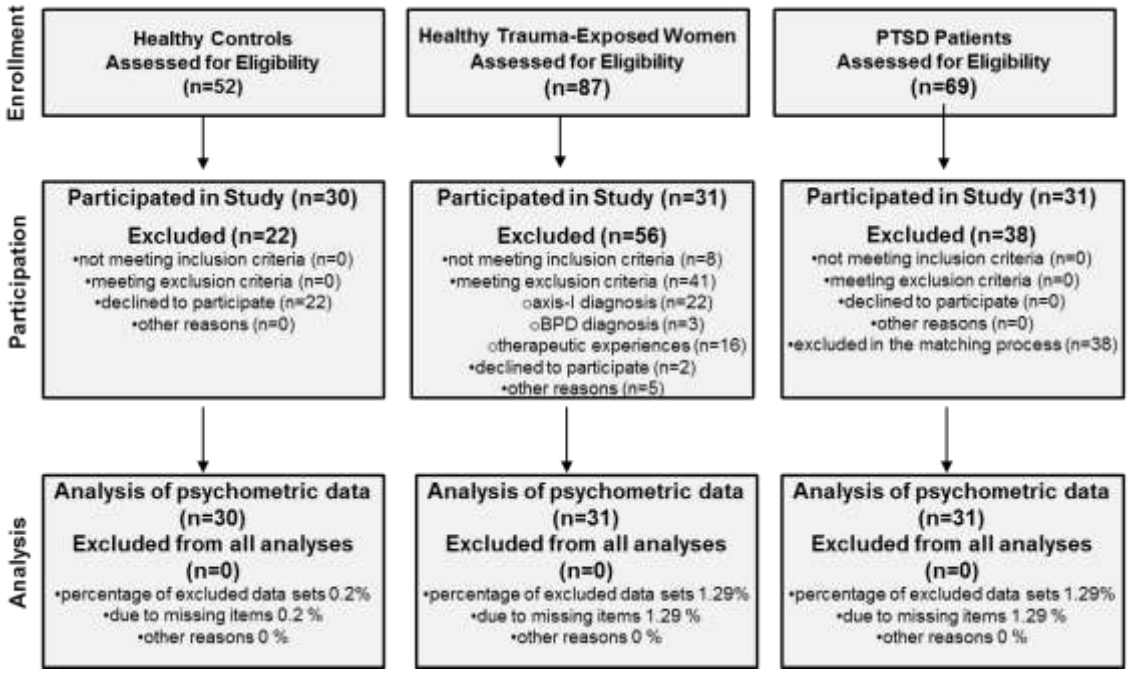

Figure 1. Participant flowchart

Enrollment was restricted to women aged between 18 and 65. For the individuals of the HTEW group, inclusion criterion was the experience of sexual or physical abuse before the age of 18. Exclusion criteria were meeting criteria for a lifetime diagnosis of any axis-I disorder or of BPD, intake of psychotropic drugs or having received a psychotherapeutic intervention in the form of seeing a therapist or counsellor. The same exclusion criteria applied for the HC group plus experiences of abuse in childhood or adolescence or any other traumatic events that met the PTSD criterion A. For the patient sample, inclusion criterion was a current diagnosis of PTSD according to DSM-5 that was related to experiences of sexual or physical abuse before the age of 18. Experiences of sexual or physical abuse had to be the index trauma, meaning the most burdensome event, leading to PTSD. Because the PTSD-patients who participated in our study also took part in a study comparing two different psychological treatments for PTSD and co-occurring BPD-features, they

additionally had to fulfill at least three criteria of BPD (including criterion 6: affective instability) as defined by the International Personality Disorder Examination (IPDE; Loranger, Janca, & Sartorius, 1997). Seventeen out of 31 participants in our PTSD sample met full criteria for BPD. The additional inclusion criterion for the PTSD-patient group, i.e. at least 3 out of 9 criteria of BPD was chosen as we wanted to include highly impaired patients with complex PTSD. Exclusion criteria for this group were a lifetime diagnosis of schizophrenia or bipolar-I disorder, current substance dependence, a body mass index <16 or intellectual disability as objectified by a verbal intelligence test (Lehrl, Merz, Burkhard, & Fischer, 1991). For safety reasons, individuals who had attempted suicide in the last 2 months were also excluded. The diagnostic interviews to assess inclusion and exclusion criteria were conducted by trained clinical psychologists using the BPD section of the IPDE, the Structured Clinical Interview for DSM-IV (SCID-I; Wittchen, 1997) and the Clinician Administered PTSD Scale for DSM-5 (CAPS; Weathers et al., 2013).

Individuals of the HTEW group were recruited through newspaper advertisements and flyers distributed at public places (e.g., cafés, supermarkets). The HC sample was recruited via the database at the Department for Psychosomatic Medicine and Psychotherapy, CIMH Mannheim, which contains contact information of pre-screened healthy controls. Data of PTSD-patients were obtained from an ongoing multi-center study at the Department for Psychosomatic Medicine and Psychotherapy, CIMH Mannheim, of the Department of Clinical Psychology and Intervention, Institute of Psychology, Goethe University Frankfurt, and the Department of Psychology, Faculty of Life Sciences, Humboldt-Universitaet zu Berlin.

Because the central inclusion criterion for individuals of the HTEW and PTSD groups was the experience of CSA or CPA, 31 PTSD-patients with a comparable level of physical and sexual abuse as the individuals in the HTEW group were taken out of the PTSD-patients pool of the multi-center study. This was achieved by matching the scores of the two subscales of Sexual Abuse and Physical Abuse of the Childhood Trauma Questionnaire (see Table 2; CTQ; D. P. Bernstein & Fink, 1998) as implemented in a study by Roy et al. (2011). Furthermore, all three groups were matched for age (HC: 31.4 ± 8.4; HTEW: 31.8 ± 12.6; PTSD-patients: 32.9 ± 8.7; F[2,89]=.189, p=.828) and years of education (HC: 11.3 ± 1.0; HTEW: 11.4 ± 0.9; PTSD-patients: 10.9 ± 1.2; H[2]=3.74, p=.155). Approval was obtained from the

independent Ethics Committee of the Medical Faculty Mannheim at Heidelberg University. All participants provided written informed consent.

2.3.2 Procedure and measures

Severity of experiences of childhood abuse was assessed by the total score and subscale scores of the CTQ (D. P. Bernstein & Fink, 1998). The CTQ is composed of 28 items subdivided into five subscales of adverse events (Physical Abuse, Physical Neglect, Sexual Abuse, Emotional Abuse, and Emotional Neglect) and a minimization/denial subscale. Participants rate the frequency of maltreatment for each item on a five-point Likert scale. The CTQ total score (all subscales except the minimization/denial scale) ranges from 25 to 125, and the five subscale scores of adverse events range from 5 to 25. In this study, individuals of the HTEW group and PTSD-patients were matched with respect to the two subscales of Physical Abuse and Sexual Abuse. The matching procedure was restricted to these two subscale scores, because experiences of physical or sexual abuse were the central inclusion criterion of the study.

The assessment of PTSD-specific psychopathology is comprised of the Davidson Trauma Scale (DTS; Davidson et al., 1997), the Trauma Related Guilt Inventory (TRGI; E. S. Kubany et al., 1996), and the German adaption (Fragebogen zu Dissoziativen Symptomen; FDS; Freyberger et al., 1998) of the Dissociative Experiences Scale (E. M. Bernstein & Putnam, 1986).

The ***DTS*** is a 17-item self-report questionnaire measuring each DSM-IV symptom of PTSD on five-point frequency and severity scales for a total possible score between 0 and 136. Subscale scores for intrusive re-experiencing, avoidance and numbness and hyperarousal can be computed separately for frequency and severity (see Fig. 2). The ***TRGI*** is a valid 32-item measure of trauma-related guilt. Every item is rated on a five-point Likert scale. It implies the three scales of global guilt (4 items with a total score ranging from 0 to 4), distress (6 items with a total score ranging from 0 to 4), and guilt cognitions (21 items with a total score ranging from 0 to 4). The ***FDS*** is an easy applicable, reliable, and valid measure to quantify dissociative experiences that is based on the Dissociative Experience Scale. It is comprised of 44 items rated on a 10-point scale with a total score ranging from 0 to 100.

The assessment of general psychopathology and psychological flexibility is comprised of the Beck Depression Inventory-II (BDI-II; Hautzinger, Keller, & Kühner,

2006), the Brief Symptom Inventory (BSI; Derogatis, 1993), the Global Assessment of Functioning (GAF; American Psychiatric Association, 2000), the Barratt Impulsiveness Scale Version 10 (BIS; Barratt, 1985), the Acceptance and Action Questionnaire-II (AAQ-II; Bond et al., 2011), and the Rosenberg Self-Esteem Scale (SES; Rosenberg, 1965).

The ***BDI-II*** is the most widely used self-report questionnaire worldwide to evaluate severity of depressive symptoms. It contains 21 items rated on a four-point Likert scale with a total score ranging from 0 to 63. The ***BSI*** is a 53-item self-report symptom inventory designed to assess psychological distress and mental disorders. All items are rated on a five-point Likert scale from 0 to 4 with a total score ranging from 0 to 4. The ***GAF*** is a single rating scale for evaluating a person's psychological, social, and occupational functioning on a hypothetical continuum of global level of functioning and ranges from 1 to 100. The ***BIS*** is a 34-item self-report questionnaire to measure impulsiveness. All items are answered on a four-point Likert scale from 1 to 4. The ***AAQ-II*** assesses experiential avoidance and psychological flexibility. Psychological flexibility is defined as the ability to fully contact the present moment with all its inherent thoughts and feelings. It implies to either persist in or change the behavior that is necessary in the pursuit of goals and values [52]. It is comprised of 7 items that are answered on a seven-point Likert scale ranging from 7 to 49, with higher summed scores indicating higher psychological inflexibility. The ***SES*** is a self-rating instrument that assesses global self-esteem. It contains ten items that are rated on a four-point Likert scale ranging from 0 to 30. Scores ranging between 15 and 25 indicate normal self-esteem, while scores below 15 indicate low self-esteem.

Quality of Life and sexual satisfaction were assessed using the Satisfaction with Life Scale (SWLS; E. Diener, Emmons, Larsen, & Griffin, 1985), the EQ-5D (The EuroQol Group, 1990), the WHOQOL-BREF (Angermeyer, Kilian, & Matschinger, 2000) and the Resources in Sexuality and Partnership (RSP; Klingler & Loewit, 1996).

The ***SWLS*** is a five-item scale that assesses general life satisfaction. Each item is rated on a seven-point scale with a total score ranging from 5 to 35. The higher the total score, the higher the level of experienced global life satisfaction is. The ***EQ-5D*** is a short questionnaire for measuring health-related quality of life. It consists of 5 items that are rated on a three-point Likert scale with a total score ranging from 5 to 15. The total score is then transformed into a scale ranging from 0 to 100. The

WHOQOL-BREF is the most frequently used questionnaire to assess QoL and consists of 26 five-point Likert scale (1–5) items. It includes four main domains of QoL (physical health, psychological health, social relationships and environment) and a facet of overall QoL and general health. All five scores are transformed into a range from 0 to 100 to ensure comparability. The ***RSP*** encompasses five domains assessing the subjective experience of joy with respect to physical attractiveness, tenderness, sexual lust and satisfaction, love and communication of sexual needs and desires. We used a modified version of the 25-item RSP and included a sixth response option, "did not happen," because many of our patients do not have sexual intercourse. On the one hand, we assessed a total RSP score of all items that were answered from 1 (very rarely enjoyable) to 5 (very often enjoyable) to assess joy and satisfaction with sexuality. On the other hand, we assessed the percentage of items that were answered with "did not happen" to assess the incidence of sexual behaviors. The questionnaire can be applied independently of sexual orientation and kind of interpersonal relationship.

Resilience was assessed using the ***Resilience Scale*** (RS; Schuhmacher, Leppert, Gunzelmann, Strauß, & Brähler, 2005), which measures resilience "as the ability … to use internal and external resources successfully to cope with developmental tasks" (Derogatis, 1993, p. 21) on a 25-item scale. It is comprised of a total score and two subscale scores. The subscale Personal Competence subsumes characteristics such as self-confidence, autonomy, mastery, mobility and endurance. The subscale Acceptance of Self and Life subsumes characteristics such as adaptability, tolerance, flexible sight towards oneself and towards one's journey through life. Items are rated on a seven-point Likert scale and summarized for each subscale.

2.3.3 Statistical analyses

All analyses were performed using SPSS (version 21;SPSS Inc.;USA). To test for differences between the HTEW group, PTSD-patients, and the HC group on the questionnaires and for age, one-way analyses of variance (ANOVAs) were applied. Post-hoc analyses were performed by pairwise comparisons (Bonferroni corrected for multiple testing). Non-parametric Kruskal-Wallis tests were used for overall comparisons in years of education, employment status, and number of children across the three groups. Pairwise post-hoc comparisons were conducted with Mann-Whitney U tests. To test for differences in marital status, chi-square tests were applied. Effect sizes were calculated as Cohen's d. A significance level of $p \leq .05$

(two-tailed) was applied for all analyses. Data were reported as arithmetic mean (AM) ± standard deviation (SD).

2.4 Results

Demographic characteristics of the three groups (HC, HTEW, PTSD-patients) are presented in Table 1. The three groups did not differ significantly in terms of age, education, marital status, and number of children, but did with regards to employment status. Herein, the HTEW and HC group did not differ significantly with 87.1% of HTEW and 76.7% of HC working full or part time. However, both groups differed significantly from PTSD-patients of whom only 42% were working full or part time.

Table 1 Sample characteristics of healthy controls, healthy trauma-exposed women and PTSD-patients

	HC	***HTEW***	***PTSD***	***Statistics***
	AM ± SD	AM ± SD	AM ± SD	
Age	31.37 ± 8.4	31.81 ± 12.6	32.9 ± 8.6	$F(2,89)=.189$, $p=.828$
Years of education	11.3 ± 1.0	11.4 ± 0.9	10.9 ± 1.2	$H(2)=3.735$, $p=.155$
Marital status	N (%)	N (%)	N (%)	$\chi^2(2)=4.645$, $p=.336$
Married/cohabiting	24 (80.0)	20 (64.5)	19 (61.3)	
Single	6 (20.0)	8 (25.8)	11 (35.5)	
Divorced/widowed	0 (0)	3 (9.7)	1 (3.2)	
Number of children	N (%)	N (%)	N (%)	$H(2)=.774$, $p=.679$
No children	22 (73.4)	21 (67.7)	19 (61.3)	$U_{HC\text{-}HTEW}=427.0$, $p=.489$, r=-.087
1	3 (10.0)	2 (6.5)	6 (19.4)	$U_{HTEW\text{-}PTSD}=475.5$, $p=.941$, r=-.011
2	3 (10.0)	2 (6.5)	4 (12.9)	$U_{HTEW\text{-}PTSD}=416.0$, $p=.402$, r=-.109
3	1 (3.3)	5 (16.1)	1 (3.2)	
4	1 (3.3)	1 (3.2)	1 (3.2)	

	HC	*HTEW*	*PTSD*	*Statistics*
	AM ± SD	AM ± SD	AM ± SD	
Employment status	N (%)	N (%)	N (%)	H (2)=14.451, p=.001
Full time	16 (53.4)	16 (51.6)	7 (22.6)	$U_{HC\text{-}HTEW}$=446.0, p=.769, r=-.039
Part time	7 (23.3)	11 (35.5)	6 (19.4)	$U_{HTEW\text{-}PTSD}$=246.5, p< *.001,* r=.439
Occasionally	4 (13.3)	3 (9.7)	3 (9.7)	$U_{HC\text{-}PTSD}$=266.0, p<*.001.*, r=-.387
Not working	3 (10.0)	1 (3.2)	15 (48.4)	
Current comorbidity	N (%)	N (%)	N (%)	
Any anxiety disorder	0 (0.0)	0 (0.0)	22 (71.0)	
Any mood disorder	0 (0.0)	0 (0.0)	21 (67.7)	
Any eating disorder	0 (0.0)	0 (0.0)	4 (12.9)	
Any substance abuse/dependence	0 (0.0)	0 (0.0)	1 (3.2)	
BPD	0 (0.0)	0 (0.0)	17 (54.8)	

Note. F and p represent the F- and p- values of the respective one-way analysis of variance (ANOVA); H and U represent the respective H and U values of the Kruskal-Wallis and Mann-Whitney-U tests; χ^2 represents the respective value of the Chi-square analysis; AM=arithmetic mean; SD=standard deviation. For Axis-II Disorders, only borderline personality disorder was assessed.

2.4.1 Experiences of childhood abuse

With regards to the CTQ total score, one-way ANOVA yielded significant differences between the three groups ($F(2,87) = 68.798$, p<.001). Post-hoc pairwise comparisons showed that PTSD-patients reported a significantly higher frequency of adverse events compared to individuals of the HTEW group (p<.001), who in turn reported a higher frequency of adverse events than HC (p<.001). As Table 2 shows, the frequency of adverse events in childhood varied between groups in the different subscales: PTSD-patients and participants in the HTEW group did not differ significantly concerning sexual (p=1.0) and physical abuse (p=.94) as a consequence of our matching procedure. However, both groups differed significantly from individuals in the HC group in these subscales (p<.001). With regard to the subscale of physical neglect, PTSD-patients experienced a significantly higher frequency of physical neglect compared to participants in the HTEW and HC groups (p<.001), whereas participants in HTEW and HC groups did not differ significantly (p=.227).

Concerning emotional abuse and emotional neglect, PTSD-patients experienced a significantly higher frequency of adverse events than individuals in the HTEW group, who in turn reported a significantly higher frequency than individuals in the HC group (p<.001).

Table 2 Childhood Trauma Questionnaire subscale and total scores of healthy controls, healthy trauma-exposed women and PTSD-patients

	HC	***HTEW***	***PTSD***	***Statistics***					
	AM ± SD	AM ± SD	AM ± SD	F	df	*p*	$d_{HTEW-HC}$	$d_{HTEW-PTSD}$	$d_{PTSD-HC}$
CTQ_Sexual Abuse	5.1 ± 0.3	10.9 ± 5.8	11.6 ± 6.0	*16.579*	*(2,87)*	<.001	*1.87*	*-.12*	*2.06*
CTQ_Physical Abuse	5.9 ± 2.4	9.5 ± 3.9	10.2 ± 4.6	*11.06*	*(2,87)*	<.001	*1.14*	*-.16*	*1.23*
CTQ_Emotional Abuse	7.1 ± 2.8	12.7 ± 5.1	18.2 ± 5.5	*43.489*	*(2,87)*	<.001	*1.42*	*-1.04*	*2.67*
CTQ_Emotional Neglect	7.8 ± 3.4	12.5 ± 4.5	18.8 ± 4.4	53.621	*(2,87)*	<.001	*1.19*	*-1.42*	*2.82*
CTQ_Physical Neglect	6.0 ± 1.9	7.2 ± 2.5	8.1 ± 3.4	30.055	*(2,87)*	<.001	*0.55*	*-0.31*	*0.79*
CTQ_Total Score	31.8 ± 7.8	52.9 ± 13.4	69.9 ± 15.3	68.798	(2,87)	<.001	1.99	-1.18	3.3

Note. F and p represent the F- and p- values of the respective one-way analysis of variance (ANOVA); d represents the pairwise compared effect sizes after Cohen; df=degrees of freedom; AM=arithmetic mean; SD=standard deviation; CTQ=Childhood Trauma Questionnaire.

2.4.2 PTSD-specific and general psychopathology

Not surprisingly, PTSD-patients reported significantly more PTSD-specific symptoms than individuals in the HTEW group in general. Examining PTSD-specific symptoms in the scope of the DTS, Fig. 2 illustrates frequency and severity mean scores for the three symptom clusters of the diagnostic criteria (intrusive experiences, avoidance of trauma related triggers, hyperarousal) separately. For the frequency subscales, PTSD-patients reported a significantly higher frequency of experienced symptoms such as intrusions, avoidance of trauma related triggers, and hyperarousal compared

to HTEW participants. The same pattern was observed for severity scores on all subscales, with PTSD-patients reporting symptoms as significantly more severe than the HTEW participants (see Fig. 2).

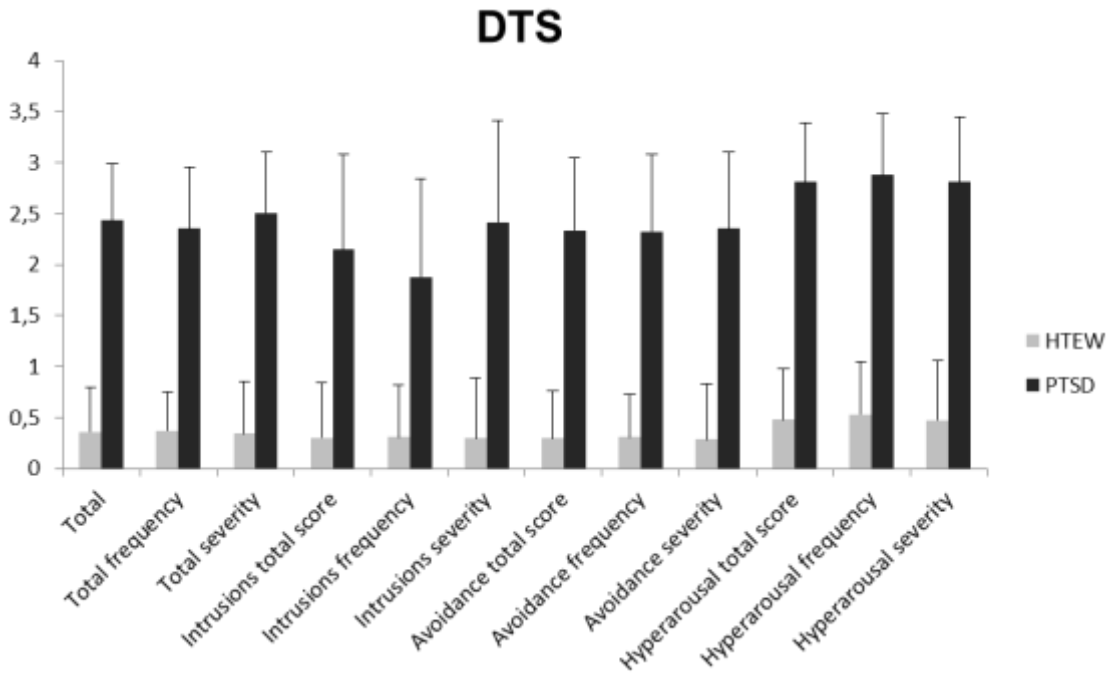

Figure 2. DTS total and subscale mean scores for individuals in the HTEW group and PTSD-patients. Error bars are depicted as standard deviations (SD)

With regard to general psychopathology, comparisons of the three groups revealed a similar pattern in all questionnaires. PTSD-patients showed significantly higher levels of psychopathology compared to the HTEW and HC groups, respectively, in terms of depression (BDI-II), psychological distress and mental disorders (BSI), global functioning (GAF), impulsivity (BIS), psychological flexibility (AAQ-II), self-esteem (SES), trauma-related guilt cognitions (TRGI), and dissociative symptoms (FDS). For detailed information, see Table 3. In all those measures, individuals in the HTEW group showed a very high level of functioning (e.g., GAF:87) and a very low level of psychopathological impairment (e.g., BSI:0.3; BDI-II:4.5), which were comparable to levels of individuals in the HC group. Effect sizes suggest that differences between HTEW participants and PTSD-patients were highest in the GAF and the AAQ-II (GAF: $d = 5.95$; AAQ-II: $d = -4.72$). The same pattern occurred between PTSD-patients and the HC group (GAF: $d = -5.77$; AAQ-II: $d = 5.49$). The highest differences between participants in the HTEW and HC groups were found in the FDS

(d = 0.6). The effect sizes for all psychopathological measures between HTEW participants and PTSD-patients ranged between −1.58 and 5.05, and for all QoL measures, between 1.57 and 3.94. Comparing individuals in the HTEW and HC groups with regard to psychopathological measures, effect sizes ranged between −0.02 and 0.6, and for QoL, between −0.01 and −0.34.

Table 3 General and PTSD-specific psychopathology of healthy controls, healthy trauma-exposed women and PTSD-patients

	HC	***HTEW***	***PTSD***	***Statistics***					
	AM ± SD	AM ±SD	AM ± SD	F	df	p	$d_{HTEW-HC}$	$d_{HTEW-PTSD}$	$d_{PTSD-HC}$
BDI-II	4.6 ±4.7	4.5 ±6.0	38.6 ± 10.1	222.268	(2,89)	<.001	-.02	-4.24	4,57
BSI	0.2 ±0.2	0.3 ±0.3	2.0 ±0.6	206.837	(2,88)	<.001	0.4	-3.78	4.46
GAF	91.1 ± 8.3	86.6 ± 8.6	48.5 ±6.5	273.92	(2,89)	<.001	-0.53	5.05	-5.77
BIS	60.3 ± 9.2	64.3 ± 9.3	83.0 ± 14.3	35.91	(2,89)	<.001	0.43	-1.58	1.92
AAQ-II	12.2 ± 5.7	13.9 ± 6.6	39.6 ±4.3	231.167	(2,89)	<.001	0.28	-4.72	5.49
SES	27.0 ± 4.1	26.9 ± 4.0	9.3 ±4.8	174.51	(2,88)	<.001	-0.02	3.99	-3.97
DTS	-	12.2 ± 14.8	82.6 ± 19.1	264.81	(1,60)	<.001	-	-4.15	-
TRGI-global guilt	-	0.6 ±0.8	2.7 ±1.2	61.570	(1,55)	<.001	-	-2.12	-
TRGI-distress	-	1.0 ±0.8	3.5 ±.4	198.623	(1,55)	<.001	-	-4.09	-
TRGI-guilt cognitions	-	1.0 ±0.5	2.1 ±0.6	46.202	(1,55)	<.001	-	-2.01	-
FDS	2.8 ±2.4	5.0 ±5.0	24.4 ± 12.2	69.402	(2,86)	<.001	0.6	-2.21	2.93

Note. F and p represent the F- and p- values of the respective one-way analysis of variance (ANOVA); d represents the pairwise compared effect sizes after Cohen; df=degrees of freedom; AM=arithmetic mean; SD=standard deviation; BDI-II=Beck Depression Inventory-II; BSI=Brief Symptom Inventory; GAF=Global Assessment of Functioning; BIS=Barratt Impulsiveness Scale; AAQ=Acceptance and Action Questionnaire; SES=Rosenberg Self-Esteem Scale; DTS=Davidson Trauma Scale; TRGI=Trauma Related Guilt Inventory; FDS=Fragebogen zu Dissoziativen Symptomen; AM=arithmetic mean; SD=standard deviation. Dashes indicate that data were not obtained.

2.4.3 Quality of Life and sexual satisfaction

Analysis of all three QoL measures (WHOQOL-BREF, SWLS, EQ-5D) showed that the groups differed significantly in subjectively experienced QoL ($F_{SWLS}(2,89)=101.206$, $p<.001$; $F_{EQ\text{-}5D}(2,88)=93.284$, $p<.001$; $F_{WHOQOL\text{-}BREF\ global}(2,88)=69.355$, $p<.001$). Post-hoc pairwise comparisons of all three measures indicated that both HTEW and HC participants reported higher QoL levels compared to PTSD-patients ($p<.001$), whereas HTEW participants showed QoL levels that were comparable to HC participants ($p=1.0$; $d_{SWLS}=0.23$; $d_{EQ5D}=-0.27$; $d_{WHOQOL\text{-}BREF}=-0.34$). This pattern also emerged in the WHOQOL-BREF subscales of satisfaction with physical health, psychological health, social relationships, and the environment (see Table 4 and Fig. 3).

Table 4 Quality of life, sexual satisfaction and resilience of healthy controls, healthy trauma-exposed women and PTSD-patients

	HC	***HTEW***	***PTSD***	***Statistics***					
	AM ± SD	AM ± SD	AM ± SD	F	df	p	$d_{HTEW\text{-}HC}$	$d_{HTEW\text{-}PTSD}$	$d_{PTSD\text{-}HC}$
SWLS	28.3 ± 4.7	27.1 ± 5.7	10.8 ± 5.6	101.206	(2,89)	<.001	0.23	2.88	-3.39
EQ-5D	97 ± 5.3	95.5 ± 5.7	66.3 ± 15.2	93.284	(2,88)	<.001	-0.27	2.82	-3.0
WHOQOL-global	81.5 ± 12.8	76.4 ± 17.4	33.9 ± 20.7	69.355	(2,88)	<.001	-0.34	2.23	-3.58
WHOQOL-physical health	86.2 ± 8.1	85.3 ± 8.3	43.3 ± 16.3	136.433	(2,88)	<.001	-0.11	3.41	-3.48
WHOQOL-psychological health	79.6 ± 14.5	78.2 ± 13.7	23.4 ± 14.1	158.372	(2,88)	<.001	-0.1	3.94	-3.93
WHOQOL-social relationships	73.9 ± 22.6	73.8 ± 17.3	38.4 ± 20.7	31.208	(2,88)	<.001	-0.01	1.86	-1.64
WHOQOL-environment	82.1 ± 9.6	83.5 ± 10.7	52.8 ± 13.3	71.948	(2,88)	<.001	0.14	2,56	-2.54
RSP-items indicating no sexual activity (in %)	10.5 ± 17.3	9.4 ± 13.9	45.8 ± 35.0	22.979	(2,89)	<.001	-0.07	-1.49	1.35

	HC	*HTEW*	*PTSD*	*Statistics*					
	AM ± SD	AM ± SD	AM ± SD	F	df	p	$d_{HTEW-HC}$	$d_{HTEW-PTSD}$	$d_{PTSD-HC}$
RSP	82.8 ± 28.4	84.8 ± 24.2	39.5 ± 33.4	24.114	(2,89)	<.001	0.08	1.57	-1.40
RS	146.2 ± 14.9	144.4 ± 14.6	83.4 ± 20.6	137.022	(2,89)	<.001	-0.13	3.47	-3.53
RS-personal competence	101.6 ± 9.7	101.1 ± 9.2	61.6 ± 16.3	109.370	(2,89)	<.001	-0.05	3.1	-3.06
RS-acceptance of self & life	44.7 ± 6.9	43.3 ± 6.6	21.8 ± 5.5	124.041	(2,89)	<.001	-0.21	3.55	-3.7

Note. F and p represent the F- and p- values of the respective one-way analysis of variance (ANOVA); d represents the pairwise compared effect sizes after Cohen; df=degrees of freedom; AM=arithmetic mean; SD=standard deviation; SWLS=Satisfaction with Life Scale; EQ-5D=Quality of life measure of the EuroQol Group; WHOQOL-BREF=World Health Organization Quality of Life BREF Version; RSP=Resources in Sexuality and Partnership; RS=Resilience Scale.

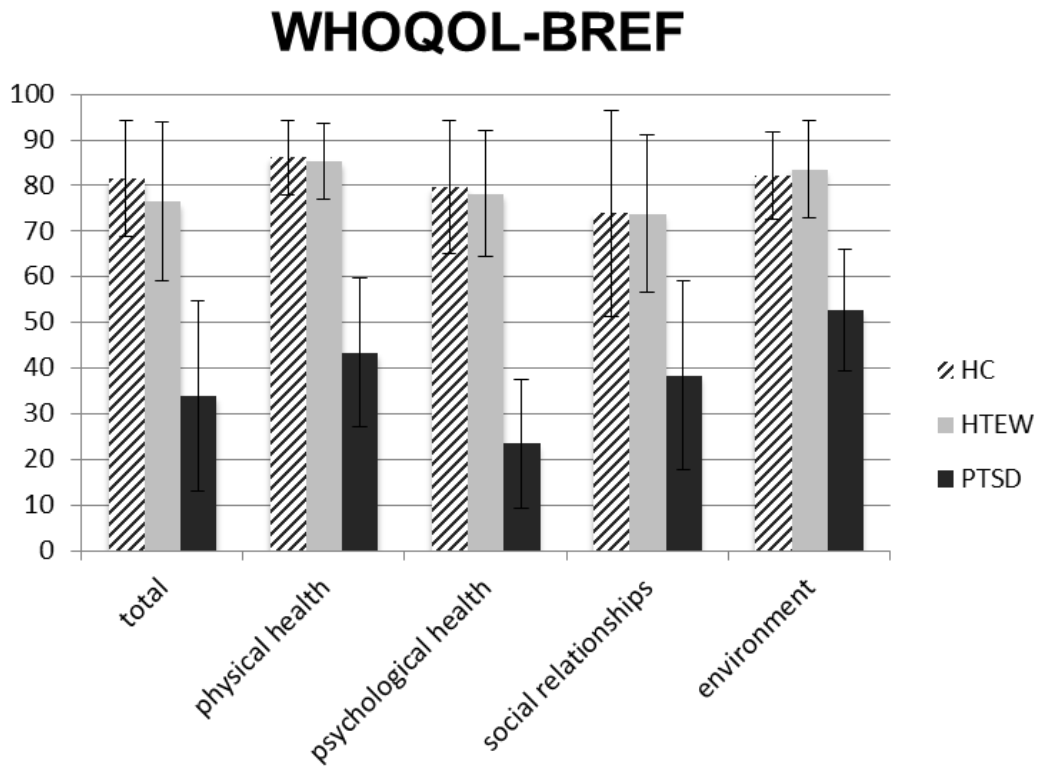

Figure 3. WHOQOL-BREF total and subscale scores for all three groups. Error bars are depicted as standard deviations (SD)

Examining the incidence of sexual behaviors, analyses of the RSP indicated that both individuals in the HTEW and HC groups participated significantly more often in

sexual activities with partners, experienced more sexual satisfaction, and had felt more attractive in the last 4 weeks compared to PTSD-patients (F(2,89)=22.979, p<.001; see Table 4 and Fig. 4).

Concerning sexual satisfaction, individuals of the HTEW and HC groups were significantly more satisfied with their sexuality compared to PTSD-patients (F(2,89)=24.114, p<.001; see Table 4). In a second step, median split analyses were conducted for the PTSD group to investigate separately the satisfaction scores of PTSD-patients for sexually active versus sexually inactive participants. The PTSD-patient group was subdivided by median split into two groups: PTSD-patients scoring more items than median with "did not happen," referred to as low_sexually_active versus PTSD-patients scoring fewer items than median with "did not happen," referred to as high_sexually_active. Whereas in the analyses of sexual satisfaction of the three groups (HTEW versus HC versus. PTSD-patients) the PTSD-patients reported significantly less sexual satisfaction, this pattern changed when PTSD-patients were split into two groups (low_sexually_active versus high_sexually_active). Post-hoc pairwise comparisons showed that individuals in the HTEW group, individuals in the HC group, and high_sexually_active PTSD-patients (AM = 67.33) did not differ significantly in sexual satisfaction ($p_{HTEW\text{-}high\_sexually\_active\_PTSD}$=.147, $d_{HTEW\text{-}HighPTSD}$=0.72; $p_{HC\text{-}high\_sexually\_active\_PTSD}$=.287, $d_{HC\text{-}HighPTSD}$=0.57) and were comparably satisfied, whereas low_sexually_active PTSD-patients (AM = 13.50) did differ significantly from all other groups (p<.001) with significantly lower satisfaction scores (RSP_{HTEW}:84.82; RSP_{HC}:82.77; $RSP_{high\_sex\_active\_PTSD}$:67.33; $RSP_{low\_sex\_active\_PTSD}$:13.50).

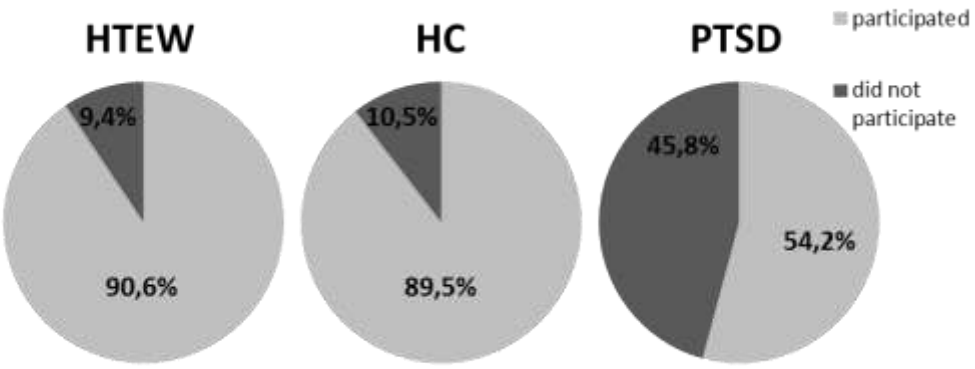

Figure 4. Percentage of items indicating participation in sexual activities with partners, and feeling more sexually satisfied and attractive within the last four weeks

2.4.4 Resilience

Individuals in both the HTEW and HC groups reported significantly higher resilience scores compared to PTSD-patients. This pattern was observed for the total score as well as the two subscale scores ($F_{total}(2,89)=137.022$, $p \leq .001$; $F_{personal\ competence}(2,89)=109.370$, $p \leq .001$; $F_{acceptance}(2,89)=124.041$, $p \leq .001$; see Table 4).

2.5 Discussion

This study investigated subthreshold symptoms of PTSD-specific and general psychopathology and impairments in global functioning, quality of life, and sexuality in women with a history of potentially traumatic CSA/CPA without current or lifetime axis-I disorders or BPD. Results of this particular group were compared to those of women with PTSD related to a history of potentially traumatic CSA/CPA and potentially other axis-I disorders and healthy control women without experiences of PTE. Overall, we did not find any psychopathology in individuals in the HTEW group, who did not show subthreshold psychopathologic symptoms or exhibit unspecific restrictions in psychological well-being. Additionally, no measurable restrictions in quality of life and sexual satisfaction were observed. They showed a high level of functioning (e.g., GAF:87 and 51.6% are working full time) and a low level of psychopathology (e.g., BSI: 0.3; BDI-II:4.5), which was comparable to those levels in the HC group.

These findings confirm that psychopathology is not an inevitable consequence of experiences of PTE. Prospective longitudinal studies and large epidemiologic studies have shown that the conditional probability of developing PTSD range between 12 and 20% (Maercker et al., 2008; Molnar, Buka, et al., 2001) and that many individuals affected by PTE did not seek psychotherapeutic help in the aftermath of PTE (Cutajar et al., 2010). Our findings suggest that PTE such as CSA or CPA per se do not explain the development of psychopathology in the aftermath of PTE. It is more likely that an interaction of different factors such as perceived support from others following trauma, health status at time of trauma, psychopathology in family of origin, psychological problems prior to trauma, peritraumatic dissociation, cognitive abilities, and personality factors plays a role in the process of overcoming PTE (DiGangi et al., 2013; Ozer, Best, Lipsey, & Weiss, 2008).

Several possible explanations could explain the differences occurring in individuals in the HTEW group versus PTSD-patients in our study. While both groups experienced childhood physical or sexual abuse, differences between the groups may be related to the experience of emotional abuse. In our study, PTSD-patients experienced a significantly higher frequency of emotional abuse and emotional neglect compared to HTEW participants. This would be in line with findings by Nash et al. (1993) stating that pathological family environments account for psychological impairments rather than does the experience of sexual abuse. A similar pattern was observed by Corso et al. (2008) concerning the impact of emotional abuse on QoL. Here, previous research shows that experiences of emotional neglect had the strongest impact on perceived QoL followed by sexual abuse and physical abuse. Contradicting these findings, Lewis et al. (2016) found that children with a history of CSA had significantly more behavioral problems with greater externalizing and internalizing problems compared to children, who were maltreated but not sexually abused. However, in our study, individuals in the HTEW group experienced a significantly higher frequency of emotional abuse and emotional neglect compared to individuals in the HC group and did not differ in psychopathology and QoL.

Furthermore, the traumatized but healthy participants in our study reported almost no guilt cognitions concerning the traumatic event, whereas PTSD-patients reported moderate to intense guilt cognitions. Our findings are in line with other studies that found that not the trauma per se but rather the meanings of traumatic events are important (DiGangi et al., 2013; Ozer et al., 2008). Guilt is defined as a belief that one should have thought, felt or acted differently (E. S. Kubany & Watson, 2003), so to speak, an evaluation of one's own behavior as failure. In the context of experiences of abuse, guilt cognitions are usually about not having defended oneself enough or having deserved what had happened (Briere & Elliott, 1994).

The result that individuals in the HC and HTEW groups have almost identical scores on the resilience scale is surprising, given the fact that overcoming a history of CSA or CPA without any indicators of psychopathological symptoms can be referred to as being resilient according to most definitions of resilience ("relatively positive psychological outcomes"; Bonanno & Diminich, 2013, p. 1; Rutter, 2006). Accordingly, the individuals in the HTEW group should have values on any resilience scale that exceed general population norm values and differ from a healthy population. One could argue that individuals in the HTEW group were resilient at a

different time of their lives, namely, the years after the trauma, and that their score has declined in the process of coping with the trauma. Considering that the Resilience Scale was designed to measure resilience in adulthood, it seems more likely that other protective factors helped these individuals in childhood or adolescence to be resilient and survive in good psychological health (for protective factors in children, see Brownlee et al., 2013). There is an increasing discussion on the operationalization of resilience as a personality pattern or bundle of protective factors (Kalisch, Müller, & Tüscher, 2015).

Some limitations of the study have to be considered. A first limitation relates to the representative state of our HTEW group for this population. In our study, the healthy subgroup of participants with a history of potentially traumatic CSA and CPA (HTEW) was highly selective due to the restrictive inclusion criteria. None of the individuals in the HTEW group in this study fulfilled the criteria of any mental axis-I disorder or BPD, never attended psychotherapy sessions, and never took psychotropic drugs. This group seemed to subjectively and objectively get along with what happened and decided to voluntarily participate in this study and communicate about what had happened to them. We chose this healthy subsample to prevent the data in the HTEW group from being affected by ramifications of other psychopathologies such as depression or anxiety disorders. This procedure strengthens our internal validity at an expense of external validity. Also, this limitation relates to the representative state of our PTSD sample. The PTSD sample in our study comprised highly impaired participants that would possibly meet the criteria for complex PTSD that comprises elevated PTSD symptoms as well as affective dysregulation. Second, all trauma data were obtained by (retrospective) self-report. Therefore, on the one hand, a participant's recollection of CSA and CPA could have been influenced by recall bias. There is controversial evidence concerning accuracy of retrospective self-report of childhood adverse events. For example, Fergusson et al. showed that claims about limitations of retrospective reports of CSA/CPA may have been overstated and that well collected retrospective data may provide valid information (Fergusson, Horwood, & Boden, 2011). Contradicting this finding, a recently published study by Mills et al. found a disparity between the incidences of CSA when measured by retrospective self-report or prospective government agency notification (Mills et al., 2016). On the other hand we do not know for sure, whether the reported psychometric data represent a relatively stable condition or might have been biased by current

psychological distress. Third, the design of this study was cross-sectional. Thus, we cannot ascertain any cause and effect relationship between experiences of PTE in childhood or adulthood and actual psychopathological characteristics, resilience scores, and satisfaction with quality of life and sexuality.

Our study has at least one implication for future research. In our study, we quantitatively examined the dimensional distribution of psychopathology in individuals in the HTEW group. A very interesting future study could conduct qualitative interviews with healthy participants affected by potentially traumatic CSA and CPA to find out what helped them overcome these experiences of PTE in such a resilient way without developing any mental disorders.

2.6 Conclusions

The present study showed that participants with a history of potentially traumatic childhood abuse without axis-I disorder or BPD show a high level of functioning and a very low level of pathological impairment that is comparable to the level of healthy controls. The contribution of this study relates to characterizing healthy participants affected by potentially traumatic CSA/CPA with regard to psychopathology. The findings of this study confirm earlier findings that experiences of PTE per se do not necessarily go along with the development of psychopathology or impaired quality of life, sexuality, self-esteem or guilt cognitions. Further studies are needed to determine what helps individuals in the aftermath of PTE to turn out in a resilient way.

3 STUDY II – Indicators of stress levels and stress responsiveness in women with a history of childhood abuse: Disentangling the effects of trauma and psychopathology.

3.1 Abstract

Background: Patients with posttraumatic stress disorder (PTSD) show alterations on a broad spectrum of variables related to stress. However, it is unclear to which extent these alterations relate to the diagnosis of PTSD, or to experiences of adverse childhood experiences (CA), such as childhood sexual or childhood physical abuse (CSA/CPA). To clarify this question, we compared healthy trauma-exposed women (HTEW; n=33) who had a history of CA, but did not meet criteria of any lifetime axis-I disorder or borderline personality disorder with a) patients with PTSD related to CA (n=33) and b) healthy controls (HC) with no history of CA (n=32). We investigated i) indicators of baseline stress levels (subjective stress ratings, dissociation, heart rate, heart rate variability, and pain sensitivity) and ii) stress responses to a validated stress induction paradigm with respect to these indicators. Large effect-sizes separated HTEW from the PTSD group with respect to subjective stress ratings, subjective dissociation ratings, heart rate, and heart rate variability, at baseline as well as with respect to subjective stress and dissociation in response to stress-induction. In contrast, effect-sizes between HTEW and HC were consistently small. The results suggest that high levels of subjective stress, dissociation, and a low heart rate variability are rather related to the diagnosis of PTSD than to experiences of CA per se. However, our data also indicate that a small proportion of altered stress response relates to CA.

3.2 Theoretical Background

By definition, a substantial part of posttraumatic stress disorder (PTSD) is related to a persistent impact of the trauma on the stress system of the person concerned. Alterations of the stress system are reflected in a variety of symptoms including chronic hyperarousal, a generally heightend startle response, disturbed sleep, as well as well as emotional distress and physiological reactivity in response to trauma-related cues (American Psychiatric Association, 2013b). All of these stress-related symptoms have been repeatedly reported to be elevated in participants with PTSD (e.g., Buckley & Kaloupek, 2001; Orr et al., 2003; Shalev, Orr, Peri, Schreiber, & Pitman, 1992; Zoladz & Diamond, 2013).

On a psychopathological level, PTSD is further characterized by high levels of baseline as well as stress-related dissociation: According to a recent meta-analysis

that breaks down the levels of dissociation by diagnoses of mental disorders (Lyssenko et al., 2017), PTSD exhibited the second highest (next to dissociative identity disorder) level of retrospectively rated trait dissociation. Likewise, PTSD-patients experienced elevated symptoms of dissociation in response to experimentally induced stress (Lanius et al., 2002) as well as in response to post military traumatic events (Bremner & Brett, 1997) as compared to traumatized participants without PTSD. In the study by Lanius and colleagues (2002), all but three participants had a history of sexual and physical abuse in childhood, adolescence and even in adulthood. When further examining the role of experiences of adverse childhood experiences (CA) on dissociation, a study by Rodriguez-Srednicki (2002) showed that female college students with a history of CA showed elevated baseline levels of dissociation as compared to female college students without such experiences. Unfortunately, the authors did not control for mental disorders which complicates differentiating between effects of CA and effects of PTSD on baseline dissociation.

On a physiological level, a broad spectrum of indicators of stress has been investigated in participants with PTSD. Among the most consistent findings is a heightened basal heart rate (HR) in PTSD. In their meta-analysis, Buckley & Kaloupek (2001) reported the basal HR to be increased in participants with PTSD in comparison to controls who have been and who have not been exposed to a previous trauma. This meta-analytic finding has been extended by recent lines of research demonstrating an increased HR in the subject's usual environment (e.g., Buckley, Holohan, Greif, Bedard, & Suvak, 2004) as well as in traumatized monozygotic twins who were discordant with respect to trauma exposure (Orr et al., 2003). In this study, veterans with PTSD showed significantly increased HR as compared to veterans not meeting criteria of PTSD as well as compared to their monozygotic twins without combat exposure. These results exclude that increased HR solely originates from a susceptibility of PTSD-patients to react to an unfamiliar environment (i.e., a familial vulnerability factor), and it seems to be an acquired sign of PTSD rather than the consequence of a trauma. Besides elevated baseline levels of HR, participants with PTSD (but not healthy controls) have also been shown to react with an increase of the HR when confronted with unpleasant stimuli (Adenauer et al., 2010). Other indicators of basal cardiovascular activity such as systolic and

diastolic blood pressure support the notion of an increased autonomic activity in PTSD, but the findings are less consistent as with HR (Zoladz & Diamond, 2013).

Studies on autonomic activity in PTSD were complemented by studies assessing the tone of the parasympathetic nervous system in participants with PTSD. Most of these studies are based on the heart rate variability (HRV), some on the respiratory sinus arrhythmia (RSA) as indicators of the activity of the parasympathetic nervous system. According to the meta-analysis by Chalmers and colleagues (2014) comprising four studies comparing the HRV (in the time domain), HRV was significantly lower in participants with PTSD than in controls not meeting the criteria of PTSD. As with basal HR, research on twins discordant for PTSD favors the interpretation that reduced HRV is a manifestation of PTSD, itself, rather than a genetic predisposition (Shah et al., 2013).

Furthermore, associations between experiences of CA and the individuals' baseline stress levels as well as responsiveness to stress have been examined on a psychophysiological level including HR and HRV. In a study by Heim and colleagues (2000), stress-responses were altered in individuals with experiences of CA. More specifically, healthy women with experiences of CA (n=14) showed increased HR in response to psychosocial stress as compared to healthy women without experiences of CA (n=12).

With respect to HRV, investigations have revealed that PTSD-patients show reduced baseline HRV as compared to trauma exposed controls without PTSD (for systematic review see Sammito et al., 2015). But, the respective studies encompassed samples with all kinds of traumatic events (e.g., combat, firemen, sexual abuse in adulthood) but not a purely CA sample. A previous study examining the role of experiences of CA on the baseline sympathetic nervous system (SNS) activation while being sexually aroused was conducted by Rellini and colleagues (2006). The authors examined baseline SNS activation of three groups: women with experiences of CSA not meeting diagnostic criteria of PTSD (n=8), women with CSA and a diagnosis of PTSD (n=10) as well as HC without a history of CSA (n=10). Both, women with a history of CSA as well as women with a history of CSA and PTSD showed heightened SNS activation as compared to HC. When examining HRV in response to distressing tasks in traumatized participants with and without PTSD, one study by Norte and colleagues (2013) has found significantly reduced HRV in PTSD

participants as compared to traumatized participants without PTSD, suggesting that impaired HRV seems to be associated with PTSD, rather than with traumatic experiences per se. But, traumatic experiences in the respective study encompassed urban violence which was not restricted to childhood and adolescence. In a study by Scheeringa and colleagues (2004), traumatized children either with but also without PTSD demonstrated a reduction of HRV in response to traumatic reminders, relative to non-traumatized controls. Children participating in that study had experienced all different kinds of traumatic events such as invasive medical procedure, domestic violence, or motor vehicle accidents. In contrast, Lorenz and colleagues (2015) only examined women with a history of CA. They could show that women with a history of CSA demonstrated reduced HRV as compared to women without a history of CSA while writing a distressing essay. But, in the group of CSA exposed participants, the authors did not control for mental disorders. In synopsis of the existing literature, it is difficult to conclusively differentiate between effects of CA and effects of PTSD on psychophysiological stress markers both at baseline as well as in response to stress due to a variation in inclusion criteria (participants experienced traumatic events from different trauma categories, mental disorders were either not assessed or not controlled for) and due to small sample sizes.

Further stress-related physiological variables which have been repeatedly investigated for PTSD in experimental settings include the startle response and pain sensitivity. While the results are not entirely consistent (for a discussion see Zoladz & Diamond, 2013), most studies have found a heightened startle response in participants with PTSD (e.g., Glover et al., 2011; Shalev, Peri, Orr, Bonne, & Pitman, 1997). With respect to sensitivity to painful stimuli induced with various methods such as a thermode (Geuze et al., 2007) or a cold pressor test (Mostoufi et al., 2014), participants with PTSD showed significantly lower pain sensitivity than both clinical and healthy controls.

Although there are also contradictory findings, suggesting no differences or even stress-induced hyperalgesia in PTSD-patients as compared to HC, the literature predominantly suggests stress-induced hypalgesia in PTSD-patients (for review see Moeller-Bertram et al., 2012). As indicated by several studies, hypalgesia in PTSD-patients is getting more pronounced in response to stress, and this increase in hypalgesia is more pronounced in participants with PTSD as compared to healthy controls (Asmundson & Katz, 2008; Defrin et al., 2008; Kraus et al., 2009; Strigo et

al., 2010). With respect to experiences of CA, a study by Fillingim & Edwards (2005) showed that women with a history of CA reported significantly decreased sensitivity to pain under baseline conditions than women without a history of CA. However, the authors do not report whether they assessed and controlled for mental disorders which prevents from disentangling the effects of a history of CA per se against effects of a diagnosed mental disorders like PTSD. In contrast, Gómez-Pérez and colleagues (2013) examined pain sensitivity via cold pressor test in trauma-exposed women with PTSD, trauma-exposed women without PTSD and HC without traumatic experiences. Trauma-exposed women with PTSD reported significantly higher pain sensitivity than HC but did not differ significantly from trauma-exposed women without PTSD, suggesting that trauma exposure per se and not the development of PTSD in the aftermaths seems to be associated with impaired pain sensitivity. But, as a limiting factor, PTSD in this study was assessed only by means of a questionnaire (Davidson Trauma Scale; Davidson et al., 1997) and it did not comprise a pure CA sample, as participants had experienced all kinds of traumatic events.

To sum up, a large body of research has shown elevated stress responsiveness in PTSD-patients as compared to HC on a broad spectrum of stress markers. Consequently, elevated subjective stress as well as dissociation are regarded as symptoms of PTSD in DSM-5 (American Psychiatric Association, 2013b). However, concerning psychophysiological stress markers (HR, HRV, pain sensitivity), to date, it is difficult to disentangle the effects of psychopathology in form of a PTSD diagnosis and trauma exposure, especially CA, per se, which is due to methodological issues: While some studies on participants with a history of CA suggest an effect or trauma exposure on altered stress responsiveness (i.e., Gómez-Pérez & López-Martínez, 2013), other studies point to an effect of PTSD on altered stress responsiveness (i.e., Lanius et al., 2002). However, the majority of studies described above included participants with a history of CA with and without PTSD, the classical trauma control design, not controlling for mental disorders other than PTSD. Therefore, results might be affected by ramifications of other mental disorders such as major depression, which is known to be related to impaired autonomic regulation, characterized by increased sympathetic and/or reduced vagal modulation (for review see A. D. Brown et al., 2009). Furthermore, previous studies included participants from different trauma categories, i.e., motor vehicle accident (e.g., Lanius et al., 2002; Scheeringa

et al., 2004) or veterans (e.g., Bremner, Narayan, et al., 1999; Norte et al., 2013) which are known to be related to different probabilities in setting off PTSD and psychopathology in trauma response (for summary see Briere & Spinazzola, 2005; Rausch et al., 2016).

Consequently, more research is needed to disentangle whether these subjective and psychophysiological variables of stress responsiveness might be seen as diagnostic correlate of PTSD rather than being related to experiences of CA per se. Therefore, studying participants with experiences of CA without any lifetime mental disorders, will elucidate whether heightened stress responsiveness is related to the diagnosis (PTSD), or rather to experiences of CA per se. Increased knowledge in this field might help identifying diagnostic markers for pathologic trauma sequelae, as well as resilience factors. Accordingly, we focused on a sample of healthy trauma exposed women (HTEW) with a history of CA who did not meet criteria of any lifetime mental disorders, and compared this sample to participants with PTSD after CA, and non-traumatized healthy women (HC). Self-ratings of stress-related variables used in these comparisons included subjective stress and dissociation; psychophysiological variables included heart rate, heart rate variability, and pain sensitivity. All of these variables were compared both at baseline and in response to an established stress-induction paradigm. We hypothesized that with regard to subjective indicators of stress, HTEW as well as HC show lower baseline stress levels as well as lower stress reactivity than PTSD-patients. With regard to psychophysiological indicators of stress, we expected that HTEW show a larger stress reactivity than HC, but a lower reactivity than PTSD-patients.

3.3 Methods

3.3.1 Sample

A total of 98 women participated in this study, of whom 33 were healthy trauma-exposed women (HTEW) with a history of CSA/CPA before the age of 18 who did not meet criteria of any lifetime axis-I diagnosis or borderline personality disorder (BPD). We also included 32 healthy controls with neither experiences of CSA/CPA nor other CA (HC), and 33 patients with PTSD related to CSA/CPA (PTSD-patients) and at least three relevant diagnostic features of BPD.

Inclusion criteria for HTEW were experiences of CSA or CPA before the age of 18. Since this study was part of a randomized controlled trial comparing two different

outpatient psychological treatments for complex PTSD and co-occurring BPD-features, inclusion criteria for PTSD-patients were a diagnosis of PTSD according to DSM-5 related to CSA and CPA and meeting at least 3 diagnostic criteria of BPD as defined by the International Personality Disorder Examination (IPDE; Loranger et al., 1997). Exclusion criteria for HTEW and HC were a lifetime diagnosis of any axis-I disorder or BPD, the intake of any psychotropic drugs, or having received a psychotherapeutic intervention ever in lifetime. Additionally, for HC, experiences of CSA/CPA or any other CA that met the PTSD criterion A were an exclusion criterion. Exclusion criteria for PTSD-patients were a lifetime diagnosis of schizophrenia or bipolar-I disorder, current substance dependence, a body mass index <16, or mental retardation.

Diagnostic interviews were conducted by trained clinical psychologists using structured clinical interviews. Lifetime axis-I disorders were assessed using the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV; Wittchen, 1997), and BPD was assessed using the IPDE (Loranger et al., 1997). PTSD in the PTSD group was additionally assessed using the Clinician Administered PTSD Scale for DSM-5 (CAPS; Weathers et al., 2013). Experiences of CA including CSA/CPA were assessed by means of the Childhood Trauma Questionnaire (CTQ; D. P. Bernstein & Fink, 1998). Additionally, further clinical measures like the Beck Depression Inventory II (BDI-II; Hautzinger et al., 2006), the Brief Symptom Inventory (BSI; Derogatis, 1993), the Davidson Trauma Scale (DTS; Davidson et al., 1997) and the Dissociative Experience Scale (DES; Freyberger et al., 1998) were acquired. All three groups were matched for age and years of education. Demographic and clinical characteristics for HTEW, HC and PTSD-patients are summarized in table 5.

Table 5 Demographic and clinical variables of healthy controls (HC), healthy trauma-exposed women (HTEW) and PTSD-patients (PTSD).

	HC (n=32)	***HTEW (n=33)***	***PTSD (n=33)***	***Statistics***					
	AM ± SD	AM ± SD	AM ± SD	*F/t*	df	*p*	$d_{HTEW-HC}$	$d_{HTEW-PTSD}$	$d_{PTSD-HC}$
Age	31.8 ± 8.3	31.7 ± 12.4	35.4 ± 11.8	1.242	(2, 95)	.293	-0.01	-0.31	0.36
BDI-II	4.3 ± 4.6	4.8 ± 6.1	34.2 ± 10.1	177.208	(2, 95)	< .001	0.09	-3.63	-4.04
BSI	0.2 ± 0.2	0.3 ± 0.3	1.8 ± 0.6	129.492	(2, 95)	< .001	0.40	-3.33	-3.97
DTS	-	11.7 ± 12.4	80.0 ± 19.1	5.098	49.452	< .001		-4.34	
DES	4.1 ± 3.3	7.0 ± 6.4	27.0 ± 12.5	71.216	(2,92)	< .001	0.59	-2.12	-2.87
Years of education	N (%)	N (%)	N (%)	*H* (2)=3.726, *p*=.155					
9 years	1 (3.1)	0 (0.0)	2 (6.1)						
10 years	9 (28.1)	11 (33.3)	15 (45.4)						
12 years	22 (68.8)	22 (66.7)	16 (48.5)						
Comorbidities (current)	N (%)	N (%)	N (%)						
Any anxiety disorder	-	-	21 (63.6)						
Any mood disorder	-	-	21 (63.6)						
Any eating disorder	-	-	3 (9.1)						
Any substance abuse	-	-	1 (3.0)						
BPD	-	-	20 (60.6)						
Psychotropic medication	N (%)	N (%)	N (%)						
Free of med.	32 (100)	33 (100)	7 (21.2)						
SSRI	-	-	9 (27.3)						
SNRI	-	-	11 (33.3)						
Other anti-depressants	-	-	11 (33.3)						
Neuroleptics	-	-	13 (39.4)						
Mood stabilizer	-	-	5 (15.2)						
Methyl-phenidate	-	-	1 (3.0)						

Note. F and p represent the F- and p- values of the respective one-way ANOVA; H represents the H of the Kruskal-Wallis test; AM=arithmetic mean; SD=standard deviation; df=degrees of freedom; d represents the pairwise compared effect sizes after Cohen. Post-hoc t-tests were performed at a significance level of p< 0.05 Bonferroni-corrected. BDI-II= Beck depression Inventory II; DTS= Davidson Trauma Scale, BSI= Brief Symptom Inventory; DES= Dissociative Experience Scale; BPD= Borderline personality disorder; SSRI= selective serotonin reuptake inhibitors; SNRI= serotonin-norepinephrine reuptake inhibitor.

3.3.2 Recruitment

PTSD-patients were recruited from a larger randomized controlled trial (RCT) comparing two different psychological treatments for PTSD after CPA/CSA. HTEW and HC were recruited via newspaper advertisements, the internet, and the distribution of flyers in public places. As the central inclusion criterion for both the HTEW and PTSD groups was a history of CSA or CPA, we were seeking to obtain similar levels of CSA/CPA in these two groups. Accordingly, the HTEW and PTSD groups were matched with respect to the subscales of “Sexual Abuse” and “Physical Abuse” of the CTQ (D. P. Bernstein & Fink, 1998; see table 6) as implemented in a study by Roy and colleagues (2011). As a result, 33 out of 69 PTSD-patients of the original sample in the RCT were selected. All participants gave their written informed consent for participation. Approval was obtained from the independent Ethics board of the Medical Faculty Mannheim at Heidelberg University.

Table 6 Childhood Trauma Questionnaire total and subscale scores of healthy controls, healthy trauma-exposed women and PTSD-patients.

	HC (n=32)	***HTEW (n=33)***	***PTSD (n=33)***	***Statistics***					
	AM ± SD	AM ± SD	AM ± SD	*F*	df	*p*	$d_{HTEW-HC}$	$d_{HTEW-PTSD}$	$d_{PTSD-HC}$
CTQ_Sexual Abuse	5.1± 0.2	11.4 ± 6.1	11.8 ± 5.9	19.008	(2, 95)	< .001	1.97	-0.07	2.16
CTQ_Physical Abuse	5.6± 1.7	9.4 ± 3.7	9.6 ± 4.3	14.015	(2, 95)	< .001	1.40	-0.05	1.32
CTQ_Emotional Abuse	6.7 ± 2.1	12.8 ± 4.9	17.4 ± 5.0	51.800	(2, 95)	< .001	1.73	-0.93	2.99
CTQ_Emotional Neglect	7.8 ± 3.4	12.3 ± 4.5	17.2 ± 4.7	40.672	(2,95)	< .001	1.14	-1.07	2.32
CTQ_Physical Neglect	5.9 ± 1.8	7.2 ± 2.4	10.1 ± 3.0	24.227	(2,95)	< .001	0.62	-1.07	1.74
CTQ_Total score	31.0 ± 6.6	52.6 ± 13.1	66.0 ± 14.9	69.153	(2, 95)	< .001	2.18	-0.96	3.24

F and p represent the F and p-values of the respective one-way analysis of variance (ANOVA); d represents the pairwise compared effect sizes after Cohen. df=degrees of freedom; AM=arithmetic mean; SD=stand deviation; CTQ=Childhood Trauma Questionnaire.

3.3.3 Experimental design

To allow habituation to the testing situation, the procedure started with a 5-minute baseline phase during which participants watched a neutral animal movie and were instructed to lean back and relax. Subsequently, thermal pain was induced using a thermode for 30 seconds (for details see below); this was followed by ratings of pain sensitivity, subjective stress rating and dissociation. The following task to induce stress was adapted from Kolotylova and colleagues (see Kolotylova et al., 2010). After a short training phase in which participants were introduced to the task, two identical consecutive stress induction phases with a duration of 4 minutes were applied by means of the Mannheim Multicomponent Stress Test (MMST; Kolotylova et al., 2010) to achieve a high level of stress. After each of the two stress induction phases, pain stimuli were applied for a duration of 30 seconds followed by ratings on pain sensitivity, subjective stress rating and dissociation. For a summary of the experimental design, see figure 5.

Figure 5. Experimental design

3.3.4 Stress induction with the Mannheim Multicomponent Stress Test (MMST)

Stress was induced by means of the MMST (Kolotylova et al., 2010) which is a validated paradigm to induce stress in a laboratory setting. This stress protocol combines cognitive, emotional, acoustic, and motivational stressors and has been shown to induce significant changes in subjective stress ratings and in cardiovascular parameters (Kolotylova et al., 2010; Reinhardt, Schmahl, Wüst, & Bohus, 2012). During the MMST, participants were asked to complete a mental arithmetic task (Paced Auditory Serial Addition Test, for description see Gronwall, 1977) whilst pictures with positive and negative valences from the international affective picture set (P. J. Lang, Bradley, & Cuthbert, 1997) were shown in the background and white noise was presented via headphones. Additionally, motivational stress was induced by the instruction of a cover story that participants can either win or lose money depending on the level of correctness of their answers. In this study, two identical

stress sessions for the duration of 4 minutes each, as adapted from Kolotylova and colleagues (2010) were performed with the MMST to effectively maintain a high level of stress during the session. The stress paradigm was presented in a dark room on a projection screen (width approximately 2 x 2 m), using the software Presentation (Neurobehavioral Systems, http://www.neurobs.com).

3.3.5 Indicators of stress

Indicators of stress were assessed from several subjective and psychophysiological parameters that are associated with stress. These parameters encompassed:

Subjective Stress Ratings: To examine the subjective stress level, participants were asked about their acute level of stress after baseline and after each of the two stress induction phases. Subjective stress was assessed by means of a 100 mm visual analogue scale (VAS) ranging from 0="not at all" to 100="very intense".

Dissociation: Acute levels of dissociation were assessed by the total score of the DSS-4 (Stiglmayr, Schmahl, Bremner, Bohus, & Ebner-Priemer, 2009), which is a brief self-report instrument to assess dissociative states. It comprises 4 items that are rated on a 10-point Likert scale ranging from 0="not at all" to 9="very strong". Dissociation was assessed after baseline phase and after each of the two stress induction phases.

Heart Rate (HR) and Heart Rate Variability (HRV): As an objective marker of stress levels and of stress responsiveness, electrocardiographic recordings of the last 4 minutes of the baseline phase as well as of the two 4-minute stress induction phases, parametrized as HR were analyzed. In addition, HRV was calculated for these periods of time (4 minutes baseline and 2 x 4 minutes stress induction). Data inspection and artifact rejection of HR data were performed using Kubios 2.1 (http://kubios.uku.fi). HRV was indexed by the heart rate variability triangular index, which is a very stable and frequently studied parameter in the spectrum of time-domain based HR analyses (Cygankiewicz & Zareba, 2013; Ewing, Martyn, Young, & Clarke, 1985). The HRV triangular index is very robust with regard to noise and artifacts during computerized processing of electrocardiographic recordings and is determined by dividing the total number of normal-to-normal intervals by the maximum of the density distribution.

Pain Sensitivity: Pain stimuli were applied to the inner side of the non-dominant forearm using a contact thermode (TSA 2001-II; Medoc Ltd, Ramat Yishai, Israel)

with a contact area of 3 x 3 cm (Covas 3.20, Medoc Ltd, Ramat Yishai, Israel). The baseline temperature of the thermode was 32 °C. For the duration of pain stimuli (30 seconds), baseline temperature increased to 46°C. In order to avoid habituation to the stimuli, the temperature of pain stimuli oscillated +/- 1°C around 46°C throughout the whole pain application (Ruscheweyh, Stumpenhorst, Knecht, & Marziniak, 2010).

As described above, a total of three pain stimuli were applied: one after the baseline phase and one after each of the two stress induction phases, respectively. Following each pain application, participants were asked to rate the stimuli's painfulness on a 100 mm VAS ranging from 0="no pain at all" to 100="worst imaginable pain" (as used by Hollins, Harper, & Maixner, 2011; Reinhardt, Kleindienst, Treede, Bohus, & Schmahl, 2013).

3.3.6 Statistical analysis

Between-group differences regarding age and self-rated psychopathology (BDI-II, BSI, DTS, and DES) were analyzed with one-way analyses of variance (ANOVAs). A non-parametric test was applied to compare differences in years of education across the three groups. Here, Kruskal-Wallis tests were used for overall comparisons. Pairwise post-hoc comparisons were conducted with Mann-Whitney U tests. These analyses were performed using IBM SPSS (version 21).

Predefined contrasts in a framework of mixed models were used to test the predefined hypotheses comparing (a) HTEW versus PTSD and (b) HTEW versus HC with respect to the dependent variables subjective stress, HR, HRV, pain, and dissociation. In order to exactly mirror the hypotheses to be tested, the group was coded into two dummy variables: HTEW_PTSD contrasting HTEW and PTSD, and HTEW_HC contrasting HTEW and HC. Furthermore, the mixed models included TIME (coded as 0, 1, and 2 for Baseline, Stress 1, and Stress 2, respectively) as a linear main effect and TIME*HTEW_PTSD and TIME*HTEW_HC as interaction terms. The interaction terms were used to test the predefined hypotheses that stress induction has a differential effect on (a) the HTEW versus PTSD groups and on (b) the HTEW versus HC groups. All parameters were estimated using restricted maximum likelihood estimates from the Newton-Raphson algorithm implemented in SAS™ PROC MIXED (v.9.4) and tested for significance using the usual Wald-test based on the comparison of the parameter estimates against their standard errors

(SE). A significance level of p≤.05 (two-tailed) was considered as statistically significant.

3.4 Results

For the analyses of psychophysiological data, the final sample consisted of 30 HTEW, 29 HC and 28 PTSD-patients. Eleven participants had to be excluded from further analyses following visual inspection due to incomplete recording (n=5, 4.9%) or technical artefacts resulting in uncorrectable noise in their heart rate data (n=6, 5.9%).

Subjective stress ratings

The levels of subjective stress ratings of healthy trauma-exposed women (HTEW) were significantly higher than in healthy controls (F(1,89)=7.62, p=.007) and significantly lower than in women with a diagnosis of PTSD (F(1,92)=19.57, p<.001) (see table 7). As illustrated in figure 6a, the effect sizes between HTEW and PTSD groups were particularly large.

A general increase of subjective stress in response to the MMST was observed. However, the predefined contrasts comparing the group specific intensities of stress responses did not significantly separate the HTEW and HC groups (F(1,92)=0.27, p=.607), but indicated a lower increase for the group of HTEW patients when compared to the PTSD group (F(1,92)=5.35, p=.023.

Dissociation

The mean level of dissociation in the HTEW group differed significantly from the level in the HC group ($F($1,92)=11.01, p=.001) and was on a lower level in the HTEW group than in the group of PTSD-patients ($F($1,92)=46.65, p<.001; see figure 6b).

As indicated by a significant main effect, the MMST resulted in a general increase of dissociation ($F(1,89)=8.77$, p<.001). Additionally, dissociation in response to the MMST differed across groups. Both of the predefined Time*Group contrasts were statistically significant (*HTEW* versus *HC:* ($F($1,92)=9.64, p=.003; *HTEW* versus *PTSD:* $F($1,92)=17.25, p<.001): In contrast to the HC group, the mean level of dissociation did increase in the HTEW group and was steepest in the PTSD group (see figure 6b).

Heart Rate (HR) and Heart Rate Variability (HRV)

Throughout the observation period of the study, HR and HRV recorded in the HTEW group were located between those of the HC and PTSD groups (see figure 6c-d): The HR in the HTEW group was significantly higher than in the HC group (F(1,81)=5.18, p=0.026), and significantly lower than in the PTSD group (F(1,81)=6.46, p=0.013). Likewise, the level of the HRV in the HTEW group was significantly lower than in the HC group ($F(1,81)=7.28$ p=.009) and significantly higher than in the PTSD group (F(1,81)=16.73, p<.001).

While an overall increase in the HR and an overall decrease in HRV were observed in response to the MMST, no interaction effects were observed for these outcome variables: The increase of the HR in the HTEW group did not differ significantly from the two other groups (*HTEW* versus *HC:* F(1,81)=0.15, p=.699; *HTEW* versus *PTSD:* F(1,81)=0.37, p=.543). Similarly, the decreases in HRV as observed in the the HTEW group did not differ significantly from the two other groups (*HTEW* versus *HC:* F(1,81)=0.69, p=.409; *HTEW* versus *PTSD:* F(1,81)=1.21, p=.275).

Pain sensitivity

The level of pain sensitivity in the HTEW group was significantly lower than in the HC group (F(1,91)=5.20, p=.025) and tended to be higher than in the PTSD group (F(1,91)=2.99, p=.087) (see figure 6d). As indicated by a significant effect of Time (F(1,91)=6.09, p=.016), pain sensitivity decreased in response to the stress-induction paradigm (MMST). With respect to a moderation of this effect by the group, no significant difference between the HTEW and HC group was detected (F(1,91)=0.90, p=.346). However, the predefined contrast comparing the slopes in the HTEW and PTSD groups indicated a steeper decline for the pain ratings in the PTSD group in response to the MMST (F(1,91)=3.96, p=.0496, see figure 6e).

Potentially confounding effects of childhood trauma severity

HTEW and PTSD groups were matched with regard to sex, age, and years of education. Furthermore, we aimed at minimizing differences in severity of childhood sexual and physical abuse between these groups to prevent dose-dependent effects of CA on the dependent variables and matched scores of the CTQ subscales of Sexual and Physical Abuse. While the matching procedure was successful for these predefined variables, post-hoc testing revealed higher scores with respect to the CTQ total score, which was due to higher scores in the subscales for physical

neglect, emotional neglect and emotional abuse (see table 6). In order to control for the potentially confounding effects of a more pronounced childhood trauma in the PTSD group, the analyses were repeated after including the CTQ total score as an independent predictor into the mixed models. However, the inclusion of the CTQ total score as a covariate did not change the results in a relelvant way. Notably, besides the steeper increase after stress induction in dissociation in the PTSD group as compared to the HTEW, all significant differences between HTEW and PTSD with respect to the levels and the responsiveness reported in table 7 remained statistically significant when controlling for the CTQ total score.

Table 7 Subjective stress ratings, heart rate variability (HRV), pain sensitivity ratings and dissociation under baseline, and after stress induction (Stress 1 and 2).

	Descriptives: Mean (SD) and Cohen's d					**Mixed models:** F-values and p-values		
	HC	***HTEW vs HC***	***HTEW***	***HTEW vs PTSD***	***PTSD***	*Time (manipulation check)*	*Group (predefined contrasts)*	*Time*Group (predefined contrasts)*
Subjective Stress Rating								
Base-line	37.7 (26.4)	d=0.11	40.4 (24.2)	d=-0.90	61.1 (21.5)	*F*(1,89)=7.21 *p*=.026	*HTEW vs HC:* *F*(1,92)=7.62 *p*=.007	*HTEW vs HC:* *F*(1,92)=0.27 *p*=.607
Stress 1	41.2 (30.2)	d=0.13	44.6 (23.7)	d=-1.45	75.6 (18.8)		*HTEW vs PTSD:* *F*(1,92)=19.57 *p*<.001	*HTEW vs PTSD:* *F*(1,92)=5.35 *p*=.023
Stress 2	42.5 (32.6)	d=-0.02	41.8 (25.4)	d=-1.69	76.9 (15.6)			
Dissociation								
Base-line	.1 (.5)	d=0.00	.1 (.4)	d=-1.67	2.2 (2.2)	*F*(1,89)=8.77 *p*=.004	*HTEW vs HC:* *F*(1,92)=11.01 *p*=.001	*HTEW vs HC:* *F*(1,92)=9.64 *p*=.003
Stress 1	.0 (.2)	d=0.66	.3 (.7)	d=-1.41	2.7 (2.8)		*HTEW vs PTSD:* *F*(1,92)=46.65 *p*<.001	*HTEW vs PTSD:* *F*(1,92)=17.25 *p*<.001
Stress 2	.0 (.0)	d=0.84	.3 (.7)	d=-1.74	3.0 (2.5)			

Table 7 Subjective stress ratings, heart rate variability (HRV), pain sensitivity ratings and dissociation under baseline, and after stress induction (Stress 1 and 2).

	Descriptives: Mean (SD) and Cohen's d					**Mixed models:** F-values and p-values		
	HC	***HTEW vs HC***	***HTEW***	***HTEW vs PTSD***	***PTSD***	*Time (manipulation check)*	*Group (predefined contrasts)*	*Time*Group (predefined contrasts)*
Heart Rate								
Base-line	74.8 (9.0)	d=0.30	78.3 (13.5)	d=-0.41	83.3 (10.6)	*F*(1,78)=18.94 *p*<.001	*HTEW vs HC:* *F*(1,81)=5.18 *p*=.026	*HTEW vs HC:* *F*(1,81)=0.15 *p*=.699
Stress 1	86.2 (12.9)	d=0.17	88.7 (15.3)	d=-0.14	90.9 (17.7)		*HTEW vs PTSD:* *F*(1,81)=6.46 *p*=.013	*HTEW vs PTSD:* *F*(1,81)=0.37 *p*=.543
Stress 2	83.8 (11.3)	d=0.29	87.4 (13.8)	d=-0.15	89.7 (16.7)			
Heart Rate Variability (HRV triangular index)								
Base-line	12.2 (4.2)	d=0.34	10.8 (4.0)	d=0.67	8.3 (3.4)	*F*(1,78)=4.70 *p*=.033	*HTEW vs HC:* *F*(1,81)=7.28 *p*=.009	*HTEW vs HC:* *F*(1,81)=0.69 *p*=.409
Stress 1	10.1 (2.8)	d=-0.15	9.7 (2.5)	d=1.04	7.0 (2.7)		*HTEW vs PTSD:* *F*(1,81)=16.73 *p*<.001	*HTEW vs PTSD:* *F*(1,81)=1.21 *p*=.275
Stress 2	10.3 (2.7)	d=-0.23	9.7 (2.5)	d=0.85	7.5 (2.7)			
Pain Sensitivity								
Base-line	64.0 (16.3)	d=-0.47	55.0 (21.7)	d=0.17	51.0 (26.1)	*F*(1,89)=6.09 *p*=.016	*HTEW vs HC:* *F*(1,91)=5.20 *p*=.025	*HTEW vs HC:* *F*(1,91)=0.90 *p*=.346
Stress 1	58.5 (21.0)	d=-0.37	50.1 (24.0)	d=0.21	44.4 (29.3)		*HTEW vs PTSD:* *F*(1,91)=2.99 *p*=.087	*HTEW vs PTSD:* *F*(1,91)=3.96 *p*=.0496
Stress 2	60.8 (23.2)	d=-0.37	52.3 (23.3)	d=0.49	39.7 (28.9)			

Note Mean scores (± SD) and effect sizes (Cohen's d) of subjective stress ratings, dissociation as parametrized by DSS-4 scores, HR, HRV and subjective pain sensitivity ratings, under baseline, and after stress induction (Stress 1 and 2). Predefined contrasts in a framework of mixed models were used to test the predefined hypotheses comparing (a) HTEW vs PTSD and (b) HTEW vs HC with respect to the dependent variables. Furthermore, the mixed models included TIME as a linear main effect and TIME*HTEW_PTSD and TIME*HTEW_HC as interaction terms. The interaction terms were used to test the predefined hypotheses that stress induction has a differential effect on (a) the HTEW vs PTSD groups and on (b) the HTEW vs HC groups.

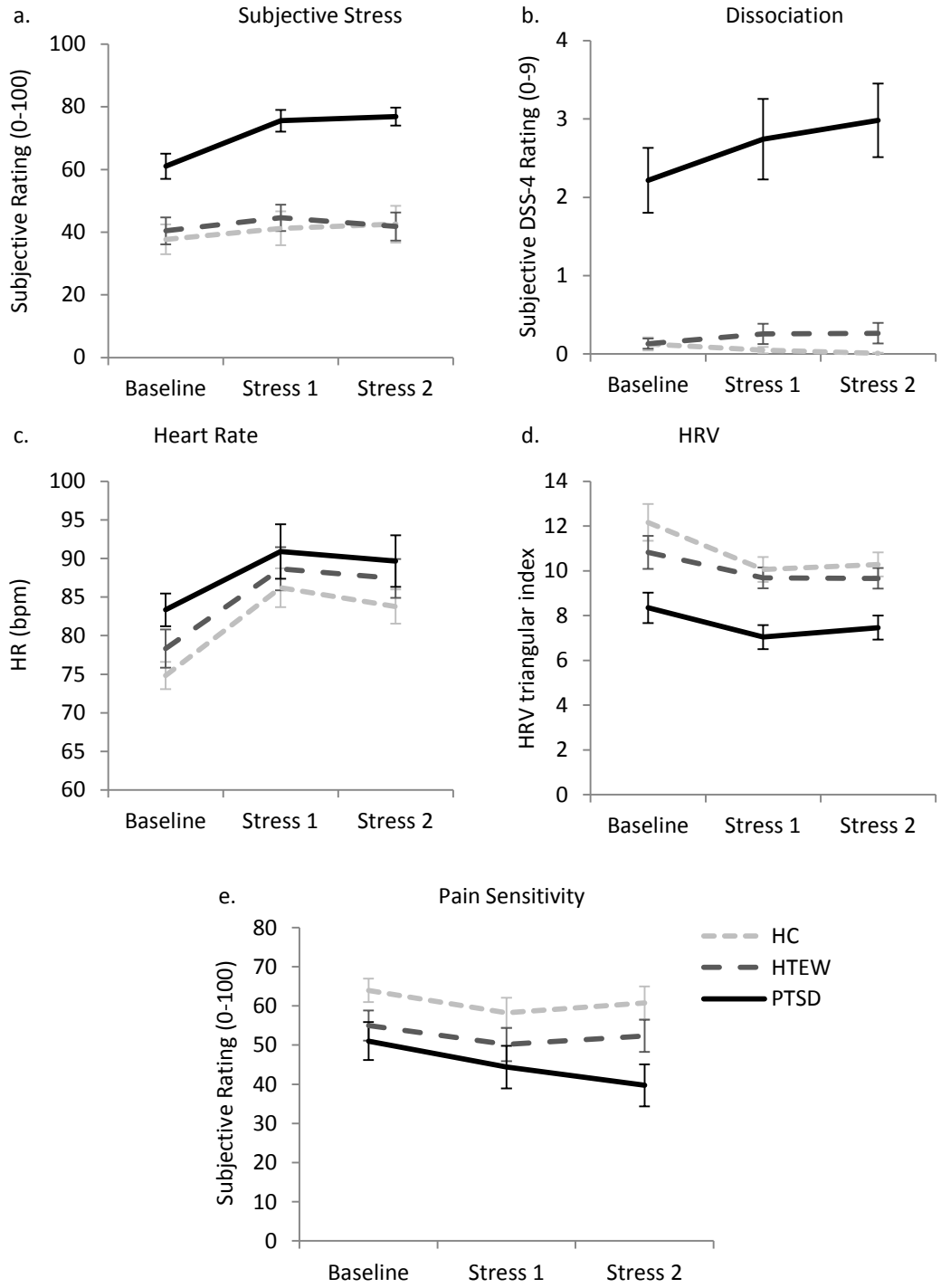

Figure 6. Mean scores (± SD) of a. subjective stress ratings, b. dissociation as parametrized by DSS-4 scores, c. HR, d. HRV and e. subjective pain sensitivity ratings, under baseline, and after stress induction (Stress 1 and 2)

3.5 Discussion

This study investigated to which extent healthy trauma-exposed women (HTEW) who have experienced adverse childhood experiences (CA) show alterations in (i) subjective and (ii) psychophysiological indicators of stress. All of these parameters

(subjective ratings of stress and dissociation on the one hand; HR, HRV, and pain sensitivity on the other hand) were assessed at baseline as well as in response to a validated stress induction paradigm (Kolotylova et al., 2010). To exclude the potentially confounding effects of co-occurring mental disorders, HTEW were required to be free of lifetime axis-I disorders and of borderline personality disorders. The group of HTEW was compared to (i) a group of healthy controls with no history of CA and (ii) a group of patients with PTSD related to CA.

While statistical testing of the mean levels consistently separated the group of HTEW from both groups of HC and PTSD, a comparison of the absolute values and effect sizes (see table 7) provides a more differentiated picture. The most distinct and clinically meaningful effects between HTEW and PTSD-patients were observed for subjective stress parameters. With regard to subjective ratings of stress and dissociation, differences in the absolute mean values between HTEW and PTSD were highly significant, with absolute mean differences being at least 7.7 times the absolute mean differences between HTEW and HC. That means that HTEW were clearly different from PTSD-patients while resembling the group of HC in these subjective parameters related to stress. With respect to the psychophysiological parameters (HR, HRV, and pain sensitivity), the picture was less clear: Although HTEW differed significantly from PTSD-patients with respect to HR and HRV, differences were not as pronounced as for the subjective ratings. With respect to pain sensitivity, comparisons of HTEW and PTSD-patients only approached significance. In comparison to HC, HTEW differed significantly for HR, HRV, and pain sensitivity. On a descriptive level, HR, HRV and pain sensitivity levels of HTEW were located in between levels of HC and of PTSD-patients. The differential results regarding subjective and psychophysiological stress parameters likely relate to the diagnostic criteria of PTSD which include both elevated levels of stress and dissociation (American Psychiatric Association, 2013b). However, it should be noted that HTEW and HC were not significantly different with respect to subjective stress and dissociation and were separated by no larger than very small effect-sizes. Our finding that the absolute differences separating the HTEW and HC groups regarding subjective stress ratings was small, while the differences between HTEW and PTSD was very large, supports previous research stating enhanced responsiveness to stress, both on a subjective as well as psychophysiological level, as one of the central pathophysiological mechanisms in PTSD and therefore seems to be a

correlate of PTSD rather than of trauma exposure per se (Buckley et al., 2004; Norte et al., 2013).

Our results on the other subjectively assessed variable, i.e. dissociation, add to a well-established body of evidence examining participants with various types of potentially traumatic events, with and without PTSD: After potentially traumatic experiences of combat, CSA, and motor vehicle accidents, trauma-exposed participants with PTSD exhibited higher levels of dissociation than trauma-exposed participants without PTSD, in non-stress conditions as well as under stress (Bremner, Narayan, et al., 1999; Bremner, Staib, et al., 1999; Gómez-Pérez & López-Martínez, 2013; Lanius et al., 2002). These findings have been fully confirmed by our study, since both the level of dissociation and the dissociative response to the stress induction were much more pronounced in the PTSD group than in the HTEW and HC groups. Our study extends previous findings as it indicates a significant, albeit small, difference between HTEW and HC in dissociation in reaction to a stress-induction paradigm. However, it has to be kept in mind that a rather low level of dissociation in HTEW might be related to the exclusion criteria in our study: In contrast to the studies by Bremner et al. (1999) and Lanius et al. (2002), trauma-exposed participants without PTSD in our study did not meet criteria of any other lifetime diagnosis of mental disorder.

On a psychophysiological level, challenges in the environment engage the sympathetic nervous system that is associated with energy mobilization, leading to a change in HR. A flexible adaptation of HR results in a high HRV, reflecting the flexibility of an organism to adjust physiological arousal in response to environmental demands (Thayer et al., 2012). With respect to the mean levels of psychophysiological parameters related to stress, HTEW reacted more sensitively to external stressors than HC, but less sensitive than PTSD-patients, as mirrored by elevated HR and reduced HRV in comparison to HC as well as lower HR and elevated HRV as compared to PTSD-patients. However, these effects were only observable at baseline, but not in response to stress.

With respect to baseline HRV, our results confirm and extend previous findings. The higher baseline level of HRV in the HTEW versus PTSD group from our study is in line with various studies (Chang et al., 2013; Dennis et al., 2014; Hauschildt, Peters, Moritz, & Jelinek, 2011; Lee & Theus, 2012) all reporting increased baseline HRV in

trauma-exposed participants without PTSD as compared to PTSD-patients pointing to reduced baseline HRV being a correlate of PTSD, rather than of trauma exposure per se. Our data add to and extend these findings with respect to experiences of CA as the respective authors studied combat veterans (Chang et al., 2013; Dennis et al., 2014), military sexual trauma (Lee & Theus, 2012) as well as experiences of abuse not specified to a certain age (Hauschildt et al., 2011). Furthermore, our study is the first demonstrating a significantly lower HRV in HTEW with a history of CA when compared to healthy women who reported no exposure to CA. However, this finding attenuates the attribution of reduced HRV being associated with PTSD rather than trauma exposure per se. However, an additional role of CA in altering HRV levels cannot be neglected.

With regard to HR, results of the present study are in line with results by the meta-analysis by Buckley and Kaloupek (2001) reporting the basal HR to be increased in participants with PTSD in comparison to controls who have been and who have not been exposed to a previous trauma. Furthermore, results of the present study are partly in line with a study by Orr and colleagues (1998) and by Linda and colleagues (1999) who both found significantly higher baseline HR levels as well as the latter larger accelerative HR responses to startle stimuli in CSA-exposed participants with current PTSD as compared to CSA-exposed participants without PTSD. Unfortunately, no comparison to HC was drawn in that study. Comparisons between HTEW and HC in our study revealed significantly higher HR levels in HTEW than in HC. Finally, results of our study are not in line with the findings by Gómez-Pérez & López-Martínez (2013), who report essentially identical levels of baseline HR in non-trauma-exposed women, trauma-exposed women, and trauma-exposed women with PTSD. It is conceivable that the differences between the findings of our study and the study by Linda and colleagues (1999), contrary to the study by Gómez-Pérez & López-Martínez (2013), are related to differences in experiences of CA. While our study focused on physical and sexual interpersonal trauma during childhood, and the study by Linda and colleagues on CSA, the study by Gómez-Pérez & López-Martínez (2013) included a broad range of traumatic experiences, with emotional abuse being the most common type of trauma.

Taken together, these findings demonstrate that alterations in psychophysiological baseline stress parameters seem to be associated with a diagnosis of PTSD, however one cannot be negligent of an additional role of the experience of CA per

se. Furthermore, alterations in psychophysiological baseline stress parameters might potentially differ relative to the type of adversity experienced. Similar observations are described in a previous study by Bekrater-Bodmann and colleagues (2015) with regard to baseline pain sensitivity: significantly lower pain perception (pain stimuli induced by a thermal grill illusion) was observed in participants with a more severe history of CPA as well as a trend in the same direction for CSA in current BPD patients as compared to remitted BPD patients and HC. Additionally, higher thermal pain thresholds were significantly related to a more severe history of sexual abuse. Other forms of CA (e.g., emotional abuse) were not associated with alterations in pain perception. These findings elucidate that experiences of CSA and CPA seem to cause more severe sequelae in HR and pain sensitivity than other forms of CA.

In the present study, we attempted to minimize differences in severity in CA by matching CTQ scores of the respective subscales of CSA and CPA. Pain sensitivity under baseline conditions of HTEW did not differ significantly from trauma-exposed women with PTSD. This is in line with findings by Gómez-Pérez et al. (2013), but not fully in line with Diener et al. (2012), who reported a significantly higher baseline pain threshold in PTSD-patients as compared to trauma-exposed participants without a diagnosis of PTSD. In contrast to pain thresholds in the study by Gómez-Pérez et al. (2013), baseline subjective pain ratings in the HTEW group were significantly lower than in participants without experiences of CA. Descriptively, HTEW in our study differed from both PTSD-patients and HC and scored about half way between the two other groups. This pattern is in line with a study by Kraus and colleagues (2009), who examined subjective stress ratings in veterans either with or without PTSD and HC. With regard to stress responsiveness, HTEW differed significantly from PTSD-patients but not from HC, with PTSD-patients showing a steeper decline in pain sensitivity over stress induction than HTEW.

One potential reason for conflicting findings in pain perception might possibly be ascribed to the usage of different stress paradigms [i.e., Mannheim Multicomponent Stress Test (Kolotylova et al., 2010), Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993)], pain induction paradigms (cold pressor test in the study by Goméz-Pérez and colleagues, impact stimulator in the study by Diener and colleagues, Thermal Sensory Analyzer in the present study) or dependent variables (pain threshold, subjective rating).

To sum up, this study attempted to disentangle the effects of trauma and psychopathology with regard to several subjective and psychophysiological indicators of stress. Results of the present study suggest that stress levels and stress responsiveness might be linked to both the diagnosis of PTSD and to traumatic events per se. However, since differences between HTEW and PTSD-patients were most distinct in subjective as compared to psychophysiological readouts, subjective stress rating and dissociation might be seen as diagnostic correlates of PTSD, rather than being related to experiences of CA per se. Results of the objective stress indicators draw a less clear picture: Although HTEW differed significantly from PTSD-patients and HC with respect to HR and HRV, differences between HTEW and PTSD-patients were not as pronounced as for the subjective indicators of stress. Apparently, psychopathology in form of a PTSD diagnosis seemed to contribute to more impaired stress responsiveness on a psychophysiological level, but this finding does not exclude an additional role of CA: The data also indicate that a small proportion of altered stress response might relate to experiences of CA per se.

Several limitations of our study should be considered. As just mentioned, individuals in the HTEW group were highly selected due to our inclusion criteria, targeting highly resilient participants. Individuals in the HTEW group did not meet criteria of any lifetime axis-I disorder or BPD, never took psychotropic medication, and never attended psychotherapy sessions. These criteria were chosen in order to strengthen the internal validity of our findings by preventing data from being affected by ramifications of other psychopathologies. Therefore, results regarding our HTEW sample might not be generalizable to trauma-exposed participants who have not developed PTSD but may present other diagnoses such as a major depressive disorder or alcohol dependence. Another limitation to be discussed relates to our PTSD sample. The PTSD sample comprised highly impaired participants, many of whom met criteria for complex PTSD, which encompasses elevated PTSD symptoms as well as affective dysregulation. Due to the high level of impairment, most participants in this group were medicated and had undergone various therapeutical interventions before participating in this study. Both aspects may have had an effect on HR, HRV and on pain sensitivity (for details see Moon, Lee, Kim, & Hwang, 2013). Therefore, the comparison of HTEW and PTSD-patients with regard to HR, HRV and pain sensitivity should be interpreted carefully. Accordingly, our findings should be complemented by future studies on unmedicated PTSD-patients. Similarly, it has to

be noted that both the HTEW and PTSD samples from our study focused on CSA/CPA and that the results from our study may not readily generalize to other types of potentially traumatic events. Another limitation relates to differences in the PTSD and HTEW groups with respect to the severity of childhood trauma. As the focus of our study was on CSA/CPA, the PTSD and HTEW groups were matched for the respective subscales of the CTQ. While this matching was successful, post-hoc analyses revealed higher levels with respect to neglect and emotional abuse in the PTSD group which resulted in unbalanced CTQ total scores. This imbalance was addressed by repeating the analyses after including the CTQ total score as an independent predictor into the mixed models. As the models controlling for the potentially confounding effects of the CTQ total score essentially confirmed the results emerging from the study we are, however, confident in the robustness of our findings. Finally, the design of our study does not allow establishing causality. Longitudinal studies are needed to clarify which facets of stress level and stress response precede the experience of CA as predisposing factors that facilitate the development of PTSD after trauma, which are component parts of PTSD, and which are general sequelae of the traumatization itself and therefore not pathognomic for PTSD. Furthermore, according to previous studies by Rabbelino and colleagues (2017) and Berens and colleagues (2017), autonomic changes (i.e. impaired parasympathetic modulation of autonomic arousal, as indicated by reduced HRV) as well as risk for psychopathology were statistically mediated by volumetric changes in the amygdala, hippocampus, and prefrontal cortex. Furthermore, these alterations also appeared to be associated with altered activation of cortical and subcortical regions involved in the central autonomic network. Thus, it would be very interesting to examine whether differences in psychophysiological measures between HTEW, PTSD-patients and HC could be potentially accounted for by volumetric or functional differences in the respective brain regions.

In conclusion, results of the present study suggest that psychopathology in form of a PTSD diagnosis seemed to contribute to a more impaired stress response as indicated by high levels of subjective stress, dissociation, and HR as well as low HRV than experiences of CA per se. However, this finding does not exclude an additional role of CA as the data also indicate that a small proportion of altered stress response might relate to experiences of CA per se.

4 General Discussion

The aim of this dissertation is to examine the psychopathological and psychophysiological sequelae of childhood abuse in women without mental disorders in order to disentangle the effects of trauma and psychopathology. Thus, two studies have been conducted to focus on relevant psychopathological and psychophysiological aspects that are known to be impaired after experiencing CA and were also reported in patients with PTSD related to CA. These relevant aspects encompass PTSD-specific psychopathology such as intrusions and dissociation as well as general psychopathology, general functioning, employment status, quality of life, and satisfaction with sexuality. Further relevant aspects that were investigated in this dissertation encompass alterations in subjective and psychophysiological baseline stress levels as well as responsiveness to stress including pain sensitivity. These features were compared between healthy trauma-exposed women (HTEW) and a) healthy women without a history of CA and b) female patients with PTSD related to experiences of CA.

Individuals in the HTEW group showed neither increased general psychopathology nor PTSD-specific psychopathological symptoms in any of the collected measures in study I or II. They did not differ significantly in psychological well-being in comparison to HC either. Furthermore, individuals in the HTEW group showed a high level of functioning. Also, no statistical relevant restrictions in quality of life and sexual satisfaction were observed in comparison between HTEW and HC.

With regard to baseline stress levels, large effect-sizes separated HTEW from the PTSD group for subjective stress ratings, subjective dissociation ratings as well as heart rate and heart rate variability. A similar pattern was observed for subjective stress and dissociation levels in response to stress induction. In contrast, effect sizes between HTEW and HC were consistently small.

This general discussion provides, first, an integration of the two presented studies into previous research. Furthermore, potentially protective factors in the aftermaths of PTE will be considered. Methodological difficulties and limitations of the studies, general difficulties in research with HTEW, as well as possible implications for future research will also be discussed in the following.

4.1 Psychopathological Impairment

As pointed out in chapter 1, previous research has tried to answer the question whether intrusive memories differ between PTE-experienced participants with or without PTSD regarding frequency or distress. Adding to this, results of Study I showed that individuals in the HTEW group reported both significantly lower frequencies of intrusions and significantly less distress caused by the intrusions as compared to individuals in the PTSD group. These findings are only partially in line with results by Kleim and colleagues (2013) who were able to show that participants with PTSD reported only marginally more intrusive memories as compared to those without PTSD. However, significant differences between groups occurred when assessing the distress caused by the intrusions, with participants without PTSD showing less distress than PTSD-patients (Kleim et al., 2013). One possible explanation for the slightly divergent findings of Kleim and colleagues concerning the frequency of intrusions may concern methodological differences. Kleim and colleagues assessed intrusive memories in an ecological momentary approach using electronic diaries. Participants were asked to record every intrusive memory for seven consecutive days the moment the intrusion occurred. This approach seeks to prevent influences of recall bias or current mood when filling out the questionnaires. In contrast, Study I assessed intrusive memories retrospectively for the last week prior to study entry. However, besides the advantages of momentary assessment, it is possible that the findings of Kleim and colleagues are affected by reactivity (i.e. an increase of intrusion frequency due to repeated inquiries), as has been observed in previous momentary assessment studies (K. Priebe et al., 2013). Another limiting factor of the study by Kleim and colleagues lies in the fact that the authors examined participants with different experiences of PTE such as assault or motor vehicle accidents. According to Breslau and colleagues (2004), different forms of PTE (e.g., rape, motor vehicle accidents, combat, etc.) set off different conditional probabilities of developing PTSD as well as differential reactions in the aftermaths of trauma. Hence, one could assume that different PTE might cause differences in frequency or distress of consecutive intrusions.

Our finding that HTEW showed significantly less frequent and less distressing intrusions than PTSD-patients confirms previous research by Michael and colleagues (2005) who examined participants with experiences of physical or sexual assault

either with or without PTSD. In a retrospective assessment, participants with PTSD reported a significantly higher frequency of intrusions, a greater number of different intrusive experiences, as well as more intense intrusions as compared to participants without PTSD. In summary, results of the present study in synopsis with previous research indicate that having highly distressing and frequent intrusions seems to be a correlate of PTSD in the aftermaths of experiences of assault or CA rather than being related to a mere exposure to these threatening situations. This effect also appears to be stable over time. In a longitudinal follow-up study over a period of 20 years, Arnberg and colleagues (2011) showed constant levels of intrusions in survivors of a traumatic school bus accident with PTSD. Those who reported intrusions nine months after the accident also reported a comparable level of intrusions after four as well as after 20 years.

Women in the HTEW group did report having intrusions, independent of meeting diagnostic criteria of PTSD or any other mental disorder. However, these intrusions were not near as frequent or distressing as in the PTSD group.

In the studies presented in this dissertation, dissociation was assessed both under baseline conditions as well as in response to experimentally induced stress by means of the DSS-4 (Stiglmayr et al., 2009). As the DSS-4 is a reliable and easy-to-use tool to assess psychological as well as somatic aspects of dissociative states at multiple times during experimental investigations, it was possible to examine state dissociation of HTEW in comparison to PTSD-patients and HC throughout the experimental investigation. Additionally, the most commonly used self-rating questionnaire for the long-term assessment of trait dissociation, the Dissociative Experiences Scale (DES; E. M. Bernstein & Putnam, 1986) was used to assess trait dissociation of the respective groups. The DES assesses the frequency with which participants experience 28 dissociative phenomena in daily life. Values > 30 are thought to indicate potential dissociative psychopathology. With regard to mean DES scores, differences between HTEW and PTSD-patients showed large effect sizes while differences between HTEW and HC only approached significance. Although none of the groups scored higher than the clinical cut-off, participants in the PTSD group scored almost 4 times higher than participants in the HTEW sample, indicating an association of trait dissociation and PTSD rather than of trait dissociation and a mere effect of CA per se.

A similar pattern was observable with regard to state dissociative levels as assessed by the DSS-4. Under baseline conditions, HTEW showed significantly lower levels of dissociation as compared to PTSD-patients and did not differ significantly when compared to the levels of HC. However, in response to experimentally induced stress, the mean level of dissociation increased in the HTEW group as compared to the HC group. The increase was not as steep as in the PTSD-group, though. On a numerical level, dissociative levels of HTEW were still very low as compared to the levels of PTSD-patients after stress-induction. Taken together, results of the present studies add to a well-established body of evidence examining individuals with various types of PTE, with and without PTSD. After different forms of PTE (combat, CSA, and motor vehicle accidents) trauma-exposed individuals with PTSD exhibited higher levels of dissociation than trauma-exposed individuals without PTSD, in non-stress conditions as well as under stress (Bremner, Narayan, et al., 1999; Bremner, Staib, et al., 1999; Gómez-Pérez & López-Martínez, 2013; Lanius et al., 2002). These findings have been fully confirmed by the present study, since both the level of dissociation and the dissociative response to the stress induction were much more pronounced in the PTSD group than in the HTEW and HC groups. Consequently, these results are in line with a vast body of evidence that has suggested considering dissociation as a diagnostic criterion of PTSD in the DSM-5. Study II extends previous findings, as it indicates a significant, albeit small, difference between HTEW and HC in dissociation in reaction to a stress-induction paradigm. However, it has to be kept in mind that a rather low level of dissociation in HTEW might be related to the strict exclusion criteria in our study. In contrast to the studies by Bremner et al. (1999) and Lanius et al. (2002), trauma-exposed individuals without PTSD in our study did not meet criteria of any other lifetime diagnoses of mental disorder.

Taken together, dissociative experiences seem to be more closely related to PTSD than to trauma per se. According to Amir and colleagues (1998), successful emotional processing of traumatic events requires the creation of an organized, well-articulated and unfragmented narrative. In line with that, state dissociation during therapy sessions seems to have negative effects on trauma-focused therapy outcome (i.e., dialectical behavior therapy for PTSD), as shown in a study by Kleindienst and colleagues (2016), in patients with PTSD. On a neurobiological level, a hypoactive hippocampus (a region linked to memory reconsolidation), as well as hypoactive regions linked to emotion processing (e.g., amygdala, anterior cingulate

cortex, and insula) were associated with state dissociation in patients with a history of interpersonal trauma (Krause-Utz et al., 2012). Accordingly, the occurrence of dissociative symptoms may prevent the expression of trauma-related emotions and cognitions which in turn might lead to an insufficient processing of the trauma itself.

Furthermore, dissociation was assumed to have an important mediating role between CSA and various mental health issues in previous research (Kisiel & Lyons, 2001). More specifically, both a history of CSA as well as dissociation were independently associated with internalizing and externalizing problems as well as less competent social functioning. Although mediation analyses were not conducted in the studies presented in this dissertation, individuals in the HTEW group differed significantly from PTSD-patients. Women in the HTEW group showed very low levels of dissociation as well as low levels of psychological impairment (i.e., good self-esteem, good general functioning, hardly any PTSD-specific or psychopathologic symptoms). In contrast, women in the PTSD-group presented elevated levels of dissociation as well as higher levels of psychopathological impairment. As differences in severity of CA-experiences were minimized by matching procedures, one could possibly hypothesize that dissociation might have played a mediating role between trauma-exposure and various mental health issues.

Apart from PTSD-specific symptomatology, low levels of general functioning were also observed after experiences of PTE (Agorastos et al., 2013; Wolf & Martin, 2008). In Study I, general functioning was assessed by the overall psychopathology measures Global assessment of functioning scale (American Psychiatric Association, 2000) and Brief Symptom Inventory (BSI; Derogatis, 1993), as well as employment status and self-esteem. Overall, individuals in the HTEW group showed no subthreshold psychopathology nor did they exhibit unspecific restrictions in psychological well-being. They showed a significantly higher level of functioning and a significantly lower level of psychopathology as compared to PTSD-patients. Levels of HTEW were comparable to those levels in the HC group. However, this finding might also be related to the strict exclusion criteria for HTEW. Therefore, future research should also aim to investigate consequences of CA in a more naturalistic sample with varying degrees of psychopathology.

The reported findings extend previous research, showing significantly lower overall psychopathology in female veterans and undergraduate students with a history of CA

as compared to female veterans and undergraduate students without a history of CA (DiLillo, Lewis, & Loreto-Colgan, 2007; Surís, 2007). However, a major limitation lies in the fact that the authors did not assess whether participants met criteria of mental disorders. Therefore, differences between groups might also be attributed to present or absent current mental disorders.

With respect to disentangling the effects of mere trauma exposure versus a diagnosis of PTSD on general functioning, the present results are in line with a study on male combat veterans, showing significantly lower GAF scores in those with PTSD as compared to traumatized participants without PTSD (Agorastos et al., 2013).

In order to examine the potential mediating role of PTSD between traumatic experiences and an impairment in general functioning, Wolf and colleagues (2008) used structural equation modeling. The assessment of a large group of veterans showed that the association between combat exposure and good general functioning was fully mediated by PTSD severity.

In the present studies, though participants were matched with regard to their educational level, individuals in the HTEW group more often reported a full-time working status as compared to individuals in the PTSD-group. The full-time working status of HTEW was comparable to that of HC. This finding is in line with results of various studies (e.g., Shah et al., 2013; M. B. Stein et al., 1997; Webb, Vincent, Jin, & Pollack, 2015) on participants from all kinds of trauma categories other than CA. In these studies, participants with PTSD also showed significantly higher rates of unemployment as well as significantly lower rates of full-time working status as compared to traumatized participants without PTSD (or partial PTSD), and non-traumatized HC. However, to the best of my knowledge, this study is the first in assessing academic functioning and employment status in HTEW with a history of CA in comparison to both HC and PTSD-patients. Previous research on academic functioning did not separate between those individuals with a history of CA that either did or did not meet diagnostic criteria of PTSD or other mental disorders. Thus, data from Study I extends and partly contradicts previous knowledge on the detrimental effects of CA on academic functioning (for an overview see meta-analyses of Paolucci, Genuis, & Violato, 2001), indicating that this specific participant group of HTEW seemed to withstand the adversities unscathed.

4.2 Quality of Life and Satisfaction with Sexuality

As elaborated in the introduction section, previous research suggests that QoL might serve as a key indicator of health and well-being. Results of Study I support this assumption. Individuals in the HTEW group who were healthy and free of mental disorders, reported significantly higher QoL levels as compared to PTSD-patients, and showed QoL levels that were comparable to those of HC participants. In the HTEW group, no statistically relevant restrictions in QoI were observed by any of the three QoL measures in comparison to HC participants. Results of the present study therefore extend previous findings by demonstrating an association of experiences of CA and affected QoL. More specifically, previous studies showed significantly lower levels of QoL in female participants with a history of CA in comparison to female participants without a history of CA (Griffin & Amodeo, 2010; A. J. Lang et al., 2004; Surís, 2007). To the best of my knowledge, participants in the respective studies encompassed female veterans, female victims of intimate partner violence, and US community women who were not assessed with regard to mental disorders. Therefore, study I expands upon these findings by examining participants with a history of CA either with PTSD or without any axis-I disorders nor BPD in order to disentangle the effects of PTSD versus CA-exposure per se on QoL.

Results of study I are in in line with a study by Cloitre and colleagues (2001), who examined the relative contributions of PTSD versus trauma exposure on current subjectively perceived physical health-status in women with a history of CA with or without PTSD. In this study, women with CA-related PTSD showed significantly lower physical well-being as compared to women with experiences of CA without PTSD. As described in chapter 1, physical well-being is an integral part of QoL and seems to be influenced by psychological factors. Accordingly, the authors conclude that PTSD might be a better predictor of lower QoL levels including physical well-being than trauma exposure per se. However, as a limiting factor, the authors did not control for mental disorders other than PTSD, which might contribute to ramifications of results by other mental disorders (e.g., depression).

Further results indicating that PTSD might be a better predictor of impaired QoL than trauma exposure per se were published by Gill and colleagues (2012), who examined women with assault either with PTSD or after recovery from PTSD, and HC. Interestingly, women who had recovered from PTSD had similar QoL scores as

women without assaultive traumatic experiences. This suggests that recovery on a psychological level might be associated with QoL levels comparable to those of non-traumatized people. However, the finding by Gill and colleagues is not specific for experiences of CA, as assaultive trauma did not necessarily imply that the assault had taken place in childhood or adolescence.

Furthermore, as already pointed out in the introduction, sexual well-being and sexual satisfaction as well as an active and positive sex life seem to have beneficial implications for psychological well-being and overall QoL. It is also known that experiences of CA and especially CSA may have detrimental effects on sexuality and are often associated with sexual dissatisfaction in adulthood (for an overview see Leonard & Follette, 2002; O'driscoll & Flanagan, 2016). Yet, the influence of CA exposure per se against the influence of CA-related PTSD, the most marked mental disorder following CSA (Cutajar et al., 2010), remained unclear. Consequently, the present study aimed to offer new insights with regard to disentangling the effects of mere CSA-exposure per se against correlates of mental disorders on sexual satisfaction and sexual engagement. For this purpose, HTEW with a history of CA were compared with both CA-related PTSD-patients and non-traumatized HC.

When examining the frequency of sexual activities, individuals in the HTEW group participated in sexual activities significantly more often than PTSD-patients. In comparison to non-traumatized HC, HTEW scored on a comparable level, indicating regular sexual activity. This finding is in line with previous studies, indicating that women with more active sexual life report better well-being (Laumann et al., 2006). Finally, intact sexuality and heightened sense of well-being potentially contributes to improved subjective health, which in turn can be mirrored in elevated QoL scores (for review see Rosen & Bachmann, 2008).

Results of the present study extend previous studies in concluding that frequency of sexual contacts is lower in women with experiences of CSA (Vaillancourt-Morel et al., 2015; Van Berlo, 2000). In the present study, we were able to present a more detailed picture by controlling for mental disorders, indicating that not the experience of CSA per se reduces the frequency of sexual contacts, but rather the occurrence of PTSD or other forms of psychopathology in the sequel of CSA.

When examining sexual satisfaction, a pattern that was very similar to the results on frequency in sexual activity occurred. Here, HTEW were significantly more satisfied

with their sexuality as compared to PTSD-patients, and scored on a highly satisfied level, comparable to that of HC. Our results expand upon the existing literature showing an association of experiences of CA with reduced sexual satisfaction (DiLillo et al., 2007; Leonard et al., 2008; Lutfey, Link, Litman, Rosen, & McKinlay, 2008; Öberg et al., 2002; Stephenson et al., 2014), but without controlling for psychopathology. Results of the present study also extend previous literature, pointing to a moderating role of PTSD (OR of 2.3 as compared to traumatized participants without PTSD) on satisfaction with sexuality in participants with experiences of PTE other than CA (Arbanas, 2010; Letourneau et al., 1996; Tran et al., 2015).

Ultimately, results on frequency and satisfaction with sexuality after experiences of CA are difficult to interpret. Potential reasons for that encompass the vast heterogeneity of individuals that are grouped under the term CSA, the diversity of sexual problems that are studied, as well as insufficient diagnostic assessment. In response to these methodological difficulties, the present study expands upon the existing literature, as only participants with a history of CSA and/or CPA were examined, satisfaction with sexuality was examined by means of validated questionnaires, and potentially mediating effects of mental disorders were taken into account.

Results of this present study suggest that impairment in sexual well-being might not be associated with experiences of CA per se, but rather seems to be related to psychological problems including PTSD. When looking more closely at PTSD symptomatology according to DSM-5, a connection between PTSD and impaired sexual functioning seems very likely. More specifically, intrusive symptoms might interfere with sexuality, since trauma-related stimuli may potentially evoke mismatching aversive emotions (i.e. shame and guilt), potential painful body-related intrusions might interfere with sexual functioning, and sexual situations themselves might elicit intrusions that could lead to avoidance behavior (Büttner, Dulz, Sachsse, Overkamp, & Sack, 2014; D'Andrea, Sharma, Zelechoski, & Spinazzola, 2011; Steil & Ehlers, 2000). Furthermore, in CSA-exposed individuals, sexual contact can be seen as a strong contextual trigger eliciting high emotional arousal, which in turn facilitates dissociative reactions (Schauer & Elbert, 2010; D. J. Stein et al., 2013). Both intrusive experiences as well as dissociation during sex are likely to contribute to sexual dissatisfaction as well as reduced frequency of sexual contacts.

Furthermore, the present results support the assumption that sexual satisfaction seems to be associated with psychological well-being and good overall QoL, as both measures correlate positively. However, due to the cross-sectional design, no cause and effect assumptions can be concluded as to whether good QoL results in sexual satisfaction or whether sexual satisfaction contributes to good QoL.

Interestingly, with regard to the treatment of sexual problems, previous research suggests that sexual problems are often distinct from psychopathology (Classen et al., 2011; Rieckert & Möller, 2000) and may require separate clinical focus. Although most of the proposed mechanisms through which experiences of CA may affect sexual functioning seem to be at the psychological level (e.g. through intrusions, dissociation, depression) (Büttner et al., 2014; Haase, Boos, Schönfeld, & Hoyer, 2009; Laurent & Simons, 2009; A. Michael & O'Keane, 2000; Polusny & Follette, 1995; D. J. Stein et al., 2013), physiological problems might also contribute to an impairment of sexuality. More specifically, previous studies have shown that in sexually healthy women, moderately increased SNS activity facilitates genital sexual arousal, whereas high SNS activation inhibits it (Lorenz, Harte, Hamilton, & Meston, 2012). Furthermore, SNS activation as induced by exercise seems to increase genital arousal in sexually healthy women, but not among women with a history of CSA (Rellini & Meston, 2006). According to the same study, women affected by experiences of CSA already show high baseline SNS. Additional activation on top of the already elevated SNS activity might push the stress system beyond a level which facilitates sexual arousal. Results of a study by Meston and colleagues (2013) support this assumption. They show that in women with a history of CSA, lower SNS activity was associated with higher sexual satisfaction. However, this effect was more pronounced for participants with low lifetime trauma as compared to participants with high lifetime trauma (e.g., chronic abuse). Results of study II, showing lower SNS activity assessed by HR levels at baseline as well as in response to stress in HTEW as compared to PTSD-patients, support this thesis. However, no effect of high versus low lifetime trauma was observable, as differences in trauma severity were minimized by means of matching procedures.

In summary, findings of Study I add to the existing literature by supporting the notion that QoL as well as sexual well-being might not be predominantly influenced by the experience of CA per se, but rather by the development of a consequential PTSD diagnosis of other mental disorders.

4.3 Psychophysiological Impairment and Altered Stress Responsiveness

As pointed out in chapter 1, abnormalities in stress levels and stress responsiveness as well as alterations on a broad spectrum of variables related to stress have been found in numerous studies on patients with PTSD and in CA-exposed individuals (Buckley & Kaloupek, 2001; Fillingim & Edwards, 2005; Pierrehumbert et al., 2009). In this dissertation, study II was conducted in order to examine whether CA-exposed HTEW, although not meeting diagnostic criteria of PTSD or other mental disorders, would also show elevated stress levels and impaired stress responsiveness as well as alterations on variables related to stress, both on a subjective and psychophysiological level. The purpose of study II was to fill the research gaps on the impact of CA-exposure on subjective and psychophysiological stress levels, stress responsiveness, and pain sensitivity, as the existing literature on CA-exposed individuals is scarce, or has limitations with regard to disentangling the effects of trauma and PTSD.

On a subjective level, stress ratings of HTEW were significantly higher than in HC and significantly lower than in women with a diagnosis of PTSD. Effect sizes between HTEW and PTSD groups were particularly large. A general increase of subjective stress in response to experimentally induced stress was observed, confirming that the stress induction procedure had shown the intended effects. However, HTEW did not differ significantly from HC, but showed lower increases when compared to the PTSD group. This significant difference between HTEW and PTSD-patients is in line with previous research by Regehr and colleagues (2007) and Pineles and colleagues (2013), assessing individuals with a history of PTE other than CA. In the study by Regehr and colleagues, police recruits with PTSD symptomatology showed higher levels of subjective stress in confrontation with work-related critical events as compared to police recruits without PTSD symptomatology. Pineles and colleagues analyzed a combined dataset of five studies using script-driven imagery on individuals with mixed PTE, showing that individuals with PTSD rated exposure to trauma-related or other stressful scripts as more unpleasant and distressing than individuals without PTSD. Therefore, results of the present study in synopsis with previous research on different trauma categories suggest a rather strong association between PTSD and altered stress responses on a subjective level, but not trauma

exposure per se. This conclusion argues against a mere effect of CA on altered subjective stress response. Accordingly, this conclusion is promoted by Carpenter and colleagues (2007) who showed comparable subjective stress ratings in response to experimentally induced stress in healthy individuals without diagnosable psychopathology, who either did or did not experience CA.

On a psychophysiological level, throughout the observation period of study II, baseline levels of HR recorded in the HTEW group were located numerically between those of HC and PTSD. Women in the HTEW group showed significantly higher HR levels than women in the HC group, and significantly lower HR levels than women in the PTSD group. As age and gender might be factors influencing HR and HRV, with overall reductions in the autonomic control of the heart with increase in age and women showing greater vagal tone than men (Abhishekh et al., 2013), all three participant groups comprised only female participants who were matched with regard to age. Results of the present study partly confirm results of previous research on participants with PTE experiences other than CA. In a meta-analysis by Buckley and Kaloupek (2001), traumatized individuals without PTSD (mostly male veterans) also showed significantly lower basal HR levels than PTSD-patients. However, traumatized and non-traumatized HC did not differ in basal HR levels. Whereas previous research on other PTE experiences (e.g., war, torture, motor-vehicle accidents, interpersonal violence, and combat exposure) suggests that a PTSD diagnosis but not trauma exposure per se does affect basal HR levels (e.g., Buckley & Kaloupek, 2001; Goodman & Griffin, 2018; Peri, Ben-Shakhar, Orr, & Shalev, 2000), results of the present study point to an additive effect of CA experiences on elevated basal HR levels. Although differences between HTEW and PTSD-patients were more pronounced than differences between HTEW and HC, we still observed significant differences between HTEW and HC.

With respect to stress responsiveness, an overall increase in HR but no interaction effects were observed in response to experimentally induced stress: The increase in HR in the HTEW group in response to the stress induction by means of the MMST did not differ significantly from the two other groups. The null findings of the present study are in line with findings by Goméz-Pérez (2013) showing no differences between trauma exposed individuals with and without PTSD and non-traumatized HC in HR in response to a cold pressor task. However, the present findings are not in line with previous research showing a faster fight/flight response in those with PTSD,

as indicated by steeper HR increases to aversive stimuli as compared to PTE-experienced participants without PTSD in combat veterans (Costanzo et al., 2016; Orr et al., 2003), victims of war and torture (Adenauer et al., 2010) and in participants with other kinds of PTE (Peri et al., 2000). Additionally, in the study by Peri and colleagues, war and torture-experienced participants without PTSD showed an indiscriminate orienting response to all stimuli, irrespective of the valence, exhibiting a permanent alertness.

To sum up, previous research to disentangle the effects of trauma and PTSD is scarce, partly inconsistent, and mostly conducted with traumatized individuals with a history other than CA. On the one hand, results by Goméz-Pérez on individuals with mixed trauma types and study II on CA-exposed individuals suggest no differences between groups in HR response to experimentally induced stress, indicating that neither trauma-exposure per se nor PTSD has an effect on stress-responsiveness. On the other hand, results by Adenauer and Orr point out that impaired HR responses to experimentally induced stress represent an acquired sign of combat, war and torture-related PTSD. Findings by Peri and colleagues further suggest a small effect of war and torture experiences on impaired HR stress responses irrespective of PTSD.

Reasons for these conflicting findings may be ascribed to the usage of different stress paradigms. Stress induction in the studies by Peri, Adenauer and Orr and colleagues encompassed a combination of visual/emotional and auditory aversive stimuli and the cold-pressor task in the Goméz-Pérez study. In contrast, stress induction in the present study encompassed a combination of cognitive, emotional, acoustic, and motivational stressors, therefore including further qualities of stress. Furthermore, the respective studies examined different trauma types: while PTE-exposed participants in the studies by Peri, Orr and Adenauer and colleagues all experienced trauma later in life in adulthood, all PTE-experienced participants in study II were affected by CA in childhood and adolescence. Hypothetically, this difference in age at the time of the traumatic experience might also account for divergent findings in psychophysiological stress-responsiveness.

With respect to HRV, the baseline level of HRV in the HTEW group was significantly lower than in the HC group, and significantly higher than in the PTSD group. These findings are partly in line with results of a systematic review by Sammito and

colleagues (2015) and a meta-analysis by Chalmers and colleagues (2014), showing reduced HRV in patients with PTSD related to all kinds of PTE, as compared to both trauma and non-trauma exposed healthy controls not meeting diagnostic criteria of PTSD. PTE in the respective studies encompassed interpersonal violence (Hauschildt et al., 2011), combat (Wahbeh & Oken, 2013), mixed PTE (Lee & Theus, 2012), combat plus military sexual trauma (Chang et al., 2013) and experiences as fire service workers (Mitani, Fujita, Sakamoto, & Shirakawa, 2006). However, two other studies did not find significant differences in baseline HRV levels between these groups as indicated by null findings in combat-exposed individuals (Agorastos et al., 2013) as well as in a mixed PTE group (Keary, Hughes, & Palmieri, 2009). Therefore, results of the present study are only partly in line with previous research. In CA-exposed individuals, those meeting diagnostic criteria of PTSD showed lower HRV baseline levels than those not meeting PTSD. This pattern has also been observed in various studies on PTE-experienced individuals other than CA, suggesting that reduced baseline HRV levels are a sign of PTSD, rather than trauma exposure per se. However, differences between previous research and the present study become apparent with regard to the comparison of HTEW and HC. While previous research either did not assess an additional HC group or showed similar HRV baseline levels of PTE-exposed individuals without PTSD and HC, HTEW in the present study showed significantly lower baseline HRV levels as compared to HC. As already described for basal HR levels, this finding suggests an additional effect of CA-exposure beyond the impact of a PTSD diagnosis. Interestingly, previous studies showed that vagal modulation of heart rate appears to be sensitive to recent experiences of stress, with lower vagally mediated HRV among healthy women who perceived more stress, as compared to healthy women who experienced less stress (Dishman et al., 2000; Miu, Heilman, & Miclea, 2009). According to these findings, one could assume that in study II, participants with similar subjective stress ratings would also show similar HRV levels. However, individuals in the HTEW and HC groups did not differ with respect to subjective stress ratings, but did differ with respect to HR and HRV levels, with HTEW showing elevated HR and reduced HRV as compared to HC. This might add to the assumption that CA-exposure might have an additional effect on psychophysiological functioning beyond the impact of a PTSD diagnosis that is independent of the subjective evaluation of stress.

With respect to HRV after stress induction by means of the MMST, an overall decrease in HRV was observed in response to experimentally induced stress. However, no interaction effects were observed, indicating that the decreases in HRV in the HTEW group did not differ significantly from the two other groups. This finding adds to a heterogeneous body of literature addressing cardiac responses to stressful tasks. While three studies failed to identify differential effects of affective cues or stressful tasks on HRV in PTSD as compared to both traumatized and non-traumatized HC (Cohen et al., 2000; Cohen et al., 1998; Hauschildt et al., 2011), two studies reported a greater decrease of HRV in response to stressful tasks such as trauma script exposures in PTSD-patients, as compared to traumatized controls without PTSD (Keary et al., 2009; Norte et al., 2013).

In a study by Dennis and colleagues (2016), elevated HR levels and reduced HRV levels after high levels of distress were only found in participants with high levels in PTSD symptomatology as assessed by the DTS, not in participants with low levels of PTSD symptomatology. These results suggest that interindividual differences in severity of PTSD symptomatology are associated with distinct autonomic responses to temporary distress rather than generalized patterns of autonomic activation in PTSD. However, it is important to mention that Dennis and colleagues, in contrast to the studies by Cohen, Hauschildt, Keary and Norte, only assessed PTSD symptomatology, not a PTSD diagnosis.

Accordingly, to the present day, there is no consensus on whether impaired stress responsiveness in HRV seems to be a correlate of PTSD or in fact to trauma exposure per se. When thinking about reasons why an individual might react more strongly to experimentally induced stress as indicated by elevated HR and reduced HRV levels, however, one might think of an impaired processing of safety signals. Previous research has assessed the processing of safety signals in traumatized individuals and has found significant impairment in those individuals with PTSD in comparison to both traumatized and non-traumatized controls without PTSD (Jenewein et al., 2016; Thome et al., 2017). Although patients with PTSD might be cognitively aware that they are safe in the laboratory setting, they still show increased physiologically responses (for review see Jovanovic, Kazama, Bachevalier, & Davis, 2012). These results suggest that PTSD-patients respond similarly to laboratory reminders of trauma or stress-evoking tasks as they do to danger situations in real life. Furthermore, according to Norte and colleagues (2013), PTSD-patients seem to

have difficulties in exhibiting physiological recovery after exposure to distressing stimuli, which might be due to a deficient vagal control.

To sum up, results of the present study suggest that psychopathology in form of a PTSD diagnosis above experiences of CA per se seem to contribute to more impaired basal stress levels as indicated by high levels of subjective stress and HR as well as low HRV. However, this finding does not exclude an independent effect of CA, as the data also indicates that a small proportion of altered stress response might relate to experiences of CA per se.

Potential causal mechanisms of this additional role of CA can be discussed. Among other mechanisms, experiences of CA may be associated with alterations in oxytocin brain systems affecting stress responses (Heim, 2009). According to Heim and colleagues (2000), oxytocin attenuates stress response, and these stress-protective effects of oxytocin seem to result from decreased amygdala activity (Kirsch et al., 2005). Besides functional alterations, a recent study by Rabellino and colleagues (2017) examines the neural underpinnings associated with autonomic changes in PTSD. According to the authors, impaired parasympathetic modulation of autonomic arousal, as indicated by reduced HRV, appears to be associated with altered activation of cortical and subcortical regions involved in the central autonomic network. Thus, it would be very interesting to examine whether differences in psychophysiological measures between HTEW, PTSD-patients and HC could be potentially accounted for by volumetric or functional differences in the respective brain regions.

As a clinical implication of study II, one could think of secondary preventive programs in form of HR and HRV biofeedback (Tan et al., 2011) for CA-exposed individuals. Even though CA-exposed individuals might not fulfill diagnostic criteria of PTSD or other axis-I disorders, they might still be at risk of atherosclerotic buildup and coronary artery disease, increased irritability, or sleeping difficulties as a consequence to sustained elevated HR levels as a function of chronic stress. Finally, future studies could also disentangle the effects of PTSD versus other mental disorders (e.g., depression) and examine differences between sexes, as research suggests that, e.g., women report greater subjective and physiological stress responses compared to men (Kudielka, Hellhammer, & Kirschbaum, 2000).

As pointed out in the theoretical background chapter, pain sensitivity constitutes another stress-related variable that seems to be impaired as a consequence to early stressors. With regard to studies at the psychophysiological level, research on trying to disentangle the effects of CA and PTSD is scarce and with significant limitations. The aim of study II was to fill this gap. In this respective study, baseline levels of pain sensitivity in the HTEW group were significantly lower than in the HC group and tended to be higher than in the PTSD group. Results of the present study are partly in line with previous research. As in the study by Goméz-Pérez (2013), HTEW and PTSD-patients with a history of all kinds of traumatic events did not differ significantly concerning their pain threshold levels, but HTEW did differ significantly from HC with regard to pain sensitivity. The difference between the present study and the study by Goméz-Pérez constitutes the direction of alterations between HTEW and HC as well as the assessed component of pain: In the present study, HTEW showed significantly lower pain sensitivity levels as assessed by subjective ratings than HC, whereas in the Goméz-Pérez study HTEW showed significantly higher pain sensitivity levels, as assessed by pain thresholds, than HC. Although the direction of alteration may be different, both studies suggest that exposure to trauma itself, not PTSD, may be associated with alterations in pain sensitivity. This conclusion is also underpinned by Tsur and colleagues (2017) showing a strong correlation of increased pain sensitivity and trauma exposure in torture survivors with and without PTSD.

Our findings are inconsistent with findings by Kraus and colleagues (2009) showing lower subjective pain sensitivity ratings in combat-exposed individuals with PTSD as compared to both combat-exposed and non-combat-exposed HC. Results by Kraus and colleagues suggest that PTSD rather than trauma-exposure itself is associated with alterations in pain sensitivity and more specifically with decreased pain sensitivity. In a longitudinal study spanning 17 years in a three-wave design to assess PTSD after torture experiences, pain thresholds of torture survivors with chronic PTSD, delayed PTSD, resilient survivors, as well as HC were studied at the last assessment within the study. Pain thresholds were similar across all four groups indicating that neither the trauma exposure per se (i.e., torture and captivity) nor PTSD were associated with changes in pain sensitivity (Defrin, Lahav, & Solomon, 2017). To sum up, the existing literature depicts a rather inconsistent picture with regard to alterations in pain sensitivity, either subjective ratings or pain thresholds, at baseline following PTE and PTSD. The question as to why and how the experience

of trauma or PTSD ultimately results in altered pain sensitivity still remains unanswered (Beck & Clapp, 2011).

Possible factors contributing to differences in stress levels might be the usage of different pain induction paradigms (i.e., heat pain stimuli, cold pressor task) and different dependent variables (pain threshold versus subjective appraisal of pain). Another possible explanation for the divergent findings on pain sensitivity in PTSD could be that participants with different types of PTE have been studied. With respect to studies showing SIH, one potential explanation might be the component of fear. Higher fear levels during experimentally induced stress might lead to an overestimation of painfulness of pain stimuli, as seen in Rhudy and colleagues (2000). As stated by Bekrater-Bodmann and colleagues (2015), significantly lower pain perception (pain stimuli induced by a thermal grill illusion) was observed in participants with a more severe history of CPA. Additionally, higher thermal pain thresholds were significantly related to a more severe history of sexual abuse. Other forms of CA (e.g., emotional abuse) were not associated with alterations in pain perception. These findings elucidate that experiences of CSA and CPA seem to cause more grave sequelae in pain sensitivity than other forms of CA. Contradicting these findings, analyses by Pieritz and colleagues (2015) indicate that emotional abuse but none of the other forms of abuse as assessed by the CTQ, were significantly related to alterations in pain sensitivity. A potential underlying mechanism responsible for these effects might be traced back to an altered neurotransmission in the sequel of CSA or CPA. Previous studies have shown that early-life stress affects N-methyl-D-aspartate (NMDA)-modulated neuroplasticity which might contribute to altered pain perception (for further information see Bekrater-Bodmann et al., 2015).

In accordance with the findings by Bekrater-Bodmann and colleagues, inclusion criteria of the present study encompassed a history of CSA or CPA for HTEW and PTSD-patients. To control for effects of trauma-severity, trauma-exposed individuals of both groups were matched with regard to the CTQ subscales of Sexual and Physical Abuse.

Further potential explanations for altered pain sensitivity come from Tsur and colleagues (2018) suggesting that trauma exposure and particularly the pathological reaction to trauma (i.e., PTSD) are associated with catastrophic and frightful

orientation to body signals, mostly driven by the symptom cluster of hyperarousal. Such a catastrophic orientation towards body signals has also been found in individuals with a history of emotional or physical abuse during childhood (Pieritz et al., 2015). These results suggest that past experiences (i.e., physical abuse) shape how an individual orients to and experiences his or her body (Sullivan, 2012). Finally, another possible explanation for impaired pain sensitivity in PTSD-patients might be that fear learning deficits prevent from safety learning (Jenewein et al., 2016). However, conceivable ramifications of these processes encompass either elevated pain sensitivity due to this catastrophic and frightful orientation or impaired fear learning. Another ramification might be decreased pain sensitivity, as the stress evoked by the catastrophic and frightful orientation towards body signals may cause dissociative reactions reducing pain sensitivity (Defrin, Schreiber, & Ginzburg, 2015; Ludäscher et al., 2009).

In response to experimentally induced stress, no significant difference between the HTEW and HC group was detected. However, the predefined contrast comparing the slopes in the HTEW and PTSD groups indicated a steeper decline for the pain ratings in the PTSD group in response to experimentally induced stress. This finding indicates a stronger effect of PTSD than of trauma exposure per se on altered pain sensitivity in response to stress. This is in line with findings by Pitman and colleagues (1990) showing reduced pain-sensitivity in veterans with PTSD as compared to veterans without PTSD in response to experimentally induced stress.

To sum up, pain sensitivity at baseline as well as in response to experimentally induced stress seems to be modulated by both PTSD and, although to a lesser extent, CA-exposure per se. Results of the present study suggest a decrease in pain sensitivity in the sequel of CA-experiences. While an overestimation of painfulness may restrict individuals in their way of living, an underestimation (i.e., reduced pain sensitivity) can lead to maladaptive behaviors such as enduring situations that harm the body. Accordingly, another goal of treating traumatized individuals either with or without PTSD could be to achieve normalization in pain sensitivity.

4.4 Potentially Protective Factors

The importance of studying potentially protective factors that might help to attenuate development of psychopathology in the aftermaths of trauma is beyond any doubt. Based on the systematic reviews mentioned in chapter 1, it seems likely that the

factors education, external attributions of responsibility and guilt, self-esteem as well as intra- and extra-family support are important factors in maintaining psychological health in the face of adversities. As this dissertation was not primarily focused on potentially protective factors, only a few of these factors were examined.

In the two studies, education was assessed as years of education at school. To prevent the results from being confounded by differences in education, all three groups were matched with regard to their educational level. Accordingly, this design is not suitable for examining the influence of education as a protective factor, but can give some guidance for matching procedures in future research.

4.4.1 The role of self-esteem and guilt

When considering factors that might attenuate the impact of experiences of CA, some researchers have considered self-esteem to be one of the psychosocial factors through which these adverse experiences may affect mental health (Soler et al., 2013). Results of Study I showed that women in the HTEW group and HC group differed significantly from women in the PTSD group in self-esteem as indicated by higher scores on the Rosenberg Self-Esteem Scale. This is in line with studies by Bungert and colleagues (2015) and Shirk and colleagues (2003) suggesting that low levels of self-esteem are correlated with psychopathology.

When comparing the two mentally healthy groups, self-esteem scores of women in the HTEW group did neither differ statistically nor numerically from women in the HC group. This finding contradicts the assumption that individuals with a history of CA generally show low levels of self-esteem (Chan et al., 2011; Turner et al., 2010). Based on the results of a cross-sectional study with Chinese teenagers, Chan and colleagues (2011) found an association between low self-esteem scores as assessed by the Rosenberg Self-Esteem Scale and a history of childhood abuse. Conforming to this are results of a three-wave longitudinal study over a period of two years by Turner and colleagues (2010). In that study, 523 participants were followed up to examine the effects of child victimization on self-concept and self-esteem as assessed by a modified version of the Rosenberg Self-Esteem Scale. Results suggest that experiences of CSA may have powerful damaging effects on self-esteem, which emerge relatively quickly after the victimization occurs. However, what limits these findings is the fact that both Chan and colleagues as well as Turner and colleagues did not control for mental disorders.

With regard to attributions of responsibility after trauma, previous research suggests that guilt seems to be positively associated with PTSD-symptomatology after all kinds of PTE, and may influence recovery from mental health problems (Beck et al., 2011; Clifton, Feeny, & Zoellner, 2017; Oktedalen, Hoffart, & Langkaas, 2015). According to Kubany and Manke (1995), PTSD might be maintained through guilt cognitions that continuously revive the traumatic memories. In participants with experiences of CA, previous research also found correlations between guilt cognitions and self-blame and poor adjustment and pathology (Aakvaag et al., 2016; Ginzburg et al., 2009; McMillen & Zuravin, 1997).

Results presented in this thesis could support this assumption: Women in the HTEW group, who showed good general functioning and hardly any psychopathology, showed significantly less guilt cognitions and significantly less distress caused by guilt cognitions as compared to women in the PTSD group. This is in line with a study by Wolfe and colleagues (1994). To the best of my knowledge, to date, only this study by Wolfe and colleagues assessed guilt cognitions of sexually abused teenagers, separated into groups according to current or absent PTSD diagnosis. In the study in question, sexually abused teenagers with PTSD showed significantly higher guilt regarding the abuse than sexually abused teenagers without PTSD. Hierarchical multiple regression analyses indicated that the child's self-reported guilt feelings explained up to 37% of the variance in PTSD symptomatology. However, PTSD was assessed by means of a PTSD related symptom checklist, which might have diminished the validity of the PTSD diagnosis.

Overall, previous research as well as results of the present studies suggests that good self-esteem as well as absence of guilt cognitions after experiences of CA seem to be associated with withstanding adversities unscathed. Whether low self-esteem or feelings of guilt precede mental health symptoms after trauma, or whether mental health symptoms leave individuals prone to feeling guilty or having low self-esteem has not been examined in detail yet. Longitudinal studies controlling for mental disorders and experiences of CA are needed in order to understand the directionality of the associations.

4.4.2 The importance of disclosing traumatic experiences and subsequent social reactions

As already stressed in the introductory section, the victim's disclosure as well as social reactions and social acknowledgement as a victim seem to play a role in the development of psychopathology in the aftermaths of PTE (Maercker & Mehr, 2006; Maercker & Müller, 2004; Mueller et al., 2008). According to a representative study with 804 participants in Canada (Hebert, Tourigny, Cyr, McDuff, & Joly, 2009), only 21% of CSA-exposed participants reported prompt disclosure and 58% disclosed with considerable delay. Those individuals who never disclosed and those with delayed disclosure were more likely to show psychopathological impairment at a clinical level.

In the studies in this dissertation, data on disclosure was assessed in a non-structured screening interview by asking whether participants had told somebody about the abuse and what kind of reactions they had received. Unfortunately, data on disclosing was not assessed in the PTSD-patients' sample. Accordingly, results of HTEW of the respective study can only be compared to results of previous studies on disclosure in PTSD-patients. In the HTEW sample, all but one woman had disclosed the experiences of CA, and they reported positive experiences in doing so. Experiences of disclosing were described as "liberating and relieving" and as "not being alone with it anymore and being supported in coping with it". Opposed to this, studies in patients with PTSD conclude that disclosure and subsequent social reactions seem to be a predictor of PTSD (Chris R Brewin, Andrews, & Valentine, 2000; Maercker et al., 2009; Ozer et al., 2008). Individuals reporting lower levels of perceived positive social reactions after the traumatic event reported higher levels of PTSD and vice versa. In a longitudinal study by Ullman & Relyea (2016), over a period of three years, posttraumatic stress symptoms in female sexual assault survivors were examined depending on social reactions received from others when disclosing assault. The authors showed that negative reactions were associated with more posttraumatic stress symptoms. Interestingly, the one woman in the present study who did not disclose her experiences before study entry was born and raised in the Philippines and immigrated to Germany only in her mid-20s. As trauma victims in traditional groups are at higher risk to feel socially excluded or even blamed for what happened when disclosing (for an overview see Herbert & Dunkel-Schetter, 1992; Ullman, 2007), being raised in a traditional setting might have been a reason for her

decision not to disclose. However, this is a correlative assumption and does not imply causation.

The present results also back the hypothesis that forming a narrative about what happened could possibly lead to recovery. According to cognitive theory for PTSD, (as in Ehlers, Clark, Hackmann, McManus, & Fennell, 2005) the process of exposing oneself to the traumatic memories by retelling what had happened might lead to an integration of these memories in the autobiographical memory. This process of disclosing and retelling the traumatic memories is key in treating PTSD in cognitive behavioral therapy, Narrative Exposure Therapy (Schauer, Schauer, Neuner, & Elbert, 2011) or testimony therapy (van Dijk, Schoutrop, & Spinhoven, 2003). Future studies could focus more strongly on the process of disclosing as well as social reactions to this process (e.g., by means of qualitative interviews) to further examine helpful reactions to disclosure.

4.5 Methodological Limitations

Despite several strengths, a number of limitations of the respective studies as well as general difficulties in doing research with HTEW need to be considered. First, both studies were realized with female participants. Although lifetime prevalence of traumatic events is slightly higher in men than in women (Hatch & Dohrenwend, 2007), the risk of developing PTSD following PTE is two times higher for women as compared to men (Breslau, 2001). This increased risk of developing PTSD can be traced back to the types of PTE. Women are much more likely to experience abuse including sexual assault (Christiansen & Elklit, 2012) which implies the highest probability of developing PTSD in the aftermaths of PTE (Kessler et al., 2017; Kessler et al., 1995; McCutcheon et al., 2010). As only women were included in the above-mentioned studies, heterogeneity due to possible gender differences in PTSD was reduced (Ullman & Filipas, 2005), but gender generalizability is yet to be shown.

Second, data on experiences of CA were obtained by retrospective self-report. There is conflicting evidence concerning the validity of self-reported experiences of PTE. On the one hand, previous research showed immense underreporting of CA experiences (Belli, 2014; L. M. Williams, 1994) in retrospective self-report. On the other hand, there has also been the opposite effect with over reporting of experiences of CA in retrospective self-report (Finkelhor, 2008). Therefore, it would

be interesting to conduct further studies with participants with official records of CA to see whether those reports differ from the ones with self-reported CA.

A third limitation relates to the representative state of our HTEW sample and generalizability of the results. Participants in the HTEW sample were extremely healthy and did neither meet criteria of any axis-diagnoses nor BPD, had never taken psychotropic medication and did not have a history of psychotherapeutic interventions. Furthermore, the PTSD-patients' sample comprised highly impaired participants with a broad spectrum of comorbidities with BPD being the most frequent one. Many of the PTSD-patients also took psychotropic medication and had undergone former psychotherapeutic treatments. Both factors have been shown to possibly influence pain sensitivity and HRV in some studies (Agelink et al., 2001; Böhm, 2000; Kemp et al., 2016; Licht, de Geus, van Dyck, & Penninx, 2010; Moon et al., 2013). Accordingly, large effect sizes in the comparisons between HTEW and PTSD-patients with regard to these two measures need to be interpreted with caution. Furthermore, no data on disclosing has been assessed in the PTSD group.

To assess stress responsiveness in HTEW, the MMST, a valid and ecological laboratory design to induce stress, was chosen. Although the MMST is a multidimensional stressor that has been shown to elicit a high degree of stress (Kolotylova et al., 2010), it remains debatable whether laboratory induced stress can evoke comparable stress as real-life stress. Therefore, future studies should include other stressors such as social or emotional stress, as well as stressors in daily life, which might be of higher ecological validity.

One general major challenge in comparing traumatized individuals with PTSD to traumatized individuals without PTSD is the question of comparability of trauma type and severity of trauma. Different confounders complicate the comparison of HTEW and PTSD-patients: Experiences of PTE can differ with regard to type of trauma (i.e. CSA, CPA, child neglect, intimate partner violence, natural disaster, motor vehicle accidents, etc.), duration and frequency (single versus multiple trauma), relationship to perpetrator (family member versus stranger), age of onset (childhood versus adulthood) and many other variables. According to the literature, impairment in the aftermaths of trauma can differ to a variable extent relative to the respective trauma characteristics. For instance, multiple traumas might cause higher psychopathological levels (i.e., in dissociation and suicidal ideation) than single

trauma (Hagenaars, Fisch, & van Minnen, 2011; LeBouthillier, McMillan, Thibodeau, & Asmundson, 2015). Childhood trauma is thought to be of greater influence in developing mental disorders than adulthood trauma, as it happens in a period when developmental processes such as emotion regulation and interpersonal functioning take place (Maercker, Michael, Fehm, Becker, & Margraf, 2004; Shipman, Edwards, Brown, Swisher, & Jennings, 2005). Early trauma might also complicate early formation of secure attachment which in turn mediates the ability to regulate stress (Schore & Schore, 2008). Differences in trauma type and severity of trauma might also contribute to the probability of developing PTSD. In a metanalysis by Brewin and colleagues (2000), the factor trauma severity had a strong effect on the probability of developing PTSD; higher trauma severity leads to a higher probability of developing PTSD and higher PTSD severity as indicated by higher scores on the Impact of Events Scale-Revised version (Weiss & Marmar, 1996). However, the authors discuss a broad heterogeneity in measuring trauma severity across primary studies. This heterogeneity in defining trauma severity decreases the reliability of effect size estimates, but it increases the confidence in validity at the same time. In line with these findings, results of a study by Griffin and colleagues (2010) indicate that women with more severe CPA scored worse on social adjustment, self-esteem and life satisfaction and were more likely to have mental health problems than women with less severe CPA scores. Thus, results in these studies support the notion that psychopathologic impairment following trauma seems to be dose-dependent.

Previous studies also showed that victims of CSA are often at risk of experiencing additional trauma types (e.g., physical abuse) as well as a multitude of traumatic situations in one type of trauma (i.e. CSA; Chard, 2005; Cloitre et al., 2010; Kessler et al., 2017), a phenomenon described as revictimization. The accumulation of multiple stressors in childhood or adolescence dramatically increases the risk of developing psychopathologies (Kessler et al., 2017; Rutter, 1987).

As the sequelae of PTE are widespread and vary due to the respective trauma characteristics, it is of utmost importance to try to minimize differences in trauma type and severity between traumatized individuals with and without PTSD in research. Therefore, in the studies presented in this thesis, the approach to minimize the influence of differences in trauma severity between HTEW and PTSD-patients by means of matched subscale scores of Physical and Sexual Abuse of the CTQ was chosen (as seen in Roy et al., 2011). Although individuals in the HTEW and PTSD

group did not differ in these two subscales, PTSD-patients presented significantly higher scores on the subscale of Emotional Abuse of the CTQ as compared to HTEW. This factor could be seen as a group confounder and may contribute to differences in psychopathology between HTEW and PTSD-patients.

Additionally, the influence of cultural factors cannot be neglected. Maercker and colleagues (2009) state that this dose respondence (the more severe the trauma the more impairment caused by it) only becomes valid in societies where social acknowledgement as a victim is still lacking (e.g., traditional cultures such as China). This factor has to be kept in mind when examining and comparing psychopathological sequelae of CA across cultures.

The adapted approach in this thesis is a first step in trying to control and minimize differences in trauma severity between comparison groups. Future studies should also consider other variables that potentially affect severity and impact of trauma such as attachment experiences and quality of family environment (for an overview see Chris R Brewin et al., 2000; Maercker, Beauducel, & Schützwohl, 2000; Pierrehumbert et al., 2009). Unfortunately, these variables were not taken into consideration in the presented studies due to feasibility reasons.

Finally, all analyses were based on cross-sectional data, thus it is not possible to infer causation or directionality between exposure to PTE and impairment or alterations in psychophysiological parameters as well as in pain sensitivity. Accordingly, future longitudinal studies are needed to gain further insight into the sequential development of abnormalities: whether the traumatic exposure caused the abnormality and the abnormality caused the PTSD, or the traumatic exposure caused the PTSD, and the PTSD caused the abnormality or whether there are further mediating variables.

4.6 Conclusions and Future Directions

The work presented in this dissertation extends previous research by assessing and comparing three participant groups with regard to a broad spectrum of psychopathological and psychophysiological functioning: CA-exposed individuals without axis-I disorders or BPD, CA-exposed individuals with PTSD, and non-CA-exposed individuals without any mental disorders. This differentiation between participant groups facilitates disentangling the effects of mere exposure to CA from

the effects of PTSD in the sequel of CA-experiences. Within the two trauma-exposed participant groups, all women had experienced CA and PTSD symptoms within the PTSD group were also related to CA.

The two studies presented in this dissertation demonstrate that large effect-sizes separated individuals in the HTEW group from the PTSD group with regard to the collected measures in general and PTSD-specific psychopathologic symptoms as well as with regard to unspecific restrictions in psychological well-being. In contrast, effect-sizes between HTEW and HC were consistently small. Furthermore, individuals in the HTEW group showed a high level of functioning. No measurable restrictions in quality of life and sexual satisfaction were observed in HTEW as compared to HC. On a psychophysiological level, HR at baseline in the HTEW group was significantly higher than in the HC group and significantly lower than in the PTSD group. Likewise, the level of the HRV in the HTEW group was significantly lower than in the HC group and significantly higher than in the PTSD group. In response to stress, the increase of HR as well as the decrease of HRV in the HTEW group did not differ significantly from the two other groups. With respect to pain sensitivity, the level of pain sensitivity in the HTEW group was significantly lower than in the HC group and tended to be higher than in the PTSD group. In response to stress, pain sensitivity decreased in response to the stress-induction paradigm, while no significant difference between the HTEW and HC group was detected. However, the predefined contrast comparing the slopes in the HTEW and PTSD groups indicated a steeper decline for the pain ratings in the PTSD group in response to stress.

Our findings, taken together with results of previous studies (e.g., Carpenter et al., 2007; Lanius et al., 2002), suggest that trauma exposure and, in the respective studies, a history of CA do not per se elicit psychopathological impairment or restricted QoL and satisfaction with sexuality. On all these subjective measures, individuals in the HTEW group scored on levels comparable to these of HC without a history of CA. However, the picture was less clear with regard to the objective psychophysiological measures such as HR and HRV and with regard to pain sensitivity, which was measured by subjective rating, but encompasses a physiological component. On a psychophysiological level, a diagnosis of PTSD seems to be associated with alterations in HR and HRV basal levels. However, an additional role of CA-exposure per se should not be neglected. With respect to pain

sensitivity in response to experimentally induced stress, results of the present study suggest a rather strong effect of CA-exposure per se.

Disentangling the effect of PTSD and of mere trauma exposure among CA-exposed individuals is of clinical significance. Improved knowledge on the sequelae of CA may help identify novel diagnostic markers to detect individuals who are at risk of developing PTSD. This early detection of vulnerability to PTSD in turn would help to prevent developing a full-blown PTSD, as preventive programs could be called into action at an earlier stage. Likewise, the recognition of these mechanisms and sequelae can help improve treatment planning in terms of potential accompanying symptoms that are not entailed as diagnostic criteria of PTSD (i.e., reduced self-esteem, reduced QoL and satisfaction with sexuality). Improving treatment programs is important, as PTSD can be seen as a disorder causing serious impairment (Kessler et al., 2009). Apart from the personal burden and suffering, PTSD causes substantial burden in form of capital loss, i.e., more days out of work (Alonso et al., 2011) as well as reduced productivity on days being at work (Ormel et al., 2008) on a societal level.

Overall, good self-esteem, absence of guilt cognitions as well as the process of disclosing seem to be important in withstanding adversities unscathed. With potentially important implications for treatment, these findings highlight the importance of routinely inquiring about CA as well as other forms of assaultive and abusive experiences. Therefore, it is of utmost importance to educate medical and healthcare professionals concerning this matter, as those individuals affected by abuse and assault more often seek treatment at medical than at psychological facilities in the first year after the assault (Kimerling & Calhoun, 1994). Good prevention programs should focus on developing and supporting the adolescent's sense of being a good and worthy person, as well as educating and training support providers and people in the community about sexual and physical assault and how to respond to it in more supportive ways (Hakimi, Bryant-Davis, Ullman, & Gobin, 2016).

Furthermore, the importance of studying potentially protective factors that might help attenuate development of psychopathology in the aftermaths of trauma is beyond any doubt. However, when thinking about reasons *why* this particular group of HTEW withstands these adverse experiences of CA unscathed, this dissertation only provides first links for an explanation. As the aim of this dissertation was to examine

whether and to which extent healthy women with experiences of CA show psychopathological and psychophysiological impairment, the *"why"* was rather understudied. Future studies should use qualitative interviews with healthy CA-exposed participants to assess the question of what helped these people to withstand the adversities without developing psychopathologies. Future qualitative and quantitative studies should help broaden the understanding of potential predisposing or protective factors in the person or the environment.

Furthermore, it would be interesting to conduct studies with HTEW from different cultural backgrounds or with different value orientation. As already mentioned in the introduction, differences in cultural beliefs and values may play a role in assessing prevalence rates of CA. Additionally, differences in cultural beliefs and values may also play a role in developing psychopathologies in the aftermaths of trauma. According to Schwartz (1994), a traditional value orientation encompasses values such as conformity, benevolence, and customs orientation, whereas a modern value orientation encompasses achievement, hedonism, and stimulation. Maercker and colleagues (2009) have found increased PTSD-symptomatology in participants with traditional value orientation as compared to participants with modern values and a similar degree of criminalization. However, as former developing countries (i.e. China) have quite evolved within the last decade, it can no longer be presumed that cross-cultural studies automatically differentiate between modern or traditional value orientations. Therefore, future studies should explicitly assess value orientation in cross-sectional studies.

Finally, future studies could also disentangle the effects of PTSD versus other mental disorders (e.g., depression) and examine differences between genders, as research suggests that women report greater subjective and physiological stress responses compared to men (Kudielka et al., 2000).

5 Summary

Experiences of childhood abuse may have detrimental effects on the psychopathological and psychophysiological level. Evidence from previous studies mainly supports the notion that CA-exposed individuals show higher psychopathological impairment, such as intrusive experiences, dissociation, impaired general functioning, as well as reduced satisfaction with QoL and sexuality, as compared to individuals who have not been exposed to CA. On a psychophysiological level, this impairment is reflected by elevated HR and reduced HRV levels. Additionally, altered pain sensitivity has been associated with a history of CA. As a major limitation, the majority of these studies did not assess or control for effects of mental disorders (i.e., PTSD), preventing from disentangling effects of trauma and a PTSD diagnosis. Another branch of research has explicitly assessed these variables in individuals with PTSD related to CA and other kind of PTE and has found comparable results in the respective variables. However, up to this point, there is a research gap of studies on CA-exposed individuals both with and without PTSD that may disentangle the effects of the mere exposure to CA per se against the effects of a consequential PTSD diagnosis.

The aim of this dissertation was to examine a broad range of relevant psychopathological and psychophysiological sequelae of childhood abuse in women without mental disorders in order to disentangle the effects of trauma and psychopathology. For this purpose, two studies were conducted in order to focus on relevant psychopathological and psychophysiological aspects that are known to be impaired after experiencing CA and in patients with PTSD related to CA. These relevant aspects encompassed a broad range of general and PTSD-specific psychopathology, general functioning, quality of life, and satisfaction with sexuality. Additionally, stress responsiveness was examined at the subjective and psychophysiological level in response to experimentally induced stress. Furthermore, pain sensitivity at baseline as well as in response to stress were examined. These features were examined in HTEW as compared to HC women without a history of CA and female patients with PTSD related to experiences of CA.

In study I, HTEW showed a high level of functioning and a very low level of pathological impairment that was comparable to the level of healthy controls. The results of study I in this dissertation suggest that PTE exposure per se does not necessarily have to go along with the development of psychopathology or impaired quality of life, sexuality, self-esteem, or guilt cognitions. Results of the present study rather suggest an association of PTSD and impairment in the reported variables.

In the second study, the most distinct and clinically meaningful effects between HTEW and PTSD-patients were observed concerning subjective stress parameters. With regard to subjective ratings of stress and dissociation, HTEW were clearly different from PTSD-patients while resembling the group of HC. With respect to the psychophysiological parameters (HR and HRV) and pain sensitivity, the picture was less clear: Although HTEW differed significantly from PTSD-patients with respect to HR and HRV, differences were not as pronounced as for the subjective ratings. With respect to pain sensitivity in response to experimentally induced stress, comparisons of HTEW and PTSD-patients only approached significance. In comparison to HC, HTEW differed significantly for HR, HRV, and pain sensitivity. On a descriptive level, HR, HRV and pain sensitivity levels of HTEW were located in between levels of HC and of PTSD-patients. Results of the second study of this dissertation reveal that differences between HTEW and PTSD-patients in baseline stress levels as well as stress responses were most distinct in subjective as compared to psychophysiological readouts. This may imply that subjective stress rating and dissociation may be seen as diagnostic correlates of PTSD rather than being related to experiences of CA per se. Results of the objective stress indicators draw a less clear picture: Although HTEW differed significantly from PTSD-patients and HC with respect to HR and HRV, differences between HTEW and PTSD-patients were not as pronounced as for the subjective indicators of stress.

Taken together, based on recent advances in this field as well as results of our own work, it is proposed that psychopathology in form of a PTSD diagnosis seems to contribute to more general psychopathology (i.e., intrusions and dissociation), impaired general functioning, reduced QoL and satisfaction with sexuality, impaired baseline stress levels as well as stress responsiveness on a subjective as well as psychophysiological level. However, results of study II also suggest that one cannot exclude an additional role of CA on psychophysiological stress responses as well as

pain sensitivity as the data also indicates that a small proportion of altered stress response may relate to experiences of CA per se.

The contribution of this dissertation is an increased knowledge on the sequelae of CA apart from developing PTSD. Improved knowledge on the sequelae of CA may help identifying novel diagnostic markers to detect individuals who are at risk of developing PTSD. An early detection of vulnerability to PTSD in turn would help prevent developing a full-blown PTSD, as preventive programs could be called into action at an earlier stage. Furthermore, the recognition of these mechanisms and sequelae can help improve treatment planning in terms of potential accompanying symptoms that are not entailed as diagnostic criteria of PTSD (i.e., reduced self-esteem, reduced satisfaction with QoL and sexuality). Furthermore, results of this dissertation suggest that good self-esteem, absence of guilt cognitions, as well as the process of disclosure seem to be important in withstanding adversities unscathed. With potentially important implications for treatment, these findings highlight the importance of routinely inquiring about CA as well as educating and training support-providers and society about sexual and physical assault. Training programs should encompass how to respond to disclosure of CA in more supportive ways. Furthermore, the implementation of prevention programs focused on developing and supporting the adolescent's sense of being a good and worthy person should be encouraged.

6 References

Aakvaag, H. F., Thoresen, S., Wentzel-Larsen, T., Dyb, G., Røysamb, E., & Olff, M. (2016). Broken and guilty since it happened: A population study of trauma-related shame and guilt after violence and sexual abuse. *Journal of Affective Disorders, 204*, 16-23. doi:10.1016/j.jad.2016.06.004

Abhishekh, H. A., Nisarga, P., Kisan, R., Meghana, A., Chandran, S., Raju, T., & Sathyaprabha, T. N. (2013). Influence of age and gender on autonomic regulation of heart. *Journal of clinical monitoring and computing, 27*(3), 259-264. doi:10.1007/s10877-012-9424-3

Adenauer, H., Catani, C., Keil, J., Aichinger, H., & Neuner, F. (2010). Is freezing an adaptive reaction to threat? Evidence from heart rate reactivity to emotional pictures in victims of war and torture. *Psychophysiology, 47*(2), 315-322. doi:10.1111/j.1469-8986.2009.00940.x

Afifi, T. O., Enns, M. W., Cox, B. J., de Graaf, R., ten Have, M., & Sareen, J. (2007). Child abuse and health-related quality of life in adulthood. *The Journal of Nervous and Mental Disease, 195*(10), 797-804. doi:10.1097/NMD.0b013e3181567fdd

Agelink, M. W., Majewski, T., Wurthmann, C., Lukas, K., Ullrich, H., Linka, T., & Klieser, E. (2001). Effects of newer atypical antipsychotics on autonomic neurocardiac function: a comparison between amisulpride, olanzapine, sertindole, and clozapine. *Journal of clinical psychopharmacology, 21*(1), 8-13.

Agorastos, A., Boel, J. A., Heppner, P. S., Hager, T., Moeller-Bertram, T., Haji, U., . . . Stiedl, O. (2013). Diminished vagal activity and blunted diurnal variation of heart rate dynamics in posttraumatic stress disorder. *Stress, 16*(3), 300-310. doi:10.3109/10253890.2012.751369

Alexander, P. C. (2014). Dual-trauma couples and intimate partner violence. *Psychological Trauma: Theory, Research, Practice, and Policy, 6*(3), 224-231. doi:10.1037/a0036404

Alonso, J., Petukhova, M., Vilagut, G., Chatterji, S., Heeringa, S., Üstün, T. B., . . . Bromet, E. (2011). Days out of role due to common physical and mental conditions: results from the WHO World Mental Health surveys. *Molecular psychiatry, 16*(12), 1234-1246. doi:10.1038/mp.2010.101

American Psychiatric Association. (2000). Global assessment of functioning scale. *Diagnostic and statistical manual of mental disorders, 4th edition, text revision. Washington, DC: American Psychiatric Association, 34*.

American Psychiatric Association. (2013a). APA. *Diagnostic and statistical manual of mental disorders, 5*.

American Psychiatric Association. (2013b). *Diagnostic and statistical manual of mental disorders (5th ed.)*.

American Psychiatric Association. (2014). *The road to resilience*. Washington DC: American Psychological Association.

Amir, N., Stafford, J., Freshman, M. S., & Foa, E. B. (1998). Relationship between trauma narratives and trauma pathology. *Journal of Traumatic Stress, 11*(2), 385-392. doi:10.1023/A:1024415523495

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., . . . Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience, 256*(3), 174-186. doi:10.1007/s00406-005-0624-4

Anda, R. F., Tietjen, G., Schulman, E., Felitti, V., & Croft, J. (2010). Adverse childhood experiences and frequent headaches in adults. *Headache: The Journal of Head and Face Pain, 50*(9), 1473-1481. doi:10.1111/j.1526-4610.2010.01756.x

Andrews, G., Corry, J., Slade, T., Issakidis, C., & Swanston, H. (2004). Child sexual abuse. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors, 2*, 1851-1940.

Angermeyer, M. C., Kilian, R., & Matschinger, H. (2000). *WHOQOL-100 und WHOQOL-BREF: Handbuch für die deutschsprachige Version der WHO-Instrumente zur Erfassung von Lebensqualität*: Hogrefe & Huber.

Arbanas, G. (2010). Does post-traumatic stress disorder carry a higher risk of sexual dysfunctions? *J Sex Med, 7*(5), 1816-1821. doi:10.1111/j.1743-6109.2010.01704.x

Arnberg, F. K., Rydelius, P. A., & Lundin, T. (2011). A longitudinal follow-up of posttraumatic stress: from 9 months to 20 years after a major road traffic accident. *Child Adolesc Psychiatry Ment Health, 5*(1), 8. doi:10.1186/1753-2000-5-8

Asmundson, G. J., & Katz, J. (2008). Understanding pain and posttraumatic stress disorder comorbidity: do pathological responses to trauma alter the perception of pain? *Pain, 138*(2), 247-249.

Bandura, A., Cioffi, D., Taylor, C. B., & Brouillard, M. E. (1988). Perceived self-efficacy in coping with cognitive stressors and opioid activation. *J Pers Soc Psychol, 55*(3), 479-488.

Barratt, C. S. (1985). Impulsiveness subtraits, arousal and information processing. In C. E. Izard & S. J. T. Izard (Eds.), *Motivation, Emotion and Personality* (pp. 137-146). Amsterdam: Elsevier.

Beck, J. G., & Clapp, J. D. (2011). A different kind of co-morbidity: Understanding posttraumatic stress disorder and chronic pain. *Psychol Trauma, 3*(2), 101-108. doi:10.1037/a0021263

Beck, J. G., McNiff, J., Clapp, J. D., Olsen, S. A., Avery, M. L., & Hagewood, J. H. (2011). Exploring negative emotion in women experiencing intimate partner violence: Shame, guilt, and PTSD. *Behavior Therapy, 42*(4), 740-750. doi:Beck, J. G., et al. (2011). "Exploring negative emotion in women experiencing intimate partner violence: Shame, guilt, and PTSD." Behavior Therapy 42(4): 740-750.

Beecher, H. K. (1946). Pain in men wounded in battle. *Annals of surgery, 123*(1), 96.

Bekrater-Bodmann, R., Chung, B. Y., Richter, I., Wicking, M., Foell, J., Mancke, F., . . . Flor, H. (2015). Deficits in pain perception in borderline personality disorder: results from the thermal grill illusion. *Pain, 156*(10), 2084.

Belli, R. F. (2014). Autobiographical memory dynamics in survey research. *SAGE Handbook of Applied Memory*, 366-384.

Bengel, J., & Lyssenko, L. (2012). *Resilienz und psychologische Schutzfaktoren im Erwachsenenalter: Stand der Forschung zu psychologischen Schutzfaktoren von Gesundheit im Erwachsenenalter*: BZgA Bundeszentrale für Gesundheitliche Aufklärung.

Berens, A. E., Jensen, S. K., & Nelson, C. A. (2017). Biological embedding of childhood adversity: from physiological mechanisms to clinical implications. *BMC medicine, 15*(1), 135. doi:10.1186/s12916-017-0895-4

Berkley, K. J. (1997). Sex differences in pain. *Behavioral and Brain Sciences, 20*(03), 371-380.

Bernstein, D. P., Ahluvalia, T., Pogge, D., & Handelsman, L. (1997). Validity of the Childhood Trauma Questionnaire in an Adolescent Psychiatric Population. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(3), 340-348. doi:10.1097/00004583-199703000-00012

Bernstein, D. P., & Fink, L. (1998). *Childhood trauma questionnaire: A retrospective self-report: Manual*: Psychological Corporation.

Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *The Journal of Nervous and Mental Disease, 174*(12), 727-735.

Berthelot, N., Godbout, N., Hébert, M., Goulet, M., & Bergeron, S. (2014). Prevalence and Correlates of Childhood Sexual Abuse in Adults Consulting for Sexual Problems. *Journal of Sex & Marital Therapy, 40*(5), 434-443. doi:10.1080/0092623X.2013.772548

Biggs, A., Aziz, Q., Tomenson, B., & Creed, F. (2004). Effect of childhood adversity on health related quality of life in patients with upper abdominal or chest pain. *Gut, 53*(2), 180-186. doi:10.1136/gut.2003.020974

Bleich, A., & Solomon, Z. (2004). Evaluation of psychiatric disability in PTSD of military origin. *Isr J Psychiatry Relat Sci, 41*(4), 268-276.

Blonigen, D. M., Carlson, M. D., Hicks, B. M., Krueger, R. F., & Iacono, W. G. (2008). Stability and change in personality traits from late adolescence to early adulthood: a longitudinal twin study. *J Pers, 76*(2), 229-266. doi:10.1111/j.1467-6494.2007.00485.x

Böhm, J. (2000). Psychopharmaka.

Bolger, K. E., & Patterson, C. J. (2003). Sequelae of child maltreatment: Vulnerability and resilience. *Resilience and vulnerability: Adaptation in the context of childhood adversities*, 156-181.

Bonanno, G. A., & Diminich, E. D. (2013). Annual Research Review: Positive adjustment to adversity–trajectories of minimal–impact resilience and emergent resilience. *Journal of Child Psychology and Psychiatry, 54*(4), 378-401. doi:10.1111/jcpp.12021

Bond, F. W., Hayes, S. C., Baer, R. A., Carpenter, K. M., Guenole, N., Orcutt, H. K., . . . Zettle, R. D. (2011). Preliminary Psychometric Properties of the Acceptance and Action Questionnaire–II: A Revised Measure of Psychological Inflexibility and Experiential Avoidance. *Behavior Therapy, 42*(4), 676-688. doi:10.1016/j.beth.2011.03.007

Bonnot, O., Anderson, G. M., Cohen, D., Willer, J. C., & Tordjman, S. (2009). Are patients with schizophrenia insensitive to pain? A reconsideration of the question. *Clin J Pain, 25*(3), 244-252. doi:10.1097/AJP.0b013e318192be97

Bornefeld-Ettmann, P., Steil, R., Lieberz, K. A., Bohus, M., Rausch, S., Herzog, J., . . . Muller-Engelmann, M. (2018). Sexual Functioning After Childhood Abuse: The Influence of Post-Traumatic Stress Disorder and Trauma Exposure. *J Sex Med, 15*(4), 529-538. doi:10.1016/j.jsxm.2018.02.016

Braver, M., Bumberry, J., Green, K., & Rawson, R. (1992). Childhood abuse and current psychological functioning in a university counseling center population. *Journal of Counseling Psychology, 39*(2), 252-257. doi:10.1037/0022-0167.39.2.252

Bremner, J. D. (1999). Acute and chronic responses to psychological trauma: where do we go from here? *Am J Psychiatry, 156*(3), 349-351. doi:10.1176/ajp.156.3.349

Bremner, J. D., & Brett, E. (1997). Trauma-related dissociative states and long-term psychopathology in posttraumatic stress disorder. *Journal of Traumatic Stress, 10*(1), 37-49. doi:10.1002/jts.2490100105

Bremner, J. D., Narayan, M., Staib, L. H., Southwick, S., McGlashan, T., & Charney, D. S. (1999). Neural Correlates of Memories of Childhood Sexual Abuse in Women With and Without Posttraumatic Stress Disorder. *Am J Psychiatry, 156*(11), 1787-1795.

Bremner, J. D., Staib, L. H., Kaloupek, D., Southwick, S. M., Soufer, R., & Charney, D. S. (1999). Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder: a positron emission tomography study. *Biological Psychiatry, 45*(7), 806-816.

Breslau, N. (2001). Gender differences in trauma and posttraumatic stress disorder. *The journal of gender-specific medicine: JGSM: the official journal of the Partnership for Women's Health at Columbia, 5*(1), 34-40.

Breslau, N., Peterson, E., Poisson, L., Schultz, L., & Lucia, V. (2004). Estimating post-traumatic stress disorder in the community: lifetime perspective and the impact of typical traumatic events. *Psychol Med, 34*(5), 889-898.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology, 68*(5), 748.

Brewin, C. R., Cloitre, M., Hyland, P., Shevlin, M., Maercker, A., Bryant, R. A., . . . Reed, G. M. (2017). A review of current evidence regarding the ICD-11 proposals for diagnosing PTSD and complex PTSD. *Clin Psychol Rev, 58*, 1-15. doi:10.1016/j.cpr.2017.09.001

Briere, J. (2002). Treating Adult Survivors of Severe Childhood. *The APSAC handbook on child maltreatment, 175*.

Briere, J., & Elliott, D. M. (1994). Immediate and long-term impacts of child sexual abuse. *The future of children*, 54-69. doi:10.2307/1602523

Briere, J., & Spinazzola, J. (2005). Phenomenology and psychological assessment of complex posttraumatic states. *Journal of Traumatic Stress, 18*(5), 401-412. doi:10.1002/jts.20048

Brown, A. D., Barton, D. A., & Lambert, G. W. (2009). Cardiovascular abnormalities in patients with major depressive disorder. *CNS drugs, 23*(7), 583-602.

Brown, G. R., McBride, L., Bauer, M. S., Williford, W. O., & Team, C. S. P. S. (2005). Impact of childhood abuse on the course of bipolar disorder: a replication study in US veterans. *Journal of Affective Disorders, 89*(1), 57-67. doi:10.1016/j.jad.2005.06.012

Brown, J., Cohen, P., Johnson, J. G., & Smailes, E. M. (1999). Childhood abuse and neglect: specificity of effects on adolescent and young adult depression and suicidality. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(12), 1490-1496.

Brownlee, K., Rawana, J., Franks, J., Harper, J., Bajwa, J., O'Brien, E., & Clarkson, A. (2013). A systematic review of strengths and resilience outcome literature relevant to children and adolescents. *Child and Adolescent Social Work Journal, 30*(5), 435-459.

Buckley, T. C., Holohan, D., Greif, J. L., Bedard, M., & Suvak, M. (2004). Twenty-four-hour ambulatory assessment of heart rate and blood pressure in chronic PTSD and non-PTSD veterans. *Journal of Traumatic Stress, 17*(2), 163-171. doi:10.1023/B:JOTS.0000022623.01190.f0

Buckley, T. C., & Kaloupek, D. G. (2001). A meta-analytic examination of basal cardiovascular activity in posttraumatic stress disorder. *Psychosomatic Medicine, 63*(4), 585-594.

Bungert, M., Liebke, L., Thome, J., Haeussler, K., Bohus, M., & Lis, S. (2015). Rejection sensitivity and symptom severity in patients with borderline personality disorder: effects of childhood maltreatment and self-esteem. *Borderline Personal Disord Emot Dysregul, 2*, 4. doi:10.1186/s40479-015-0025-x

Büttner, M., Dulz, B., Sachsse, U., Overkamp, B., & Sack, M. (2014). Trauma und sexuelle Störungen. *Psychotherapeut, 59*(5), 385-391. doi:10.1007/s00278-014-1068-y

Calam, R., Horne, L., Glasgow, D., & Cox, A. (1998). Psychological disturbance and child sexual abuse: A follow-up study. *Child Abuse & Neglect, 22*(9), 901-913. doi:10.1016/S0145-2134(98)00068-4

Campbell, F. C. (2008). *Elements of metallurgy and engineering alloys*: ASM International.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., . . . Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62*(10), 1080-1087. doi:10.1016/j.biopsych.2007.05.002

Centers for Disease Control Prevention. (2000). Measuring healthy days: Population assessment of health-related quality of life. *Atlanta, Ga: CDC, 40*.

Chalmers, J. A., Quintana, D. S., Abbott, M. J., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Frontiers in psychiatry, 5*, 80. doi:10.3389/fpsyt.2014.00080

Chan, K. L., Brownridge, D. A., Yan, E., Fong, D. Y., & Tiwari, A. (2011). Child maltreatment polyvictimization: Rates and short-term effects on adjustment in a representative Hong Kong sample. *Psychology of Violence, 1*(1), 4. doi:10.1037/a0020284

Chang, H.-A., Chang, C.-C., Tzeng, N.-S., Kuo, T. B., Lu, R.-B., & Huang, S.-Y. (2013). Decreased cardiac vagal control in drug-naïve patients with posttraumatic stress disorder. *Psychiatry investigation, 10*(2), 121-130. doi:10.4306/pi.2013.10.2.121

Chard, K. M. (2005). An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *Journal of Consulting and Clinical Psychology, 73*(5), 965. doi:10.1037/0022-006X.73.5.965

Chen, L. P., Murad, M. H., Paras, M. L., Colbenson, K. M., Sattler, A. L., Goranson, E. N., . . . Prokop, L. J. (2010). *Sexual abuse and lifetime diagnosis of psychiatric disorders: systematic review and meta-analysis.* Paper presented at the Mayo Clinic Proceedings.

Christiansen, D., & Elklit, A. (2012). *Sex Differences in PTSD*: INTECH Open Access Publisher.

Cicchetti, D., & Rogosch, F. A. (2001). Diverse patterns of neuroendocrine activity in maltreated children. *Dev Psychopathol, 13*(3), 677-693.

Classen, C. C., Palesh, O. G., Cavanaugh, C. E., Koopman, C., Kaupp, J. W., Kraemer, H. C., . . . Spiegel, D. (2011). A comparison of trauma-focused and present-focused group therapy for survivors of childhood sexual abuse: A randomized controlled trial. *Psychological Trauma: Theory, Research, Practice, and Policy, 3*(1), 84. doi:10.1037/a0020096

Clifton, E. G., Feeny, N. C., & Zoellner, L. A. (2017). Anger and guilt in treatment for chronic posttraumatic stress disorder. *J Behav Ther Exp Psychiatry, 54*, 9-16. doi:10.1016/j.jbtep.2016.05.003

Cloitre, M., Cohen, L. R., Edelman, R. E., & Han, H. (2001). Posttraumatic stress disorder and extent of trauma exposure as correlates of medical problems and perceived health among women with childhood abuse. *Women & Health, 34*(3), 1-17. doi:10.1300/J013v34n03_01

Cloitre, M., Garvert, D. W., Weiss, B., Carlson, E. B., & Bryant, R. A. (2014). Distinguishing PTSD, Complex PTSD, and Borderline Personality Disorder: A latent class analysis. *Eur J Psychotraumatol, 5*. doi:10.3402/ejpt.v5.25097

Cloitre, M., Stovall-McClough, K. C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C. L., . . . Petkova, E. (2010). Treatment for PTSD related to childhood abuse: A randomized controlled trial. *American Journal of Psychiatry, 167*(8), 915-924. doi:10.1176/appi.ajp.2010.09081247

Coghill, R. C., McHaffie, J. G., & Yen, Y.-F. (2003). Neural correlates of interindividual differences in the subjective experience of pain. *Proceedings of the National Academy of Sciences, 100*(14), 8538-8542. doi:10.1073/pnas.1430684100

Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research, 96*(1), 1-13.

Cohen, H., Kotler, M., Matar, M. A., Kaplan, Z., Loewenthal, U., Miodownik, H., & Cassuto, Y. (1998). Analysis of heart rate variability in posttraumatic stress disorder patients in response to a trauma-related reminder. *Biological Psychiatry, 44*(10), 1054-1059. doi:10.1016/S0006-3223(97)00475-7

Coker, A. L., Smith, P. H., Thompson, M. P., McKeown, R. E., Bethea, L., & Davis, K. E. (2002). Social support protects against the negative effects of partner violence on mental health. *Journal of women's health & gender-based medicine, 11*(5), 465-476. doi:10.1089/15246090260137644.

Corso, P. S., Edwards, V. J., Fang, X., & Mercy, J. A. (2008). Health-related quality of life among adults who experienced maltreatment during childhood. *Am J Public Health, 98*(6), 1094-1100. doi:10.2105/AJPH.2007.119826

Costanzo, M., Jovanovic, T., Norrholm, S. D., Ndiongue, R., Reinhardt, B., & Roy, M. J. (2016). Psychophysiological Investigation of Combat Veterans with Subthreshold Post-traumatic Stress Disorder Symptoms. *Mil Med, 181*(8), 793-802. doi:10.7205/milmed-d-14-00671

Craft, R. M., Mogil, J. S., & Aloisi, A. M. (2004). Sex differences in pain and analgesia: the role of gonadal hormones. *European Journal of Pain, 8*(5), 397-411. doi:10.1016/j.ejpain.2004.01.003

Cutajar, M. C., Mullen, P. E., Ogloff, J. R. P., Thomas, S. D., Wells, D. L., & Spataro, J. (2010). Psychopathology in a large cohort of sexually abused children followed up to 43 years. *Child Abuse & Neglect, 34*(11), 813-822. doi:10.1016/j.chiabu.2010.04.004

Cygankiewicz, I., & Zareba, W. (2013). Heart rate variability. *Handb Clin Neurol, 117*(3), 379-393.

d'Ardenne, P., Capuzzo, N., Fakhoury, W. K., Jankovic-Gavrilovic, J., & Priebe, S. (2005). Subjective quality of life and posttraumatic stress disorder. *The Journal of Nervous and Mental Disease, 193*(1), 62-65.

D'Andrea, W., Sharma, R., Zelechoski, A. D., & Spinazzola, J. (2011). Physical health problems after single trauma exposure: When stress takes root in the body. *Journal of the American Psychiatric Nurses Association, 17*(6), 378-392. doi:10.1177/1078390311425187

Dannlowski, U., Stuhrmann, A., Beutelmann, V., Zwanzger, P., Lenzen, T., Grotegerd, D., . . . Bauer, J. (2012). Limbic scars: long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry, 71*(4), 286-293. doi:10.1016/j.biopsych.2011.10.021

Davidson, J. R., Book, S. W., Colket, J. T., Tupler, L. A., Roth, S., David, D., . . . Feldman, M. E. (1997). Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychol Med, 27*(1), 153-160.

Davydov, D. M., Stewart, R., Ritchie, K., & Chaudieu, I. (2010). Resilience and mental health. *Clinical Psychology Review, 30*(5), 479-495. doi:10.1016/j.cpr.2010.03.003

Defrin, R., Ginzburg, K., Solomon, Z., Polad, E., Bloch, M., Govezensky, M., & Schreiber, S. (2008). Quantitative testing of pain perception in subjects with PTSD–implications for the mechanism of the coexistence between PTSD and chronic pain. *Pain, 138*(2), 450-459.

Defrin, R., Lahav, Y., & Solomon, Z. (2017). Dysfunctional Pain Modulation in Torture Survivors: The Mediating Effect of PTSD. *J Pain, 18*(1), 1-10. doi:10.1016/j.jpain.2016.09.005

Defrin, R., Schreiber, S., & Ginzburg, K. (2015). Paradoxical pain perception in posttraumatic stress disorder: the unique role of anxiety and dissociation. *The Journal of Pain, 16*(10), 961-970. doi:10.1016/j.jpain.2015.06.010

Dennis, P. A., Dedert, E. A., Van Voorhees, E. E., Watkins, L. L., Hayano, J., Calhoun, P. S., . . . Beckham, J. C. (2016). Examining the Crux of Autonomic Dysfunction in Posttraumatic Stress Disorder: Whether Chronic or Situational Distress Underlies Elevated Heart Rate and Attenuated Heart Rate Variability. *Psychosomatic Medicine*.

Dennis, P. A., Watkins, L., Calhoun, P. S., Oddone, A., Sherwood, A., Dennis, M. F., . . . Beckham, J. C. (2014). Posttraumatic stress, heart-rate variability, and the mediating role of behavioral health risks. *Psychosomatic Medicine, 76*(8), 629.

Derogatis, L. R. (1993). *BSI, Brief Symptom Inventory: Administration, scoring, and procedures manual (4th ed.)*. Minneapolis: MN: National Computer Systems.

Dickinson, L. M., deGruy III, F. V., Dickinson, W. P., & Candib, L. M. (1999). Health-related quality of life and symptom profiles of female survivors of sexual abuse. *Archives of family medicine, 8*(1), 35-43.

Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of personality assessment, 49*(1), 71-75. doi:10.1207/s15327752jpa4901_13

Diener, S. J., Wessa, M., Ridder, S., Lang, S., Diers, M., Steil, R., & Flor, H. (2012). Enhanced stress analgesia to a cognitively demanding task in patients with posttraumatic stress disorder. *Journal of Affective Disorders, 136*(3), 1247-1251.

DiGangi, J. A., Gomez, D., Mendoza, L., Jason, L. A., Keys, C. B., & Koenen, K. C. (2013). Pretrauma risk factors for posttraumatic stress disorder: A systematic review of the literature. *Clinical Psychology Review, 33*(6), 728-744. doi:10.1016/j.cpr.2013.05.002

DiLillo, D., Lewis, T., & Loreto-Colgan, A. D. (2007). Child Maltreatment History and Subsequent Romantic Relationships. *Journal of Aggression, Maltreatment & Trauma, 15*(1), 19-36. doi:10.1300/J146v15n01_02

Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L., & Blair, S. N. (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *International Journal of Psychophysiology, 37*(2), 121-133. doi:10.1016/S0167-8760(00)00085-4

Domhardt, M., Münzer, A., Fegert, J. M., & Goldbeck, L. (2014). Resilience in Survivors of Child Sexual Abuse A Systematic Review of the Literature. *Trauma, Violence, & Abuse*, 1524838014557288. doi:10.1177/1524838014557288

Drossman, D. A., Talley, N. J., Leserman, J., Olden, K. W., & Barreiro, M. A. (1995). Sexual and physical abuse and gastrointestinal illness: review and recommendations. *Annals of internal medicine, 123*(10), 782-794. doi:10.7326/0003-4819-123-10-199511150-00007

Dube, S. R., Anda, R. F., Whitfield, C. L., Brown, D. W., Felitti, V. J., Dong, M., & Giles, W. H. (2005). Long-term consequences of childhood sexual abuse by gender of victim. *American Journal of Preventive Medicine, 28*(5), 430-438. doi:10.1016/j.amepre.2005.01.015

Dufton, L. M., Konik, B., Colletti, R., Stanger, C., Boyer, M., Morrow, S., & Compas, B. E. (2008). Effects of stress on pain threshold and tolerance in children with recurrent abdominal pain. *Pain, 136*(1), 38-43. doi:10.1016/j.pain.2007.06.012

Duncan, R. D., Saunders, B. E., Kilpatrick, D. G., Hanson, R. F., & Resnick, H. S. (1996). Childhood physical assault as a risk factor for PTSD, depression, and substance abuse: findings from a national survey. *American Journal of orthopsychiatry, 66*(3), 437.

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., & Fennell, M. (2005). Cognitive therapy for post-traumatic stress disorder: development and evaluation. *Behaviour Research and Therapy, 43*(4), 413-431. doi:Disclosing itself ist wichtig und dann noch die Reaktion aus dem Umfeld!!

Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology, 107*(3), 508-519.

Elzinga, B. M., Schmahl, C. G., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse-related PTSD. *Neuropsychopharmacology, 28*(9), 1656-1665.

Everill, J., & Waller, G. (1995). Disclosure of sexual abuse and psychological adjustment in female undergraduates. *Child Abuse & Neglect, 19*(1), 93-100. doi:10.1016/0145-2134(94)00102-Z

Ewing, D. J., Martyn, C. N., Young, R. J., & Clarke, B. F. (1985). The value of cardiovascular autonomic function tests: 10 years experience in diabetes. *Diabetes care, 8*(5), 491-498.

Fagundes, C. P., Glaser, R., & Kiecolt-Glaser, J. K. (2013). Stressful early life experiences and immune dysregulation across the lifespan. *Brain, behavior, and immunity, 27*, 8-12. doi:10.1016/j.bbi.2012.06.014

Feiring, C., Taska, L., & Lewis, M. (2002). Adjustment following sexual abuse discovery: the role of shame and attributional style. *Developmental psychology, 38*(1), 79. doi:10.1037/0012-1649.38.1.79

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., . . . Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse

Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14*(4), 245-258. doi:10.1016/S0749-3797(98)00017-8

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008). Exposure to childhood sexual and physical abuse and adjustment in early adulthood. *Child Abuse & Neglect, 32*(6), 607-619. doi:10.1016/j.chiabu.2006.12.018

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2011). Structural equation modeling of repeated retrospective reports of childhood maltreatment. *International journal of methods in psychiatric research, 20*(2), 93-104. doi:10.1002/mpr.337

Fergusson, D. M., & Lynskey, M. T. (1997). Physical punishment/maltreatment during childhood and adjustment in young adulthood. *Child Abuse & Neglect, 21*(7), 617-630. doi:10.1016/S0145-2134(97)00021-5

Fergusson, D. M., McLeod, G. F., & Horwood, L. J. (2013). Childhood sexual abuse and adult developmental outcomes: Findings from a 30-year longitudinal study in New Zealand. *Child Abuse & Neglect, 37*(9), 664-674. doi:10.1016/j.chiabu.2013.03.013

Fillingim, R. B., & Edwards, R. R. (2005). Is self-reported childhood abuse history associated with pain perception among healthy young women and men? *The Clinical journal of pain, 21*(5), 387-397.

Finkelhor, D. (2008). *Childhood victimization: Violence, crime, and abuse in the lives of young people*: oxford university Press.

Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2015). Prevalence of Childhood Exposure to Violence, Crime, and Abuse: Results From the National Survey of Children's Exposure to Violence. *JAMA Pediatr, 169*(8), 746-754. doi:10.1001/jamapediatrics.2015.0676

Fletcher, D., & Sarkar, M. (2013). Psychological resilience. *European Psychologist*. doi:10.1027/1016-9040/a000124

Flisher, A. J., Kramer, R. A., Hoven, C. W., Greenwald, S., Alegria, M., Bird, H. R., . . . Moore, R. E. (1997). Psychosocial characteristics of physically abused children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(1), 123-131. doi:10.1097/00004583-199701000-00026

Freyberger, H. J., Spitzer, C., Stieglitz, R. D., Kuhn, G., Magdeburg, N., & Bernstein Carlson, E. (1998). Fragebogen zu dissoziativen Symptomen (FDS) Deutsche Adaptation, Reliabilität und Validität der amerikanischen Dissociative Experience Scale (DES). *Psychotherapie, Psychosomatik, Medizinische Psychologie, 48*(6), 223-229.

Frid, M., Singer, G., Oei, T., & Rana, C. (1981). Reactions to ischemic pain: interactions between individual, situational and naloxone effects. *Psychopharmacology (Berl), 73*(2), 116-119. doi:10.1007/BF00429200

Fuller-Thomson, E., Brennenstuhl, S., & Frank, J. (2010). The association between childhood physical abuse and heart disease in adulthood: Findings from a representative community sample. *Child Abuse & Neglect, 34*(9), 689-698. doi:10.1016/j.chiabu.2010.02.005

Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., & Vlahov, D. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *New England Journal of Medicine, 346*(13), 982-987. doi:10.1056/NEJMsa013404

Geuze, E., Westenberg, H. G., Jochims, A., de Kloet, C. S., Bohus, M., Vermetten, E., & Schmahl, C. (2007). Altered pain processing in veterans with posttraumatic stress disorder. *Archives of General Psychiatry, 64*(1), 76-85.

Geva, N., Pruessner, J., & Defrin, R. (2014). Acute psychosocial stress reduces pain modulation capabilities in healthy men. *PAIN®, 155*(11), 2418-2425. doi:10.1016/j.pain.2014.09.023

Ghate, D., Creighton, S., & Field, J. (2002). A national study of parents, children and discipline. *Swindon: Economic and Social Reserach Council*.

Gibson, S. J., & Helme, R. D. (2001). Age-related differences in pain perception and report. *Clinics in geriatric medicine, 17*(3), 433-456.

Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Burden and consequences of child maltreatment in high-income countries. *The Lancet, 373*(9657), 68-81. doi:10.1016/S0140-6736(08)61706-7

Gill, J. M., Saligan, L., Lee, H., Rotolo, S., & Szanton, S. (2012). Women in recovery from PTSD have similar inflammation and quality of life as non-traumatized controls. *Journal of Psychosomatic Research, 74*(4), 301-306. doi:10.1016/j.jpsychores.2012.10.013

Ginzburg, K., Butler, L. D., Giese-Davis, J., Cavanaugh, C. E., Neri, E., Koopman, C., . . . Spiegel, D. (2009). Shame, guilt, and posttraumatic stress disorder in adult survivors of childhood sexual abuse at risk for human immunodeficiency virus: outcomes of a randomized clinical trial of group psychotherapy treatment. *The Journal of Nervous and Mental Disease, 197*(7), 536-542. doi:10.1097/NMD.0b013e3181ab2ebd

Ginzburg, K., Koopman, C., Butler, L. D., Palesh, O., Kraemer, H. C., Classen, C. C., & Spiegel, D. (2006). Evidence for a dissociative subtype of post-traumatic stress disorder among help-seeking childhood sexual abuse survivors. *J Trauma Dissociation, 7*(2), 7-27. doi:10.1300/J229v07n02_02

Girdler, S. S., Maixner, W., Naftel, H. A., Stewart, P. W., Moretz, R. L., & Light, K. C. (2005). Cigarette smoking, stress-induced analgesia and pain perception in men and women. *Pain, 114*(3), 372-385. doi:10.1016/j.pain.2004.12.035

Glover, E. M., Phifer, J. E., Crain, D. F., Norrholm, S. D., Davis, M., Bradley, B., . . . Jovanovic, T. (2011). Tools for translational neuroscience: PTSD is associated with heightened fear responses using acoustic startle but not skin conductance measures. *Depression and Anxiety, 28*(12), 1058-1066. doi:10.1002/da.20880

Golding, J. M. (1999). Sexual assault history and headache: five general population studies. *The Journal of Nervous and Mental Disease, 187*(10), 624-629.

Gómez-Pérez, L., & López-Martínez, A. E. (2013). Association of trauma, posttraumatic stress disorder, and experimental pain response in healthy young women. *The Clinical journal of pain, 29*(5), 425-434.

Goodman, B. F., & Griffin, M. G. (2018). Prospectively predicting PTSD status with heart rate reactivity and recovery in interpersonal violence survivors. *Psychiatry Res, 259*, 270-276. doi:10.1016/j.psychres.2017.10.036

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., . . . Pickering, R. P. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of clinical psychiatry, 69*(4), 533.

Griffin, M. L., & Amodeo, M. (2010). Predicting long-term outcomes for women physically abused in childhood: Contribution of abuse severity versus family environment. *Child Abuse & Neglect, 34*(10), 724-733. doi:10.1016/j.chiabu.2010.03.005

Gronwall, D. (1977). Paced auditory serial-addition task: a measure of recovery from concussion. *Perceptual and motor skills, 44*(2), 367-373.

Gunderson, J. G., & Sabo, A. N. (1993). The Phenomenological and Conceptual Interface between borderline personality disorder and PTSD. *Am J Psychiatry, 150*, 19-27. doi:10.1176/ajp.150.1.19

Gustafson, T., & Sarwer, D. (2004). Childhood sexual abuse and obesity. *Obesity reviews, 5*(3), 129-135. doi:10.1111/j.1467-789X.2004.00145.x

Haase, A., Boos, A., Schönfeld, S., & Hoyer, J. (2009). Sexuelle Dysfunktionen und sexuelle Zufriedenheit bei Patientinnen mit posttraumatischer Belastungsstörung. *Verhaltenstherapie, 19*(3), 161-167. doi:10.1159/000228725

Hagenaars, M. A., Fisch, I., & van Minnen, A. (2011). The effect of trauma onset and frequency on PTSD-associated symptoms. *Journal of Affective Disorders, 132*(1), 192-199.

Hakimi, D., Bryant-Davis, T., Ullman, S. E., & Gobin, R. L. (2016). Relationship between negative social reactions to sexual assault disclosure and mental health outcomes of black and white female survivors. doi:dx.doi.org/10.1037/tra0000245

Hatch, S. L., & Dohrenwend, B. P. (2007). Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *American journal of community psychology, 40*(3-4), 313-332. doi:10.1007/s10464-007-9134-z

Hauschildt, M., Peters, M. J. V., Moritz, S., & Jelinek, L. (2011). Heart rate variability in response to affective scenes in posttraumatic stress disorder. *Biological Psychology, 88*(2–3), 215-222. doi:10.1016/j.biopsycho.2011.08.004

Häuser, W., Schmutzer, G., Brähler, E., & Glaesmer, H. (2011). Maltreatment in Childhood and Adolescence. *Dtsch Arztebl, 108*(17), 287-294. doi:10.3238/arztebl.2011.0287

Hautzinger, M., Keller, F., & Kühner, C. (2006). BDI-II Beck Depressions-Inventar.

Hebert, M., Tourigny, M., Cyr, M., McDuff, P., & Joly, J. (2009). Prevalence of childhood sexual abuse and timing of disclosure in a representative sample of adults from Quebec. *Can J Psychiatry, 54*(9), 631-636. doi:10.1177/070674370905400908

Hecht, D. B., & Hansen, D. J. (2001). The environment of child maltreatment: Contextual factors and the development of psychopathology. *Aggression and Violent Behavior, 6*(5), 433-457. doi:10.1016/S1359-1789(00)00015-X

Heim, C. (2009). Childhood trauma and adult stress responsiveness.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., . . . Nemeroff, C. B. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *JAMA, 284*(5), 592-597. doi:10.1001/jama.284.5.592

Herbert, T. B., & Dunkel-Schetter, C. (1992). Negative social reactions to victims: An overview of responses and their determinants.

Herrman, H., Stewart, D. E., Diaz-Granados, N., Berger, E. L., Jackson, B., & Yuen, T. (2011). What is resilience? *The Canadian Journal of Psychiatry, 56*(5), 258-265.

Hollins, M., Harper, D., & Maixner, W. (2011). Changes in pain from a repetitive thermal stimulus: The roles of adaptation and sensitization. *PAIN®, 152*(7), 1583-1590. doi:10.1016/j.pain.2011.02.049

Hulme, P. A. (2010). Childhood Sexual Abuse, HPA Axis Regulation, and Mental Health. *Western Journal of Nursing Research, 33*(8), 1069-1097. doi:10.1177/0193945910388949

Jackson, J. L., Calhoun, K. S., Amick, A. E., Maddever, H. M., & Habif, V. L. (1990). Young adult women who report childhood intrafamilial sexual abuse: Subsequent adjustment. *Archives of Sexual Behavior, 19*(3), 211-221. doi:10.1007/bf01541547

Janet, P. (1889). *L'automatisme psychologique [Mental automatism]*. Paris, France: Nouvelle Edition.

Jenewein, J., Erni, J., Moergeli, H., Grillon, C., Schumacher, S., Mueller-Pfeiffer, C., . . . Hasler, G. (2016). Altered Pain Perception and Fear-Learning Deficits in Subjects With Posttraumatic Stress Disorder. *J Pain, 17*(12), 1325-1333. doi:10.1016/j.jpain.2016.09.002

Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M., & Bernstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry, 56*(7), 600-606. doi:10.1001/archpsyc.56.7.600

Jovanovic, T., Kazama, A., Bachevalier, J., & Davis, M. (2012). Impaired safety signal learning may be a biomarker of PTSD. *Neuropharmacology, 62*(2), 695-704. doi:10.1016/j.neuropharm.2011.02.023

Kalisch, R., Müller, M. B., & Tüscher, O. (2015). Advancing empirical resilience research. *Behavioral and Brain Sciences, 38*, e128. doi:10.1017/S0140525X15000023

Keary, T. A., Hughes, J. W., & Palmieri, P. A. (2009). Women with posttraumatic stress disorder have larger decreases in heart rate variability during stress tasks. *International Journal of Psychophysiology, 73*(3), 257-264. doi:10.1016/j.ijpsycho.2009.04.003

Kemp, A. H., Fráguas, R., Brunoni, A. R., Bittencourt, M. S., Nunes, M. A., Dantas, E. M., . . . Koenig, J. (2016). Differential associations of specific selective serotonin reuptake inhibitors with resting-state heart rate and heart rate variability: implications for health and well-being. *Psychosomatic Medicine, 78*(7), 810-818.

Kendall-Tackett, K. A., Williams, L. M., & Finkelhor, D. (1993). Impact of sexual abuse on children: A review and synthesis of recent empirical studies. *Psychological Bulletin, 113*(1), 164-180. doi:10.1037/0033-2909.113.1.164

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Benjet, C., Bromet, E. J., Cardoso, G., . . . Ferry, F. (2017). Trauma and PTSD in the WHO World Mental Health Surveys. *Eur J Psychotraumatol, 8*(sup5), 1353383. doi:10.1080/20008198.2017.1353383

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Chatterji, S., Lee, S., Ormel, J., . . . Wang, P. S. (2009). The global burden of mental disorders: an update from the WHO World Mental Health (WMH) surveys. *Epidemiology and Psychiatric Sciences, 18*(1), 23-33. doi:10.1017/S1121189X00001421

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry, 52*(12), 1048-1060. doi:10.1001/archpsyc.1995.03950240066012

Kimerling, R., & Calhoun, K. S. (1994). Somatic symptoms, social support, and treatment seeking among sexual assault victims. *Journal of Consulting and Clinical Psychology, 62*(2), 333. doi:10.1037/0022-006X.62.2.333

Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., . . . Meyer-Lindenberg, A. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of neuroscience, 25*(49), 11489-11493. doi:10.1523/JNEUROSCI.3984-05.2005

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*(1-2), 76-81. doi:10.1159/000119004

Kisiel, C. L., & Lyons, J. S. (2001). Dissociation as a mediator of psychopathology among sexually abused children and adolescents. *American Journal of Psychiatry, 158*(7), 1034-1039. doi:10.1176/appi.ajp.158.7.1034

Kleim, B., Graham, B., Bryant, R. A., & Ehlers, A. (2013). Capturing intrusive re-experiencing in trauma survivors' daily lives using ecological momentary assessment. *Journal of Abnormal Psychology, 122*(4), 998-1009. doi:10.1037/a0034957

Kleindienst, N., Limberger, M. F., Ebner-Priemer, U. W., Keibel-Mauchnik, J., Dyer, A., Berger, M., . . . Bohus, M. (2011). Dissociation predicts poor response to Dialectial Behavioral Therapy in female patients with Borderline Personality Disorder. *J Pers Disord, 25*(4), 432-447. doi:10.1521/pedi.2011.25.4.432

Kleindienst, N., Priebe, K., Görg, N., Dyer, A., Steil, R., Lyssenko, L., . . . Bohus, M. (2016). State dissociation moderates response to dialectical behavior therapy for posttraumatic stress disorder in women with and without borderline personality disorder. *Eur J Psychotraumatol, 7*.

Klingler, O. J., & Loewit, K. K. (1996). Der Fragebogen "Ressourcen in Sexualität und Partnerschaft" (RSP)- Konzeption und Ergebnisse zur Validität. *Zeitschrift für Differentielle und Diagnostische Psychologie, 17*(4), 268-275.

Knefel, M., Tran, U. S., & Lueger-Schuster, B. (2016). The association of posttraumatic stress disorder, complex posttraumatic stress disorder, and borderline personality disorder from a network analytical perspective. *J Anxiety Disord, 43*, 70-78. doi:10.1016/j.janxdis.2016.09.002

Kogan, S. M. (2004). Disclosing unwanted sexual experiences: Results from a national sample of adolescent women. *Child Abuse & Neglect, 28*(2), 147-165. doi:10.1016/j.chiabu.2003.09.014

Kolotylova, T., Koschke, M., Bär, K.-J., Ebner-Priemer, U., Kleindienst, N., Bohus, M., & Schmahl, C. (2010). Entwicklung des „Mannheimer Multikomponenten-Stress-Test" (MMST). *PPmP - Psychotherapie · Psychosomatik · Medizinische Psychologie, 60*(2), 64-72. doi:10.1055/s-0028-1103297

Krahé, B., & Berger, A. (2013). Men and women as perpetrators and victims of sexual aggression in heterosexual and same-sex encounters: A study of first-year college students in Germany. *Aggressive behavior, 39*(5), 391-404. doi:10.1002/ab.21482

Kraus, A., Geuze, E., Schmahl, C., Greffrath, W., Treede, R.-D., Bohus, M., & Vermetten, E. (2009). Differentiation of pain ratings in combat-related posttraumatic stress disorder. *PAIN®, 143*(3), 179-185.

Krause-Utz, A., Oei, N. Y., Niedtfeld, I., Bohus, M., Spinhoven, P., Schmahl, C., & Elzinga, B. (2012). Influence of emotional distraction on working memory performance in borderline personality disorder. *Psychol Med, 42*(10), 2181-2192. doi:10.1017/S0033291712000153

Krug, E. G., Mercy, J. A., Dahlberg, L. L., & Zwi, A. B. (2002). The world report on violence and health. *The Lancet, 360*(9339), 1083-1088. doi:10.1016/S0140-6736(02)11133-0

Kubany, E. S., Haynes, S. N., Abueg, F. R., Manke, F. P., Brennan, J. M., & Stahura, C. (1996). Development and validation of the Trauma-Related Guilt Inventory (TRGI). *Psychol Assess, 8*(4), 428-444. doi:10.1037/1040-3590.8.4.428

Kubany, E. S., & Manke, F. P. (1995). Cognitive therapy for trauma-related guilt: Conceptual bases and treatment outlines. *Cognitive and Behavioral Practice, 2*(1), 27-61. doi:10.1016/S1077-7229(05)80004-5

Kubany, E. S., & Watson, S. B. (2003). Guilt: Elaboration of a multidimensional model. *The Psychological Record, 53*(1), 51.

Kudielka, B. M., Hellhammer, D., & Kirschbaum, C. (2000). *Sex differences in human stress response* (Vol. 3): Academic Press, San Diego.

Lang, A. J., Stein, M. B., Kennedy, C. M., & Foy, D. W. (2004). Adult psychopathology and intimate partner violence among survivors of childhood maltreatment. *J Interpers Violence, 19*(10), 1102-1118.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). *International Affective Picture System (IAPS): Technical manual and affective ratings*. Gainesville: The Center for Research in Psychophysiology, University of Florida.

Lanius, R. A., Brand, B., Vermetten, E., Frewen, P. A., & Spiegel, D. (2012). The dissociative subtype of posttraumatic stress disorder: Rationale, clinical and neurobiological evidence, and implications. *Depression and Anxiety, 29*(8), 701-708. doi:10.1002/da.21889

Lanius, R. A., Williamson, P. C., Bluhm, R. L., Densmore, M., Boksman, K., Neufeld, R. W. J., . . . Menon, R. S. (2005). Functional connectivity of dissociative responses in posttraumatic stress disorder: A functional magnetic resonance imaging investigation. *Biological Psychiatry, 57*(8), 873-884. doi:10.1016/j.biopsych.2005.01.011

Lanius, R. A., Williamson, P. C., Boksman, K., Densmore, M., Gupta, M., Neufeld, R. W., . . . Menon, R. S. (2002). Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biological Psychiatry, 52*(4), 305-311. doi:10.1016/S0006-3223(02)01367-7

Laumann, E. O. (1994). *The social organization of sexuality: Sexual practices in the United States*: University of Chicago Press.

Laumann, E. O., Paik, A., Glasser, D. B., Kang, J.-H., Wang, T., Levinson, B., . . . Gingell, C. (2006). A cross-national study of subjective sexual well-being among older women and men: findings from the Global Study of Sexual Attitudes and Behaviors. *Archives of Sexual Behavior, 35*(2), 143-159. doi:10.1007/s10508-005-9005-3

Laurent, S. M., & Simons, A. D. (2009). Sexual dysfunction in depression and anxiety: Conceptualizing sexual dysfunction as part of an internalizing dimension. *Clinical Psychology Review, 29*(7), 573-585. doi:10.1016/j.cpr.2009.06.007

Lazarus, R. S. (1991). Progress on a cognitive-motivational-relational theory of emotion. *American psychologist, 46*(8), 819.

LeBouthillier, D. M., McMillan, K. A., Thibodeau, M. A., & Asmundson, G. J. (2015). Types and number of traumas associated with suicidal ideation and suicide attempts in PTSD: Findings from a US nationally representative sample. *Journal of Traumatic Stress, 28*(3), 183-190. doi:10.1002/jts.22010

Lee, E. A. D., & Theus, S. A. (2012). Lower heart rate variability associated with military sexual trauma rape and posttraumatic stress disorder. *Biological research for nursing, 14*(4), 412-418.

Leeb, R. T., Paulozzi, L., Melanson, C., Simon, T., & Arias, I. (2008). Child Maltreatment Surveillance: Uniform Definitions for Public Health and Recommended Data Elements, Version 1.0 Atlanta (GA): Centers for

Deasease Control and Prevention, National Center for Injury Prevention and Control.

Lehrl, S., Merz, J., Burkhard, G., & Fischer, S. (1991). Mehrfach-Wortschatz Test. *Göttingen: Hogrefe*.

Leonard, L. M., & Follette, V. M. (2002). Sexual functioning in women reporting a history of child sexual abuse: Review of the empirical literature and clinical implications. *Annual review of sex research, 13*(1), 346-388.

Leonard, L. M., Iverson, K. M., & Follette, V. M. (2008). Sexual Functioning and Sexual Satisfaction Among Women Who Report a History of Childhood and/or Adolescent Sexual Abuse. *Journal of Sex & Marital Therapy, 34*(5), 375-384. doi:10.1080/00926230802156202

Letourneau, E. J., Resnick, H. S., Kilpatrick, D. G., Saunders, B. E., & Best, C. L. (1996). Comorbidity of sexual problems and posttraumatic stress disorder in female crime victims. *Behavior Therapy, 27*(3), 321-336. doi:10.1016/S0005-7894(96)80020-7

Lewis, R. W., Fugl-Meyer, K. S., Corona, G., Hayes, R. D., Laumann, E. O., Moreira, E. D., Jr., . . . Segraves, T. (2010). Definitions/epidemiology/risk factors for sexual dysfunction. *J Sex Med, 7*(4 Pt 2), 1598-1607. doi:10.1111/j.1743-6109.2010.01778.x

Lewis, T., McElroy, E., Harlaar, N., & Runyan, D. (2016). Does the impact of child sexual abuse differ from maltreated but non-sexually abused children? A prospective examination of the impact of child sexual abuse on internalizing and externalizing behavior problems. *Child Abuse Negl, 51*, 31-40. doi:10.1016/j.chiabu.2015.11.016

Licht, C. M., de Geus, E. J., van Dyck, R., & Penninx, B. W. (2010). Longitudinal evidence for unfavorable effects of antidepressants on heart rate variability. *Biological Psychiatry, 68*(9), 861-868. doi:10.1016/j.biopsych.2010.06.032

Liddell, B. J., Kemp, A. H., Steel, Z., Nickerson, A., Bryant, R. A., Tam, N., . . . Silove, D. (2016). Heart rate variability and the relationship between trauma exposure age, and psychopathology in a post-conflict setting. *BMC Psychiatry, 16*, 1-9. doi:10.1186/s12888-016-0850-5

Linda J. Metzger, S. P. O., Nancy J. Berry, Caryl E. Ahern, Natasha B. Lasko, und Roger K. Pitman. (1999). Physiologic Reactivity to Startling Tones in Women With Posttraumatic Stress Disorder. *Journal of Abnormal Psychology, 108*(2), 347-352.

Loranger, A. W., Janca, A., & Sartorius, N. (1997). *Assessment and diagnosis of personality disorders: The ICD-10 international personality disorder examination (IPDE)*: Cambridge University Press.

Lorenz, T. K., Harte, C. B., Hamilton, L. D., & Meston, C. M. (2012). Evidence for a curvilinear relationship between sympathetic nervous system activation and women's physiological sexual arousal. *Psychophysiology, 49*(1), 111-117. doi:10.1111/j.1469-8986.2011.01285.x

Lorenz, T. K., Harte, C. B., & Meston, C. M. (2015). Changes in Autonomic Nervous System Activity are Associated with Changes in Sexual Function in Women with a History of Childhood Sexual Abuse. *J Sex Med, 12*(7), 1545-1554. doi:10.1111/jsm.12908

Ludäscher, P., Valerius, G., Stiglmayr, C., Mauchnik, J., Lanius, R. A., Bohus, M., & Schmahl, C. (2009). Pain Sensitivity and neural processing during dissociative states in patients with borderline personality disorder with and without comorbid posttraumatic stress disorder: a pilot study. *J Psychiatry Neurosci, 35*(3), 177-184.

Lutfey, K. E., Link, C. L., Litman, H. J., Rosen, R. C., & McKinlay, J. B. (2008). An examination of the association of abuse (physical, sexual, or emotional) and female sexual dysfunction: results from the Boston Area Community Health Survey. *Fertility and sterility, 90*(4), 957-964. doi:10.1016/j.fertnstert.2007.07.1352

Lyssenko, L., Schmahl, C., Bockhacker, L., Vonderlin, R., Bohus, M., & Kleindienst, N. (2017). Dissociation in Psychiatric Disorders: A Meta-Analysis of Studies Using the Dissociative Experiences Scale. *Am J Psychiatry*, appiajp201717010025. doi:10.1176/appi.ajp.2017.17010025

Mackenzie, G., Blaney, R., Chivers, A., & Vincent, O. (1993). The incidence of child sexual abuse in Northern Ireland. *International journal of epidemiology, 22*(2), 299-305. doi:10.1093/ije/22.2.299

MacMillan, H. L., Fleming, J. E., Streiner, D. L., Lin, E., Boyle, M. H., Jamieson, E., . . . Beardslee, W. R. (2001). Childhood abuse and lifetime psychopathology in a community sample. *American Journal of Psychiatry, 158*(11), 1878-1883. doi:10.1176/appi.ajp.158.11.1878

MacMillan, H. L., Jamieson, E., & Walsh, C. A. (2003). Reported contact with child protection services among those reporting child physical and sexual abuse: results from a community survey. *Child Abuse & Neglect, 27*(12), 1397-1408. doi:10.1016/j.chiabu.2003.06.003

Maercker, A., Beauducel, A., & Schützwohl, M. (2000). Trauma severity and initial reactions as precipitating factors for posttraumatic stress symptoms and chronic dissociation in former political prisoners. *Journal of Traumatic Stress, 13*(4), 651-660. doi:10.1023/A:1007862217298

Maercker, A., Forstmeier, S., Wagner, B., Glaesmer, H., & Brähler, E. (2008). Posttraumatische Belastungsstörungen in Deutschland. *Der Nervenarzt, 79*(5), 577-586. doi:10.1007/s00115-008-2467-5

Maercker, A., & Mehr, A. (2006). What if victims read a newspaper report about their victimization? A study on the relationship to PTSD symptoms in crime victims. *European Psychologist, 11*(2), 137-142.

Maercker, A., Michael, T., Fehm, L., Becker, E. S., & Margraf, J. (2004). Age of traumatisation as a predictor of post-traumatic stress disorder or major depression in young women. *The British Journal of Psychiatry, 184*(6), 482-487. doi:10.1192/bjp.184.6.482

Maercker, A., Mohiyeddini, C., Müller, M., Xie, W., Yang, Z. H., Wang, J., & Müller, J. (2009). Traditional versus modern values, self-perceived interpersonal factors, and posttraumatic stress in Chinese and German crime victims. *Psychology and Psychotherapy: Theory, Research and Practice, 82*(2), 219-232. doi:10.1348/147608308X380769

Maercker, A., & Müller, J. (2004). Social acknowledgment as a victim or survivor: a scale to measure a recovery factor of PTSD. *Journal of Traumatic Stress, 17*(4), 345-351.

Maikovich-Fong, A. K., & Jaffee, S. R. (2010). Sex differences in childhood sexual abuse characteristics and victims' emotional and behavioral problems: Findings from a national sample of youth. *Child Abuse & Neglect, 34*(6), 429-437. doi:10.1016/j.chiabu.2009.10.006

Masten, A. S., & Obradovic, J. (2008). Disaster preparation and recovery: Lessons from research on resilience in human development. *Ecology and Society, 13*(1), 9.

McCauley, J., Kern, D. E., Kolodner, K., Dill, L., Schroeder, A. F., DeChant, H. K., . . . Bass, E. B. (1997). Clinical characteristics of women with a history of

childhood abuse: unhealed wounds. *JAMA, 277*(17), 1362-1368. doi:10.1001/jama.1997.03540410040028

McCutcheon, V. V., Heath, A. C., Nelson, E. C., Bucholz, K. K., Madden, P. A., & Martin, N. G. (2010). Clustering of trauma and associations with single and co-occurring depression and panic attack over twenty years. *Twin Research and Human Genetics, 13*(01), 57-65. doi:10.1375/twin.13.1.57

McHorney, C. A. (1999). Health status assessment methods for adults: Past accomplishments and future challenges 1. *Annual review of public health, 20*(1), 309-335. doi:10.1146/annurev.publhealth.20.1.309

McLeer, S. V., Deblinger, E., Atkins, M. S., Foa, E. B., & Ralphe, D. L. (1988). Post-traumatic stress disorder in sexually abused children. *Journal of the American Academy of Child & Adolescent Psychiatry, 27*(5), 650-654. doi:10.1097/00004583-198809000-00024

McMillen, C., & Zuravin, S. (1997). Attributions of blame and responsibility for child sexual abuse and adult adjustment. *J Interpers Violence, 12*(1), 30-48.

Meagher, M. W., Arnau, R. C., & Rhudy, J. L. (2001). Pain and emotion: effects of affective picture modulation. *Psychosomatic Medicine, 63*(1), 79-90.

Meston, C. M., & Lorenz, T. A. (2013). Physiological stress responses predict sexual functioning and satisfaction differently in women who have and have not been sexually abused in childhood. *Psychological Trauma: Theory, Research, Practice, and Policy, 5*(4), 350-358. doi:10.1037/a0027706

Meston, C. M., Rellini, A. H., & Heiman, J. R. (2006). Women's history of sexual abuse, their sexuality, and sexual self-schemas. *J Consult Clin Psychol, 74*(2), 229-236. doi:10.1037/0022-006x.74.2.229

Michael, A., & O'Keane, V. (2000). Sexual dysfunction in depression. *Hum Psychopharmacol, 15*(5), 337-345. doi:10.1002/1099-1077(200007)15:5

Michael, T., Ehlers, A., Halligan, S., & Clark, D. (2005). Unwanted memories of assault: what intrusion characteristics are associated with PTSD? *Behaviour Research and Therapy, 43*(5), 613-628.

Mills, R., Kisely, S., Alati, R., Strathearn, L., & Najman, J. (2016). Self-reported and agency-notified child sexual abuse in a population-based birth cohort. *J Psychiatr Res, 74*, 87-93. doi:10.1016/j.jpsychires.2015.12.021

Mitani, S., Fujita, M., Sakamoto, S., & Shirakawa, T. (2006). Effect of autogenic training on cardiac autonomic nervous activity in high-risk fire service workers for posttraumatic stress disorder. *Journal of Psychosomatic Research, 60*(5), 439-444. doi:10.1016/j.jpsychores.2005.09.005

Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: trait versus state, and the effects of autogenic training. *Autonomic Neuroscience, 145*(1), 99-103. doi:10.1016/j.autneu.2008.11.010

Moeller-Bertram, T., Keltner, J., & Strigo, I. A. (2012). Pain and post traumatic stress disorder–Review of clinical and experimental evidence. *Neuropharmacology, 62*(2), 586-597.

Möhler, E., Matheis, V., Poustka, L., Marysko, M., Finke, P., Kaufmann, C., . . . Resch, F. (2009). Mothers with a history of abuse tend to show more impulsiveness. *Child Abuse & Neglect, 33*(3), 123-126. doi:10.1016/j.chiabu.2008.06.002

Molnar, B. E., Berkman, L. F., & Buka, S. L. (2001). Psychopathology, childhood sexual abuse and other childhood adversities: relative links to subsequent suicidal behaviour in the US. *Psychol Med, 31*(06), 965-977. doi:10.1017/S0033291701004329Published

Molnar, B. E., Buka, S. L., & Kessler, R. C. (2001). Child sexual abuse and subsequent psychopathology: results from the National Comorbidity Survey. *Am J Public Health, 91*(5), 753-760.

Moon, E., Lee, S.-H., Kim, D.-H., & Hwang, B. (2013). Comparative study of heart rate variability in patients with schizophrenia, bipolar disorder, post-traumatic stress disorder, or major depressive disorder. *Clinical Psychopharmacology and Neuroscience, 11*(2), 137-143.

Mostoufi, S., Godfrey, K. M., Ahumada, S. M., Hossain, N., Song, T., Wright, L. J., . . . Afari, N. (2014). Pain sensitivity in posttraumatic stress disorder and other anxiety disorders: a preliminary case control study. *Annals of general psychiatry, 13*(1), 31. doi:10.1186/s12991-014-0031-1

Mueller, J., Moergeli, H., & Maercker, A. (2008). Disclosure and social acknowledgement as predictors of recovery from posttraumatic stress: A longitudinal study in crime victims. *The Canadian Journal of Psychiatry, 53*(3), 160-168.

Mullen, P. E., & Fergusson, D. M. (1999). *Childhood sexual abuse: An evidence-based perspective* (Vol. 40): Sage Publications.

Mullen, P. E., Martin, J. L., Anderson, J. C., Romans, S. E., & Herbison, G. P. (1996). The long-term impact of the physical, emotional, and sexual abuse of children: A community study. *Child Abuse & Neglect, 20*(1), 7-21. doi:10.1016/0145-2134(95)00112-3

Münzer, A., Fegert, J. M., Ganser, H. G., Loos, S., Witt, A., & Goldbeck, L. (2016). Please tell! Barriers to disclosing sexual victimization and subsequent social support perceived by children and adolescents. *J Interpers Violence, 31*(2), 355-377.

Nash, M. R., Hulsey, T. L., Sexton, M. C., Harralson, T. L., & Lambert, W. (1993). Long-term sequelae of childhood sexual abuse: Perceived family environment, psychopathology, and dissociation. *Journal of Consulting and Clinical Psychology, 61*(2), 276.

Neumann, D. A., Houskamp, B. M., Pollock, V. E., & Briere, J. (1996). The long-term sequelae of childhood sexual abuse in women: A meta-analytic review. *Child Maltreatment, 1*(1), 6-16.

Norte, C. E., Souza, G. G. L., Vilete, L., Marques-Portella, C., Coutinho, E. S. F., Figueira, I., & Volchan, E. (2013). They know their trauma by heart: an assessment of psychophysiological failure to recover in PTSD. *Journal of Affective Disorders, 150*(1), 136-141. doi:10.1016/j.jad.2012.11.039

Nurcombe, B. (2000). Child sexual abuse I: psychopathology. *Australian and New Zealand Journal of Psychiatry, 34*(1), 85-91. doi:10.1046/j.1440-1614.2000.00642.x

O'driscoll, C., & Flanagan, E. (2016). Sexual problems and post-traumatic stress disorder following sexual trauma: A meta-analytic review. *Psychology and Psychotherapy: Theory, Research and Practice, 89*(3), 351-367. doi:10.1111/papt.12077

Öberg, K., Fugl-Meyer, A., & Fugl-Meyer, K. S. (2004). On categorization and quantification of women's sexual dysfunctions: An epidemiological approach. *International Journal of Impotence Research, 16*(3), 261. doi:10.1038/sj.ijir.3901151

Öberg, K., Fugl-Meyer, K. S., & Fugl-Meyer, A. R. (2002). On sexual well-being in sexually abused Swedish women: Epidemiological aspects. *Sexual and Relationship Therapy, 17*(4), 329-341. doi:10.1080/1468199021000017182

Oktedalen, T., Hoffart, A., & Langkaas, T. F. (2015). Trauma-related shame and guilt as time-varying predictors of posttraumatic stress disorder symptoms during imagery exposure and imagery rescripting--A randomized controlled trial. *Psychother Res, 25*(5), 518-532. doi:10.1080/10503307.2014.917217

Ormel, J., Petukhova, M., Chatterji, S., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M. C., . . . De Girolamo, G. (2008). Disability and treatment of specific mental and physical disorders across the world. *The British Journal of Psychiatry, 192*(5), 368-375. doi:10.1192/bjp.bp.107.039107

Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Hu, F. B., Shalev, A. Y., & Pitman, R. K. (2003). Physiologic responses to sudden, loud tones in monozygotic twins discordant for combat exposure: association with posttraumatic stress disorder. *Archives of General Psychiatry, 60*(3), 283-288. doi:10.1001/archpsyc.60.3.283

Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Peri, T., & Pitman, R. K. (2000). De novo conditioning in trauma-exposed individuals with and without posttraumatic stress disorder. *Journal of Abnormal Psychology, 109*(2), 290-298. doi:10.1037/0021-843X.109.2.290

Orr, S. P., Meyerhoff, J. L., Edwards, J. V., & Pitman, R. K. (1998). Heart rate and blood pressure resting levels and responses to generic stressors in Vietnam veterans with posttraumatic stress disorder. *Journal of Traumatic Stress, 11*(1), 155-164. doi:10.1023/A:1024421502881

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2008). *Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis.* Paper presented at the Annual Meeting of the International Society for Traumatic Stress Studies, 14th, Nov, 1998, Washington, DC, US; This article is based on a paper presented at the aforementioned meeting.

Pace, T. W., & Heim, C. M. (2011). A short review on the psychoneuroimmunology of posttraumatic stress disorder: from risk factors to medical comorbidities. *Brain, behavior, and immunity, 25*(1), 6-13. doi:10.1016/j.bbi.2010.10.003

Paolucci, E. O., Genuis, M. L., & Violato, C. (2001). A meta-analysis of the published research on the effects of child sexual abuse. *The Journal of psychology, 135*(1), 17-36. doi:10.1080/00223980109603677

Paris, J. (1994). *Borderline personality disorder: A multidimensional approach*: American Psychiatric Pub.

Pennebaker, J. W., & Seagal, J. D. (1999). Forming a story: The health benefits of narrative. *J Clin Psychol, 55*(10), 1243-1254. doi:10.1002/(SICI)1097-4679(199910)55:10<1243::AID-JCLP6>3.0.CO;2-N

Pereda, N., Guilera, G., Forns, M., & Gómez-Benito, J. (2009). The prevalence of child sexual abuse in community and student samples: A meta-analysis. *Clinical Psychology Review, 29*(4), 328-338. doi:10.1016/j.cpr.2009.02.007

Peri, T., Ben-Shakhar, G., Orr, S. P., & Shalev, A. Y. (2000). Psychophysiologic assessment of aversive conditioning in posttraumatic stress disorder. *Biological Psychiatry, 47*(6), 512-519. doi:10.1016/S0006-3223(99)00144-4

Perkonigg, A., Kessler, R. C., Storz, S., & Wittchen, H. U. (2000). Traumatic events and post-traumatic stress disorder in the community: prevalence,risk factors and comorbidity. *Acta Psychiatrica Scandinavica, 101*(1), 46-59. doi:10.1034/j.1600-0447.2000.101001046.x

Pfeiffer, C., Wetzels, P., & Enzmann, D. (1999). *Innerfamiliäre Gewalt gegen Kinder und Jugendliche und ihre Auswirkungen*: KFN.

Pieritz, K., Rief, W., & Euteneuer, F. (2015). Childhood adversities and laboratory pain perception. *Neuropsychiatr Dis Treat, 11*, 2109-2116. doi:10.2147/ndt.s87703

Pierrehumbert, B., Torrisi, R., Glatz, N., Dimitrova, N., Heinrichs, M., & Halfon, O. (2009). The influence of attachment on perceived stress and cortisol response to acute stress in women sexually abused in childhood or adolescence. *Psychoneuroendocrinology, 34*(6), 924-938. doi:10.1016/j.psyneuen.2009.01.006

Pineles, S. L., Suvak, M. K., Liverant, G. I., Gregor, K., Wisco, B. E., Pitman, R. K., & Orr, S. P. (2013). Psychophysiologic reactivity, subjective distress, and their associations with PTSD diagnosis. *J Abnorm Psychol, 122*(3), 635-644. doi:10.1037/a0033942

Pitman, R. K., Orr, S. P., van der Kolk, B. A., Greenberg, M. S., Meyerhoff, J. L., & Mougey, E. H. (1990). Analgesia: A new dependent variable for the biological study of posttraumatic stress disorder.

Pitman, R. K., Van der Kolk, B. A., Orr, S. P., & Greenberg, M. S. (1990). Naloxone-reversible analgesic response to combat-related stimuli in posttraumatic stress disorder: a pilot study. *Archives of General Psychiatry, 47*(6), 541-544.

Polusny, M. A., & Follette, V. M. (1995). Long-term correlates of child sexual abuse: Theory and review of the empirical literature. *Applied and Preventive Psychology, 4*(3), 143-166. doi:10.1016/S0962-1849(05)80055-1

Porges, S. W., Doussard-Roosevelt, J. A., Portales, A. L., & Greenspan, S. I. (1996). Infant regulation of the vagal “brake” predicts child behavior problems: A psychobiological model of social behavior. *Developmental psychobiology, 29*(8), 697-712. doi:10.1002/(SICI)1098-2302(199612)29:8<697::AID-DEV5>3.0.CO;2-O

Priebe, G., & Svedin, C. G. (2008). Child sexual abuse is largely hidden from the adult society: An epidemiological study of adolescents’ disclosures. *Child Abuse & Neglect, 32*(12), 1095-1108. doi:10.1016/j.chiabu.2008.04.001

Priebe, K., Kleindienst, N., Zimmer, J., Koudela, S., Ebner-Priemer, U., & Bohus, M. (2013). Frequency of intrusions and flashbacks in patients with posttraumatic stress disorder related to childhood sexual abuse: An electronic diary study. *Psychol Assess, 25*(4), 1370.

Rabellino, D., D'andrea, W., Siegle, G., Frewen, P. A., Minshew, R., Densmore, M., . . . Lanius, R. A. (2017). Neural correlates of heart rate variability in PTSD during sub-and supraliminal processing of trauma-related cues. *Human Brain Mapping, 38*(10), 4898-4907. doi:10.1002/hbm.23702

Rapaport, M. H., Clary, C., Fayyad, R., & Endicott, J. . (2005). Quality-of-life impairment in depressive and anxiety disorders. *American Journal of Psychiatry, 162*(6), 1171-1178. doi:10.1176/appi.ajp.162.6.1171

Rausch, S., Herzog, J., Thome, J., Ludäscher, P., Müller-Engelmann, M., Steil, R., . . . Kleindienst, N. (2016). Women with exposure to childhood interpersonal violence without psychiatric diagnoses show no signs of impairment in general functioning, quality of life and sexuality. *Borderline Personality Disorder and Emotion Dysregulation, 3*(1), 13. doi:10.1186/s40479-016-0048-y

Regehr, C., LeBlanc, V., Jelley, R. B., Barath, I., & Daciuk, J. (2007). Previous trauma exposure and PTSD symptoms as predictors of subjective and biological response to stress. *Can J Psychiatry, 52*(10), 675-683. doi:10.1177/070674370705201008

Reinhardt, T., Kleindienst, N., Treede, R.-D., Bohus, M., & Schmahl, C. (2013). Individual Modulation of Pain Sensitivity under Stress. *Pain Medicine, 14*(5), 676-685. doi:10.1111/pme.12090

Reinhardt, T., Schmahl, C., Wüst, S., & Bohus, M. (2012). Salivary cortisol, heart rate, electrodermal activity and subjective stress responses to the Mannheim Multicomponent Stress Test (MMST). *Psychiatry Research, 198*(1), 106-111. doi:10.1016/j.psychres.2011.12.009

Rellini, A., & Meston, C. (2007). Sexual Function and Satisfaction in Adults Based on the Definition of Child Sexual Abuse. *J Sex Med, 4*(5), 1312-1321. doi:10.1111/j.1743-6109.2007.00573.x

Rellini, A., & Meston, C. M. (2006). Psychophysiological sexual arousal in women with a history of child sexual abuse. *Journal of Sex & Marital Therapy, 32*(1), 5-22. doi:10.1080/00926230500229145

Rhudy, J. L., & Meagher, M. W. (2000). Fear and anxiety: divergent effects on human pain thresholds. *Pain, 84*(1), 65-75.

Rieckert, J., & Möller, A. T. (2000). Rational-emotive behavior therapy in the treatment of adult victims of childhood sexual abuse. *Journal of Rational-Emotive & Cognitive-Behavior Therapy, 18*(2), 87-101. doi:10.1023/A:1007824719770

Rinne, T., Westenberg, H. G., den Boer, J. A., & van den Brink, W. (2000). Serotonergic blunting to meta-chlorophenylpiperazine (m-CPP) highly correlates with sustained childhood abuse in impulsive and autoaggressive female borderline patients. *Biological Psychiatry, 47*(6), 548-556. doi:10.1016/S0006-3223(99)00181-X

Roberts, B. W., Walton, K. E., & Viechtbauer, W. (2006). Patterns of mean-level change in personality traits across the life course: a meta-analysis of longitudinal studies. *Psychol Bull, 132*(1), 1-25. doi:10.1037/0033-2909.132.1.1

Rodriguez-Srednicki, O. (2002). Childhood sexual abuse, dissociation, and adult self-destructive behavior. *Journal of Child Sexual Abuse, 10*(3), 75-89. doi:10.1300/J070v10n03_05

Rom, O., & Reznick, A. Z. (2016). The Stress Reaction: A Historical Perspective. *Adv Exp Med Biol, 905*, 1-4. doi:10.1007/5584_2015_195

Romans, S. E., Gendall, K. A., Martin, J. L., & Mullen, P. E. (2001). Child sexual abuse and later disordered eating: A New Zealand epidemiological study. *International Journal of Eating Disorders, 29*(4), 380-392. doi:10.1002/eat.1034

Rorty, M., Yager, J., & Rossotto, E. (1994). Childhood Sexual, Physical, and Psychological Abuse. *Am J Psychiatry, 151*, 1123. doi:10.1176/ajp.151.8.1122

Rosen, R. C., & Bachmann, G. A. (2008). Sexual well-being, happiness, and satisfaction, in women: The case for a new conceptual paradigm. *Journal of Sex & Marital Therapy, 34*(4), 291-297. doi:10.1080/00926230802096234

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: University Press.

Roy, A., Carli, V., & Sarchiapone, M. (2011). Resilience mitigates the suicide risk associated with childhood trauma. *Journal of Affective Disorders, 133*(3), 591-594. doi:10.1016/j.jad.2011.05.006

Ruscheweyh, R., Stumpenhorst, F., Knecht, S., & Marziniak, M. (2010). Comparison of the cold pressor test and contact thermode-delivered cold stimuli for the assessment of cold pain sensitivity. *The Journal of Pain, 11*(8), 728-736. doi:10.1016/j.jpain.2009.10.016

Rutter, M. (1985). Resilience in the face of adversity. Protective factors and resistance to psychiatric disorder. *The British Journal of Psychiatry, 147*(6), 598-611. doi:10.1192/bjp.147.6.598

Rutter, M. (1987). Continuities and discontinuities from infancy.

Rutter, M. (2006). Implications of resilience concepts for scientific understanding. *Ann N Y Acad Sci, 1094*(1), 1-12. doi:10.1196/annals.1376.002

Sachs-Ericsson, N., Blazer, D., Plant, E. A., & Arnow, B. (2005). Childhood sexual and physical abuse and the 1-year prevalence of medical problems in the National Comorbidity Survey. *Health Psychology, 24*(1), 32. doi:10.1037/0278-6133.24.1.32

Sammito, S., Thielmann, B., Zimmermann, P., & Böckelmann, I. (2015). Einfluss einer posttraumatischen Belastungsstörung auf die Herzfrequenzvariabilität als Marker des autonomen Nervensystems–eine systematische Literaturübersicht. *Fortschritte der Neurologie· Psychiatrie, 83*(01), 30-37.

Sanders, B., & Giolas, MH (1991). Dissociation and childhood trauma in psychologically disturbed adolescents. *The American Journal of Geriatric Psychiatry, 148*(1), 50-54.

Scarinci, I. C., McDonald-Haile, J., Bradley, L. A., & Richter, J. E. (1994). Altered pain perception and psychosocial features among women with gastrointestinal disorders and history of abuse: a preliminary model. *The American journal of medicine, 97*(2), 108-118. doi:10.1016/0002-9343(94)90020-5

Schaeffer, N. C. (2000). Asking questions about threatening topics: A selective overview. *The science of self-report: Implications for research and practice*, 105-121.

Schauer, M., & Elbert, T. (2010). Dissociation Following Traumatic Stress. *Zeitschrift für Psychologie / Journal of Psychology, 218*(2), 109-127. doi:10.1027/0044-3409/a000018

Schauer, M., Schauer, M., Neuner, F., & Elbert, T. (2011). *Narrative exposure therapy: A short-term treatment for traumatic stress disorders*: Hogrefe Publishing.

Scheeringa, M. S., Zeanah, C. H., Myers, L., & Putnam, F. (2004). Heart period and variability findings in preschool children with posttraumatic stress symptoms. *Biological Psychiatry, 55*(7), 685-691. doi:10.1016/j.biopsych.2004.01.006

Scheiderer, E. M., Wood, P. K., & Trull, T. J. (2015). The comorbidity of borderline personality disorder and posttraumatic stress disorder: revisiting the prevalence and associations in a general population sample. *Borderline Personality Disorder and Emotion Dysregulation, 2*(1), 1-16. doi:10.1186/s40479-015-0032-y

Schmahl, C., Meinzer, M., Zeuch, A., Fichter, M., Cebulla, M., Kleindienst, N., . . . Bohus, M. (2010). Pain sensitivity is reduced in borderline personality disorder, but not in posttraumatic stress disorder and bulimia nervosa. *The World Journal of Biological Psychiatry, 11*(2-2), 364-371. doi:10.3109/15622970701849952

Schore, J. R., & Schore, A. N. (2008). Modern attachment theory: The central role of affect regulation in development and treatment. *Clinical Social Work Journal, 36*(1), 9-20. doi:10.1007/s10615-007-0111-7

Schuhmacher, J., Leppert, K., Gunzelmann, T., Strauß, B., & Brähler, E. (2005). Die Resilienzskala- Ein Fragebogen zur Erfassung der psychischen Widerstandsfähigkeit als Personmerkmal. *Zeitschrift für Klinische Psychologie, Psychiatrie und Psychotherapie, 53*(1), 16-39.

Schwartz, S. H. (1994). *Beyond individualism/collectivism: New cultural dimensions of values*: Sage Publications, Inc.

Selye, H. (1973). The Evolution of the Stress Concept: The originator of the concept traces its development from the discovery in 1936 of the alarm reaction to modern therapeutic applications of syntoxic and catatoxic hormones. *American scientist, 61*(6), 692-699.

Shah, A. J., Lampert, R., Goldberg, J., Veledar, E., Bremner, J. D., & Vaccarino, V. (2013). Posttraumatic stress disorder and impaired autonomic modulation in male twins. *Biological Psychiatry, 73*(11), 1103-1110. doi:10.1016/j.biopsych.2013.01.019

Shalev, A. Y., Orr, S. P., Peri, T., Schreiber, S., & Pitman, R. K. (1992). Physiologic responses to loud tones in Israeli patients with posttraumatic stress disorder. *Archives of General Psychiatry, 49*(11), 870-875. doi:10.1001/archpsyc.1992.01820110034005

Shalev, A. Y., Peri, T., Orr, S. P., Bonne, O., & Pitman, R. K. (1997). Auditory startle responses in help-seeking trauma survivors. *Psychiatry Research, 69*(1), 1-7. doi:10.1016/S0165-1781(96)03001-6

Shevlin, M., Hyland, P., Karatzias, T., Fyvie, C., Roberts, N., Bisson, J. I., . . . Cloitre, M. (2017). Alternative models of disorders of traumatic stress based on the new ICD-11 proposals. *Acta Psychiatr Scand, 135*(5), 419-428. doi:10.1111/acps.12695

Shields, M. E., Hovdestad, W. E., Pelletier, C., Dykxhoorn, J. L., O'Donnell, S. C., & Tonmyr, L. (2016). Childhood maltreatment as a risk factor for diabetes: findings from a population-based survey of Canadian adults. *BMC Public Health, 16*(1), 879. doi:10.1186/s12889-016-3491-1

Shipman, K., Edwards, A., Brown, A., Swisher, L., & Jennings, E. (2005). Managing emotion in a maltreating context: A pilot study examining child neglect. *Child Abuse & Neglect, 29*(9), 1015-1029. doi:10.1016/j.chiabu.2005.01.006

Shirk, S., Burwell, R., & Harter, S. (2003). Strategies to modify low self-esteem in adolescents. *Cognitive therapy with children and adolescents: A casebook for clinical practice, 2*, 189-213.

Sippel, L. M., Pietrzak, R. H., Charney, D. S., Mayes, L. C., & Southwick, S. M. (2015). How does social support enhance resilience in the trauma-exposed individual? *Ecology and Society, 20*(4).

Soler, L., Kirchner, T., Paretilla, C., & Forns, M. (2013). Impact of poly-victimization on mental health: the mediator and/or moderator role of self-esteem. *J Interpers Violence, 28*(14), 2695-2712. doi:10.1177/0886260513487989

Spataro, J., Mullen, P. E., Burgess, P. M., Wells, D. L., & Moss, S. A. (2004). Impact of child sexual abuse on mental health. *The British Journal of Psychiatry, 184*(5), 416-421. doi:10.1192/bjp.184.5.416

Springs, F. E., & Friedrich, W. N. (1992). *Health risk behaviors and medical sequelae of childhood sexual abuse.* Paper presented at the Mayo Clinic Proceedings.

Stauss, H. M. (2003). Heart rate variability. *Am J Physiol Regul Integr Comp Physiol, 285*(5), R927-931. doi:10.1152/ajpregu.00452.2003

Steil, R., & Ehlers, A. (2000). Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. *Behaviour Research and Therapy, 38*(6), 537-558. doi:10.1016/S0005-7967(99)00069-8

Stein, D. J., Koenen, K. C., Friedman, M. J., Hill, E., McLaughlin, K. A., Petukhova, M., . . . Kessler, R. C. (2013). Dissociation in posttraumatic stress disorder: evidence from the world mental health surveys. *Biol Psychiatry, 73*(4), 302-312. doi:10.1016/j.biopsych.2012.08.022

Stein, J. A., Golding, J. M., Siegel, J. M., Burnam, M. A., & Sorenson, S. B. (1988). Long-term psychological sequelae of child sexual abuse: The Los Angeles Epidemiologic Catchment Area study.

Stein, M. B., Walker, J. R., Hazen, A. L., & Forde, D. R. (1997). Full and partial posttraumatic stress disorder: findings from a community survey. *American Journal of Psychiatry, 154*(8), 1114-1119.

Steiner, H., Carrion, V., Plattner, B., & Koopman, C. (2003). Dissociative symptoms in posttraumatic stress disorder: diagnosis and treatment. *Child Adolesc Psychiatr Clin N Am, 12*(2), 231-249, viii.

Stephenson, K. R., Pulverman, C. S., & Meston, C. M. (2014). Assessing the association between childhood sexual abuse and adult sexual experiences in women with sexual difficulties. *Journal of Traumatic Stress, 27*(3), 274-282. doi:10.1002/jts.21923

Stiglmayr, C., Schmahl, C., Bremner, J. D., Bohus, M., & Ebner-Priemer, U. (2009). Development and Psychometric Characteristics of the DSS-4 as a Short Instrument to Assess Dissociative Experience during Neuropsychological Experiments. *Psychopathology, 42*(6), 370-374.

Stoltenborgh, M., van IJzendoorn, M. H., Euser, E. M., & Bakermans-Kranenburg, M. J. (2011). A Global Perspective on Child Sexual Abuse: Meta-Analysis of Prevalence Around the World. *Child Maltreatment, 16*(2), 79-101. doi:10.1177/1077559511403920

Strigo, I. A., Simmons, A. N., Matthews, S. C., Grimes, E. M., Allard, C. B., Reinhardt, L. E., . . . Stein, M. B. (2010). Neural Correlates of Altered Pain Response in Women with Posttraumatic Stress Disorder from Intimate Partner Violence. *Biological Psychiatry, 68*(5), 442-450. doi:10.1016/j.biopsych.2010.03.034

Sullivan, M. J. (2012). The communal coping model of pain catastrophising: Clinical and research implications. *Canadian Psychology/Psychologie canadienne, 53*(1), 32. doi:10.1037/a0026726

Surís, A., Lind, L., Kashner, T. M., & Borman, P. D. . (2007). Mental health, quality of life, and health functioning in women veterans differential outcomes associated with military and civilian sexual as.

Sutker, P. B., Davis, J. M., Uddo, M., & Ditta, S. R. (1995). War zone stress, personal resources, and PTSD in Persian Gulf War returnees. *Journal of Abnormal Psychology, 104*(3), 444-452.

Tan, G., Dao, T. K., Farmer, L., Sutherland, R. J., & Gevirtz, R. (2011). Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): A pilot study. *Applied psychophysiology and biofeedback, 36*(1), 27-35. doi:10.1007/s10484-010-9141-y

Terr, L. C. (2003). Childhood traumas: An outline and overview. *Focus, 1*(3), 322-334.

Thayer, J. F., Åhs, F., Fredrikson, M., Sollers, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews, 36*(2), 747-756. doi:10.1016/j.neubiorev.2011.11.009

Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: looking up and down from the brain. *Psychoneuroendocrinology, 30*(10), 1050-1058. doi:10.1016/j.psyneuen.2005.04.014

The EuroQol Group. (1990). EuroQol - a new facility for the measurement of health-related quality of life. *Health Policy, 16*(3), 199-208. doi:10.1016/0168-8510(90)90421-9

The WHOQOL Group. (1998). The World Health Organization quality of life assessment (WHOQOL): development and general psychometric properties. *Social Science & Medicine, 46*(12), 1569-1585. doi:10.1016/S0277-9536(98)00009-4

Thome, J., Hauschild, S., Koppe, G., Liebke, L., Rausch, S., Herzog, J. I., . . . Hermans, D. (2017). Generalisation of fear in PTSD related to prolonged childhood maltreatment: an experimental study. *Psychol Med*, 1-12. doi:10.1017/S0033291717003713

Tolin, D. F., & Foa, E. B. (2006). Sex differences in trauma and posttraumatic stress disorder: A quantitative review of 25 years of research. *Psychological Bulletin, 132*(6), 959-992. doi:10.1037/0033-2909.132.6.959

Tran, J. K., Dunckel, G., & Teng, E. J. (2015). Sexual dysfunction in veterans with post-traumatic stress disorder. *J Sex Med, 12*(4), 847-855. doi:10.1111/jsm.12823

Tsur, N., Defrin, R., & Ginzburg, K. (2017). Posttraumatic Stress Disorder, Orientation to Pain, and Pain Perception in Ex-Prisoners of War Who Underwent Torture. *Psychosom Med, 79*(6), 655-663. doi:10.1097/psy.0000000000000461

Tsur, N., Defrin, R., Lahav, Y., & Solomon, Z. (2018). The traumatized body: Long-term PTSD and its implications for the orientation towards bodily signals. *Psychiatry Res, 261*, 281-289. doi:10.1016/j.psychres.2017.12.083

Turk, D. C., & Gatchel, R. J. (2013). *Psychological approaches to pain management: a practitioner's handbook*: Guilford publications.

Turner, H. A., Finkelhor, D., & Ormrod, R. (2010). The effects of adolescent victimization on self-concept and depressive symptoms. *Child Maltreatment, 15*(1), 76-90. doi:10.1177/1077559509349444

Ullman, S. E. (2007). Relationship to perpetrator, disclosure, social reactions, and PTSD symptoms in child sexual abuse survivors. *J Child Sex Abus, 16*(1), 19-36. doi:10.1300/J070v16n01_02

Ullman, S. E., & Filipas, H. H. (2005). Gender differences in social reactions to abuse disclosures, post-abuse coping, and PTSD of child sexual abuse survivors. *Child Abuse Negl, 29*(7), 767-782. doi:10.1016/j.chiabu.2005.01.005

Ullman, S. E., & Relyea, M. (2016). Social support, coping, and posttraumatic stress symptoms in female sexual assault survivors: a longitudinal analysis. *Journal of Traumatic Stress, 29*(6), 500-506. doi:10.1002/jts.22143

Vaillancourt-Morel, M. P., Godbout, N., Labadie, C., Runtz, M., Lussier, Y., & Sabourin, S. (2015). Avoidant and compulsive sexual behaviors in male and female survivors of childhood sexual abuse. *Child Abuse Negl, 40*, 48-59. doi:10.1016/j.chiabu.2014.10.024

Valle, L. A., & Silovsky, J. F. (2002). Attributions and adjustment following child sexual and physical abuse. *Child Maltreatment, 7*(1), 9-24.

Van Berlo, W., & Ensink, B. (2000). Problems with sexuality after sexual assault. *Annual review of sex research, 11*(1), 235-257.

van der Kolk, B. A., Roth, S., Pelcovitz, D., Sunday, S., & Spinazzola, J. (2005). Disorders of extreme stress: The empirical foundation of a complex adaptation to trauma. *Journal of Traumatic Stress, 18*(5), 389-399. doi:10.1002/jts.20047

van Dijk, J. A., Schoutrop, M. J., & Spinhoven, P. (2003). Testimony therapy: treatment method for traumatized victims of organized violence. *Am J Psychother, 57*(3), 361-373.

Van Houdenhove, B., Neerinckx, E., Lysens, R., Vertommen, H., Van Houdenhove, L., Onghena, P., . . . D'Hooghe, M.-B. (2001). Victimization in chronic fatigue

syndrome and fibromyalgia in tertiary care: a controlled study on prevalence and characteristics. *Psychosomatics, 42*(1), 21-28. doi:10.1176/appi.psy.42.1.21

van IJzendoorn, M., Prinzie, P., Euser, E., Groeneveld, M., Brillenslijper-Kater, S., van Noort-van der Linden, A., . . . Klein Velderman, M. (2007). Kindermishandeling in Nederland in Nederland anno 2005: de Nationale Prevalentiestudie Mishandeling van Kinderen en Jeugdigen (NPM-2005) Leiden Attachment Research program.

Vonderlin, R., Kleindienst, N., Alpers, G. W., Bohus, M., Lyssenko, L., & Schmahl, C. (2018). Dissociation in victims of childhood abuse or neglect: a meta-analytic review. *Psychol Med*, 1-10. doi:10.1017/s0033291718000740

Wagnild, G. M., & Young, H. M. (1993). Development and Psychometric Evaluation of the Resilience Scale. *Journal of nursing measurement, 1*(2), 165-178.

Wahbeh, H., & Oken, B. (2013). Peak high-frequency HRV and peak alpha frequency higher in PTSD. *Applied psychophysiology and biofeedback, 38*(1), 57-69. doi:10.1007/s10484-012-9208-z

Walker, C. E., Bonner, B. L., & Kaufman, K. L. (1988). *The physically and sexually abused child: Evaluation and treatment*: Pergamon Press.

Weathers, F., Blake, D., Schnurr, P., Kaloupek, D., Marx, B., & Keane, T. (2013). The clinician-administered PTSD scale for DSM-5 (CAPS-5). *Interview available from the National Center for PTSD at www. ptsd. va. gov.*

Webb, A. K., Vincent, A. L., Jin, A. B., & Pollack, M. H. (2015). Physiological reactivity to nonideographic virtual reality stimuli in veterans with and without PTSD. *Brain and Behavior, 5*(2), n/a-n/a. doi:10.1002/brb3.304

Webster, R. E. (2001). Symptoms and long-term outcomes for children who have been sexually assaulted. *Psychology in the Schools, 38*(6), 533-547. doi:10.1002/pits.1041

Weiss, D. S., & Marmar, C. R. (1996). *The Impact of Events Scale - Revised*. New York: Guilford Press.

Werner, E. E. (1993). Risk, resilience, and recovery: Perspectives from the Kauai Longitudinal Study. *Development and Psychopathology, 5*(4), 503-515. doi:10.1017/S095457940000612X

Wetzels, P. (1997). *Zur Epidemiologie physischer und sexueller Gewalterfahrungen in der Kindheit: Ergebnisse einer repräsentativen retrospektiven Prävalenzstudie für die BRD*: KFN.

Whiffen, V. E., & MacIntosh, H. B. (2005). Mediators of the link between childhood sexual abuse and emotional distress a critical review. *Trauma, Violence, & Abuse, 6*(1), 24-39. doi:10.1177/1524838004272543

Williams, L. (1994). Recall of Childhood Trauma: A Prospective Study of Women's Memories of Child Sexual Abuse. *Journal of Consulting and Clinical Psychology, 62*(6), 1167-1176.

Williams, L. M. (1994). Recall of childhood trauma: a prospective study of women's memories of child sexual abuse. *Journal of Consulting and Clinical Psychology, 62*(6), 1167.

Wise, L. A., Zierler, S., Krieger, N., & Harlow, B. L. (2001). Adult onset of major depressive disorder in relation to early life violent victimisation: a case-control study. *The Lancet, 358*(9285), 881-887. doi:10.1016/S0140-6736(01)06072-X

Wittchen, W. (1997). Gruschwitz, & Zaudig (1997) H.-U. Wittchen, U. Wunderlich, S. Gruschwitz, M. Zaudig. *SKID I. Strukturiertes Klinisches Interview für DSM-IV. Achse I: Psychische Störungen. Interviewheft und Beurteilungsheft. Eine*

deutschsprachige, erweiterte Bearbeitung der amerikanischen Originalversion des SCID I, Hogrefe, Göttingen.

Wolf, E. J., & Martin, E. (2008). Structural equation modeling of associations among combat exposure, PTSD symptom factors, and global assessment of functioning. *Journal of rehabilitation research and development, 45*(3), 359. doi:10.1682/JRRD.2007.06.0085

Wolf, E. J., Miller, M. W., Reardon, A. F., Ryabchenko, K. A., Castillo, D., & Freund, R. (2012). A latent class analysis of dissociation and posttraumatic stress disorder: Evidence for a dissociative subtype. *Archives of General Psychiatry, 69*(7), 698-705. doi:10.1001/archgenpsychiatry.2011.1574

Wolfe, D. A., Sas, L., & Wekerle, C. (1994). Factors associated with the development of posttraumatic stress disorder among child victims of sexual abuse. *Child Abuse & Neglect, 18*(1), 37-50.

World Health Organization. (1987). Working group on concepts of sexual health. EURO, Copenhagen.

World Health Organization. (1995). The World Health Organization Quality of Life Assessment. Field Trial Version for Adults. Administration Manual. *World Health Organization, Geneva*.

Wyatt, G. E., & Peters, S. D. (1986). Issues in the definition of child sexual abuse in prevalence research. *Child Abuse & Neglect, 10*(2), 231-240. doi:https://doi.org/10.1016/0145-2134(86)90084-0

Zanarini, M. C. (2000). Childhood experiences associated with the development of borderline personality disorder. *Psychiatric Clinics of North America, 23*(1), 89-101. doi:10.1016/S0193-953X(05)70145-3

Zanarini, M. C., Frankenburg, F. R., Reich, D. B., & Fitzmaurice, G. M. (2016). Fluidity of the Subsyndromal Phenomenology of Borderline Personality Disorder Over 16 Years of Prospective Follow-Up. *Am J Psychiatry, 173*(7), 688-694. doi:10.1176/appi.ajp.2015.15081045

Zoladz, P. R., & Diamond, D. M. (2013). Current status on behavioral and biological markers of PTSD: a search for clarity in a conflicting literature. *Neuroscience & Biobehavioral Reviews, 37*(5), 860-895. doi:10.1016/j.neubiorev.2013.03.024

7 Publications

- **Rausch, S.,** Herzog, J., Thome, J., Ludäscher, P., Müller-Engelmann, M., Steil, R., Priebe, K., Fydrich, T., & Kleindienst, N. (2016). Women with exposure to childhood interpersonal violence without mental disorders show no signs of impairment in general functioning, quality of life and sexuality. *Borderline Personality Disorder and Emotion Dysregulation*, 3(1), 13. doi:10.1186/s40479-016-0048-y
- Bornefeld-Ettmann, P., Steil, R., Lieberz, K. A., Bohus, M., **Rausch, S.**, Herzog, J., Priebe, K., Fydrich, T., & Müller-Engelmann, M. (2018). Sexual Functioning After Childhood Abuse: The Influence of Post-Traumatic Stress Disorder and Trauma Exposure. *The journal of sexual medicine*, 15(4), 529-538. doi. 10.1016/j.jsxm.2018.02.016
- Lieberz, K., Mueller-Engelmann, M., Bornefeld-Ettmann, P., Priebe, K., Weidmann, A., Fydrich, T., Geniole, S., McCormick, S., **Rausch, S.**, Thome, J., & Steil, R. (2018). Detecting implicit cues of aggressiveness in male faces in revictimized female PTSD patients and healthy controls. *Psychiatry Research*, 267, 429-437. doi: 10.1016/j.psychres.2018.05.061
- Thome, J., Hauschild, S., Koppe, G., Liebke, L., **Rausch, S.,** Herzog, J., . . . Lis, S. (2017). Generalisation of fear in PTSD related to prolonged childhood maltreatment: An experimental study. *Psychological Medicine*, 1-12. doi:10.1017/S0033291717003713
- Dittmann, C., Müller-Engelmann, M., Stangier, U., Priebe, K., Fydrich, T., Görg, N., **Rausch, S**., Resick, P., & Steil, R. (2017). Disorder-and Treatment-Specific Therapeutic Competence Scales for Posttraumatic Stress Disorder Intervention: Development and Psychometric Properties. *Journal of Traumatic Stress.* doi:10.1002/jts.22236
- Herzog, J., Niedtfeld, I., **Rausch, S.**, Thome, J., Müller-Engelmann, M., Steil, R., Priebe, K., Bohus, M. & Schmahl, C. (2017). Increased recruitment of cognitive control in the presence of traumatic stimuli in complex PTSD *European Archives of Psychiatry and Clinical Neuroscience.* doi: 10.1007/s00406-017-0822-x
- Bornefeld-Ettmann, P., Steil, R., Höfling, V., Weßlau, C., Lieberz, K. A., **Rausch, S.**, Priebe, K., Fydrich, T., & Müller-Engelmann, M. (2017). Validation of the

German Version of the Sexual Self-Esteem Inventory for Women and its Application in a Sample of Sexually and Physically Abused Women. *Sex Roles,* 1-14. doi: 10.1007/s11199-017-0849-5

- Wieser, M. J., Miskovic, V., **Rausch, S.**, & Keil, A. (2014). Different time course of visuocortical signal changes to fear-conditioned faces with direct or averted gaze: A ssVEP study with single-trial analysis. *Neuropsychologia*, *62*, 101-110.

Congress Contributions (selected):

- **Rausch, S.**, Priebe, K., Ludäscher, P., Thome, J., Herzog, J., Kleindienst, N., Schmahl, & Bohus, M. (2016). Dissociation and pain sensitivity after childhood abuse in adults with and without Posttraumatic Stress Disorder. 4th Borderline Congress (ESSPD), Vienna, Austria.- *Talk*
- **Rausch, S.**, Priebe, K., Ludäscher, P., Thome, J., Herzog, J., Kleindienst, N., Schmahl, & Bohus, M. (2016). Psychopathological and psychophysiological sequelae of childhood abuse in women without mental disorders. 5th BMBF Netzwerktreffen, Mannheim, Germany. – *Poster presentation*
- **Rausch, S.**, Priebe, K., Ludäscher, P., Thome, J., Herzog, J., Kleindienst, N., Schmahl, & Bohus, M. (2015). Dissociation and pain sensitivity after childhood abuse in adults with and without Posttraumatic Stress Disorder. 4th BMBF Netzwerktreffen, Berlin, Germany. - *Talk*
- **Rausch, S.**, Priebe, K., Ludäscher, P., Thome, J., Herzog, J., Kleindienst, N., Schmahl, & Bohus, M. (2015). Dissociation and pain sensitivity after childhood abuse in adults with and without Posttraumatic Stress Disorder. International Society for Traumatic Stress Studies, New Orleans, USA. – *Poster presentation*
- **Rausch, S.**, Steil, R., Priebe, K., Ludäscher, P., Schmahl, C., Fydrich, T., & Bohus, M. (2014). Treating psychosocial and neural consequences of childhood abuse in adults. 3rd Borderline Congress (ESSPD), Rome, Italy.- *Talk*

8 List of Tables

9 List of Figures

10 Curriculum Vitae

PERSONAL DETAILS

Name:	Sophie Christina Rausch
Date of birth:	November 13th, 1988
Place of birth:	Seeheim-Jugenheim, Germany
Marital status:	Married
Father:	Georg Rausch
Mother:	Hildegard Rausch

EDUCATION AND ACADEMIC CAREER

1995 - 1999	Hinkelstein Elementary School, Alsbach-Hähnlein
1999 - 2008	Altes Kurfürstliches Gymnasium, Bensheim
06.06. 2008	Abitur (Grade: 1.8)
2009 - 2010	Undergraduate Studies in Psychology Julius-Maximilians-University Würzburg
19.08.2010	Undergraduate Certificate, Grade: 2.32
2010 - 2012	Graduate Studies and Diploma in Psychology Julius-Maximilians-University Würzburg
2012 – 2013	Center for the Studies of Emotion and Attention, University of Florida, Gainesville, USA Diploma Thesis: The role of Eye-Gaze in an Aversive Conditioning Paradigm - A Study with Cortical Steady-state Visual Evoked Potentials, Grade: 1.3
06.08.2013	Diploma Certificate in Psychology, Grade: 1.43
2013 - 2016	Postgraduate training and research assistant at the Central Institute of Mental Health, Mannheim, Medical Faculty Mannheim, Ruprecht- Karls-University Heidelberg
2013 - 2018	Postgraduate training at the Zentrum für Psychologische Psychotherapie Mannheim, certified as Psychologische Psychotherapeutin, Grade: 1.33

11 Acknowledgements

Allen voran gilt mein Dank meinem Doktorvater Prof. Dr. Martin Bohus für seine Kreativität und seine wegweisenden wissenschaftlichen Anregungen. Seine Begeisterung für die Forschung war stets ansteckend und motivierend. Ohne ihn wären die Studien, die in dieser Arbeit vorgestellt werden, nicht möglich gewesen. Bedanken möchte ich mich auch für die Möglichkeit, Erfahrungen auf nationalen und internationalen Kongressen, sowie psychotherapeutische Erfahrungen im stationären und ambulanten Kontext gesammelt haben zu dürfen.
Ebenfalls herzlich bedanken möchte ich mich bei Prof. Dr. Christian Schmahl für die Unterstützung meiner Promotion im Rahmen des Graduiertenprogrammes. Ein großes Dankeschön geht an Dr. Nikolaus Kleindienst und Dr. Petra Ludäscher für die Hilfe in allen methodische Fragen, die vielen Denkanstöße und ihre wertschätzende Haltung. Vielen Dank an Dr. Stefanie Lis für die Programmierung des Experiments. Mein ganz besonderer Dank gilt meiner Mentorin Dr. Inga Niedfeldt. Liebe Inga, danke für deine großartige Unterstützung in jeglichen Bereichen. Du bist der Hit!!!
Nicht weniger ans Herz gewachsen sind mir meine lieben Kollegen aus C4. Danke für all die schönen Mittagspausen und Gelegenheiten, den Kopf zwischendurch wieder frei zu bekommen. Insbesondere danke ich meiner Dissertation Group Nora, Caro & Juli für die emotionale Unterstützung sowie Nora für die gemeinsame Zeit im Spaß-Büro und konstruktives Feedback.
Weiterhin möchte ich mich ausdrücklich bei allen Probanden bedanken, die sich trotz Thematisierung ihrer traumatischen Erfahrungen bereit erklärt haben, sich mit ihrer Studienteilnahme für die Wissenschaft zu engagieren. Das vorletzte Dankeschön gilt meinem Großonkel Schorsch: Lieber Petter Schorsch, mit deinen 95 Jahren bist du der größte Fan meiner wissenschaftlichen Arbeit. Es ist so schön, dich so stolz zu sehen. Zu guter Letzt möchte ich vor allem meiner Familie und meinen Freunden danken, die auf indirektem Weg zu dem Gelingen dieser Arbeit beigetragen haben.

Liebe Mama, lieber Papa, lieber Stefan, ohne euch wäre diese Arbeit niemals so zustande gekommen. Diese Arbeit widme ich euch.