Erschienen in: Clinical Psychological Science ; 5 (2017), 6. - S. 974-984
https://dx.doi.org/10.1177/2167702617719946

aps ASSOCIATION FOR PSYCHOLOGICAL SCIENCE

Empirical Article

Clinical Psychological Science
2017, Vol. 5(6) 974–984

DOI: 10.1177/2167702617719946
www.psychologicalscience.org/CPS
SAGE

Mental Defeat and Cumulative Trauma Experiences Predict Trauma-Related Psychopathology: Evidence From a Postconflict Population in Northern Uganda

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Abstract
The peritraumatic cognitive process of mental defeat, the complete loss of inner resistance, has been identified as a key predictor of PTSD. Yet, most evidence on cognitive risk factors stems from industrialized countries where survivors typically report few traumata. Research from postconflict settings indicates that individual differences decrease with accumulating traumatic experiences, as almost everybody develops PTSD at extreme levels of trauma load. Would this leave less room for the impact of cognitive processes? In a sample of 227 Ugandan rebel war survivors, we investigated whether mental defeat influences trauma-related psychopathology in regression models accounting for cumulative trauma exposure. We found strong main effects of mental defeat on lifetime PTSD risk, current PTSD severity and dissociative symptoms, but no mental defeat × trauma load interaction effects. Our results indicate that peritraumatic mental defeat is central to understand individual differences in psychological reactions after single traumatic events as well as multiple traumatization.

Keywords
mental defeat, cognitive risk factors, posttraumatic stress disorder, shutdown dissociation, building block effect

Received 12/29/16; Revision accepted 6/3/17

Traumatic experiences such as accidents, natural disasters, or sexual assaults can lead to the development of posttraumatic stress disorder (PTSD). However, although 50% to 60% of individuals in Western countries report at least one traumatic experience, only a small fraction (5%–10%) presents with PTSD (Kessler et al., 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Hence, the development of persistent PTSD symptoms after a single or few traumatic experiences seems to be an exception. Accordingly, there must be individual risk and resilience factors that influence PTSD development and symptom trajectories after traumatic experiences. The knowledge of such factors is crucial to identify as early as possible individuals at risk and to allocate therapeutic resources accordingly.

Current cognitive models, such as the cognitive model of PTSD by Ehlers and Clark (2000), explain how cognitive risk factors can contribute to individual differences in PTSD susceptibility. According to their model, persistent PTSD develops if trauma survivors process the traumatic experiences in a way that they represent a current threat that is present in the here and now. Two key processes have been identified that lead to an increased individual vulnerability for PTSD: (a) memory disturbances, including a weak elaboration and contextualization of the traumatic experiences, and (b) negative cognitive appraisals of the trauma or its consequences (Ehlers & Clark, 2000).

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Peritraumatic cognitive processes might thus represent important risk factors for PTSD development, as they influence both memory formation and appraisals of the traumatic experience. These processes help understand pathways to developing PTSD and its maintenance, and, importantly, are also useful targets for psychosocial interventions, such as trauma-focused cognitive behavioral therapy. Among these cognitive processes, mental defeat has been identified as one of the most important peritraumatic predictors of PTSD development and chronification in survivors of violent assault (Kleim, Ehlers, & Glucksman, 2007). Mental defeat describes a state of mentally giving up during the trauma, associated with a perceived loss of dignity, autonomy, sense of being human, free will, and self-esteem (Dunmore, Clark, & Ehlers, 1999, 2001). The experience of mental defeat during the trauma is likely to contribute to the development of negative self-appraisals (Kleim, Ehlers, & Glucksman, 2012) regarding the trauma itself (e.g., "I am weak"; Dunmore et al., 2001, p. 1079) and its consequences (e.g., "My personality has changed for the worse"; Ehlers & Clark, 2000, p. 322). Furthermore, mental defeat and the subsequent development of negative self-beliefs might lead to maladaptive coping styles (Dunmore et al., 2001). As the trauma memories will trigger not only fear but also negative self-appraisals, trauma survivors who experienced peritraumatic mental defeat have an increased likelihood to engage in avoidance behavior, which prevents them from reprocessing and reorganizing the poorly elaborated trauma memories and accordingly contributes to the maintenance of PTSD symptoms (Dunmore et al., 2001; Ehlers, Clark et al., 1998). One study investigated survivors of sexual and physical assault (Dunmore, Clark, & Ehlers, 1997) and reported that those with persistent PTSD retrospectively reported more often experiences of peritraumatic mental defeat compared to those who recovered from it. The same group of authors conducted a larger study on 92 survivors of sexual or physical assault (Dunmore et al., 1999). Individuals who developed PTSD reported higher levels of prior trauma and childhood abuse, and they were more likely to report experiences of mental defeat compared to assault survivors without PTSD. Furthermore, those individuals who spontaneously recovered from PTSD reported lower levels of mental defeat than those who maintained PTSD symptoms (Dunmore et al., 1999). Moreover, survivors of political imprisonment in the former German Democratic Republic with lifetime PTSD reported significantly elevated mental defeat compared to those without PTSD. Mental defeat also predicted PTSD symptom severity in this sample beyond the effect of trauma severity during imprisonment (Ehlers, Maercker, & Boos, 2000).

These cross-sectional investigations were followed by several prospective studies investigating assault survivors shortly after the trauma, showing that mental defeat predicted PTSD symptom trajectories up to 6 to 9 months after the assault (Dunmore et al., 2001; Freeman et al., 2013; Kleim et al., 2007; Kleim et al., 2012). Even more, mental defeat was a central predictor of PTSD development above and beyond acute stress disorder symptoms shortly after the trauma (Kleim et al., 2007). Furthermore, individuals reporting high levels of peritraumatic mental defeat showed poorer outcomes of exposure-based psychotherapy, indicating the need for additional cognitive therapy elements (Ehlers, Clark et al., 1998). In sum, mental defeat seems to be an important predictor for PTSD development and treatment response in samples from industrialized countries.

Yet, evidence from conflict settings clearly indicates the limits of individual risk and resilience factors. With an increasing number of different lifetime traumatic event types (e.g., child maltreatment, war experiences, torture, abduction, sexual violence), the prevalence rates of PTSD increase in a dose-dependent manner and approach 100% at extreme levels of traumatic load (Catani et al., 2010; Kolassa, Ertl, Kolassa, Onyut, & Elbert, 2010; Mollica, McInnes, Poole, & Tor, 1998; Neugebauer et al., 2009; Neuner et al., 2004; Schauer et al., 2003; Wilker et al., 2015). Furthermore, the likelihood of spontaneous remission decreases with increasing traumatic load (Kolassa et al., 2010). This strong effect of the experience of different traumatic event types has been termed *building block effect* (Schauer et al., 2003) and can be explained by the fear network model as formulated by the work group of Elbert (Elbert & Schauer, 2002; Elbert, Rockstroh, Kolassa, Schauer, & Neuner, 2006; Wilker, Elbert, & Kolassa, 2014; Wilker & Kolassa, 2013) as an extension of Foa and Kozak (1986) and Lang (1979), which assumes that the emotional, sensory, and cognitive elements of traumatic memories are stored in a highly associative memory structure. The model posits that with an increasing number of events, which accumulate in the fear memory network, the network expands and the connections strengthen, which increases the likelihood of its activation by trauma reminders. Accordingly, individual risk factors might lose their importance with accumulating lifetime trauma exposure. However, the majority of studies investigating mental defeat stems from industrialized countries where survivors typically report few different traumatic event types. The study of Ehlers, Maercker, and Boos (2000) represents an important exception, as they investigated former political imprisoners who survived different types of torture. Their results provide initial evidence that mental defeat might

remain a central predictor of PTSD even after multiple and extreme traumatization (Ehlers et al., 2000).

Next to PTSD symptoms, survivors of multiple traumata are prone to suffer from dissociative symptoms, which include immobility, depersonalization, and derealization (Vermetten & Spiegel, 2014). According to evolutionary frameworks, such as the defense cascade model (Schauer & Elbert, 2010; see also Hagenaars, Oitzl, & Roelofs, 2014; Kozlowska, Walker, McLean, & Carrive, 2015; Lang, Bradley, & Cuthbert, 1998), shutdown dissociation occurs as an adaptive response of the body when fight or flight reactions are no longer possible. Hence, the choice of the body's reaction depends on an appraisal process that evaluates the perceived intensity of the threat in relation to the individual's own power (Schauer & Elbert, 2010). If an individual feels powerless, or defeated, dissociation is likely to occur. Furthermore, trauma reminders are likely to trigger the same reactions that were produced at the time of the trauma (Elbert & Schauer, 2002; Schauer & Elbert, 2010; Wilker & Kolassa, 2013), which can lead to persisting dissociative symptoms in trauma survivors. Because mental defeat by definition occurs in situations in which the individual feels completely powerless, one might expect that individuals who experienced peritraumatic mental defeat might also be more prone to shutdown dissociative symptoms as opposed to survivors who did not experience mental defeat. Furthermore, shutdown dissociation was found to be highest in patient populations who survived traumatic events with high proximity to the perpetrator such as physical or sexual abuse (Schalinski, Elbert, & Schauer, 2011; Schalinski, Schauer, & Elbert, 2015) and might hence be of particular relevance to our study population, who experienced high rates of interpersonal violence in the rebel war in northern Uganda.

Given the paucity of literature regarding the role of cognitive processes in PTSD development from survivors of mass conflict, the present study aimed at investigating whether mental defeat, a cognitive risk factor, would predict PTSD prevalence and symptomatology in survivors of multiple trauma. In particular, we investigated whether mental defeat would predict PTSD above and beyond the effect of cumulative trauma exposure. Because previous studies from conflict settings indicate that individual risk and resilience factors play only a subordinate role at extreme levels of trauma load, we also examined whether the effects of mental defeat are stronger at lower levels of trauma exposure by testing potential trauma load × mental defat interaction effects. Furthermore, we analyzed whether mental defeat would also predict current symptoms of shutdown dissociation in this population.

Method

Procedure

Data collection took place in villages of Nwoya district in northern Uganda. This area was severely affected by the war between the Lord's Resistance Army (LRA) rebel group and the Ugandan governmental forces, which lasted almost two decades. The atrocities committed during this war included forced recruitment and abductions of children and young adults, killings, mutilations, and sexual offenses. Data collection took place in 2013; accordingly, 8 years had passed since the cease-fire agreement between the LRA and the governmental troops in 2005.

We first introduced the study procedures to the villagers in community meetings, explained the study aims and procedures, and invited interested community members to approach us to schedule an appointment. Inclusion criteria comprised (a) age between 18 and 65, (b) a history of trauma exposure, (c) absence of psychotic symptoms, and (d) absence of signs of alcohol addiction. In total, 240 individuals were interviewed. We excluded 13 individuals from further analyses for the following reasons: signs of current alcohol abuse (n = 10), difficulties in understanding the interview questions (n = 2), and a history of psychotic symptoms (n = 1). The resulting sample of 227 was 54% female, with a mean age of 33.29 (SD = 10.56, range = 18–62). This study is part of a larger project investigating the genetics of PTSD, and the study sample has been also analyzed in a previous publication investigating how to best quantify traumatic load for gene × environment interaction studies (Wilker et al., 2015).

After a detailed explanation of the study protocol participants gave written informed consent. The procedures followed the declaration of Helsinki and were approved by the institutional review board of Gulu University, Uganda, the Ugandan National Council for Science and Technology (UNCST), and the ethics committee of the German Psychological Society (Deutsche Gesellschaft für Psychologie).

All information was gathered by structured diagnostic interviews that were performed by trained Ugandan interviewers under the supervision of psychologists from the universities of Ulm and Konstanz. The interviewers attended a 6-week training on the concepts of quantitative data collection, counseling skills, mental health disorders, trauma, and PTSD. All diagnostic instruments were translated into Luo, the local language of northern Uganda. The translations from English to Luo were followed by blind back-translations into English, and group discussions with independent interpreters, to ensure a valid translation of the instruments.

At the beginning of the interview, we assessed demographic data, as well as information regarding exposure to the LRA war (e.g., displacements, abductions). Next, we investigated the amount of trauma exposure by means of a 62-item event list. This event list comprised general traumatic experiences (e.g., natural disasters, accidents), war-related traumatic events (e.g., being close to combat), as well as events specific for the LRA conflict (e.g., being forced to kill somebody by the LRA). We calculated the number of different traumatic event types experienced to assess the amount of trauma exposure (*traumatic load*). As previously shown in the same sample, this variable can be reliably measured in LRA war survivors and leads to the best prediction of lifetime PTSD (Wilker et al., 2015). Therefore, it was suggested to include the number of different traumatic event types in studies, which intend to assess individual risk factors in traumatized populations (Wilker et al., 2015).

After completing the event list, participants were asked to identify their worst traumatic experience. The extent of mental defeat was assessed for the worst traumatic event using the Mental Defeat Questionnaire (MDQ) in the form of an interview (Dunmore et al., 1999, 2001). The MDQ comprises 11 unipolar items (e.g., "I lost any will-power," "I felt destroyed as a person") and requires responses on a 5-point Likert-type scale ranging from *not at all* to *very strong*. The MDQ showed a good internal consistency in the present sample (Cronbach's $\alpha = .89$).

We further assessed the diagnosis of current and lifetime PTSD according to *DSM–IV*, as well as PTSD symptom severity employing the Posttraumatic Diagnostic Scale (PDS; Foa, 1995) as a diagnostic interview. The reliability and validity of the PTSD diagnostics by trained local interviewers in northern Uganda have been verified in a prior investigation (Ertl et al., 2010).

In addition, we assessed dissociative symptoms by means of the Shutdown Dissociation Scale (Shut-D; Schalinski et al., 2015). In contrast to other scales measuring dissociative responding that were developed based on clinical observations, the items of the Shut-D were generated based on the theoretical assumptions of the evolutionary framework of the defense cascade model (Schalinski et al., 2015; Schauer & Elbert, 2010). The Shut-D includes 13 unipolar items (e.g., "Have you fainted?" "Have you felt like you were outside of your body?" "Have you felt suddenly weak and warm?" "Have you felt nauseous? Have you felt as though you were about to throw up? Have you felt yourself break out in a cold sweat?") investigating current bodily dissociative symptoms for the past 6 months. Participants were requested to answer on a 4-point scale ranging from *never* to *several times a week*. In a validation study including three samples and a total of 225 patients and 68 healthy controls, the scale showed good psychometric characteristics in healthy controls, individuals with PTSD, depression, psychosis, dissociative identity disorder, and borderline personality disorder (Schalinski et al., 2015). The Shut-D exhibited good internal reliability, excellent test-retest reliability, a high convergent validity with the Dissociative Experiencing Scale, and satisfactory predictive validity (Schalinski et al., 2015). The Shut-D showed a high internal reliability in the present study too (Cronbach's $\alpha = .91$).

Demographics of the study sample

Of the total sample, 50 participants (22%) met *DSM–IV* criteria for current PTSD, whereas 163 participants (72%) fulfilled criteria for lifetime PTSD. Hence, of the subsample of 163 study participants who ever fulfilled the *DSM–IV* criteria for PTSD, 113 individuals showed spontaneous remission. The mean PTSD symptom severity as indicated by the PDS sum score was 4.61 ($SD = 5.92$, range = 0–32). The majority of individuals ($n = 140$, 62%) had been abducted by the LRA. Respondents reported an average number of 26.93 ($SD = 9.48$, range = 2–61) different types of traumatic events. The mean value of the MDQ was 27.26 ($SD = 12.33$, range = 0–44). Table 1 summarizes demographic and clinical information separately for the different diagnostic groups. Table S1 in the Supplemental Material available online displays the correlations between the clinical variables.

Statistics

For the demographic description of the study sample, we calculated means and standard deviations, if the variables were normally distributed, and median and range in case of nonnormality. Demographic and clinical variables were compared between individuals who never met the diagnostic criteria for PTSD, individuals with a current diagnosis of PTSD, and individuals who once fulfilled the diagnostic criteria for PTSD but did not display the symptoms anymore at the time of the assessment (remitted PTSD; Table 1). For the statistical evaluation of group differences, we calculated ANOVA F tests, if test residuals met the assumption of normality, and Kruskal–Wallis H tests as a nonparametric alternative, if this requirement was not met. If the global comparisons indicated significant differences between the groups, we next performed post hoc tests with multiple-testing correction to determine which groups differed significantly. In case of significant ANOVA results, we calculated Tukey's honestly significant difference as a parametric post hoc test, whereas the multiple comparison test after Kruskal–Wallis (Giraudoux, 2014;

Table 1. Demographic and Clinical Variables by Diagnostic Group

Variable	Never PTSD (n = 64)	Lifetime PTSD (n = 163)		Statistic[a]	p value
		Remitted PTSD (n = 113)	Current PTSD (n = 50)		
Demographics					
n female (%)	32 (50)	65 (58)	25 (50)	$\chi^2(2) = 1.29$	.52
Median age [range]	30 [18–57]	33 [18–62]	30 [18–54]	$H(2) = 2.38$	.30
Median years of school education [range]	6 [0–16]	5 [0–14]	5 [0–16]	$H(2) = 0.51$	.78
Trauma exposure and clinical variables					
Median age at worst event [range]	18 [4–44]	18 [6–45]	16.5 [6–50]	$H(2) = 1.17$	.56
Mean number of lifetime traumatic event types experienced [SD]	20.66 [8.14][b]	26.98 [7.99][c]	34.86 [8.22][d]	$F(2, 224) = 43.34$	<.0001
Median MDQ score [range]	15 [0–44][b]	32 [0–44][c]	36.5 [1–44][c]	$H(2) = 62.11$	<.0001
Median PDS sum score [range]	0 [0–10][b]	1 [0–12][b]	13 [6–32][c]	$H(2) = 123.65$	<.0001
Median Shut-D score [range]	0 [0–13][b]	1 [0–16][c]	8.5 [0–34][d]	$H(2) = 72.09$	<.0001

Note: MDQ = Mental Defeat Questionnaire; PDS = Posttraumatic Diagnostic Scale; Shut-D = Shutdown Dissociation Questionnaire.
[a]Statistic refers to χ^2 tests for categorical data, and ANOVA F test for continuous data if test residuals were normally distributed, Kruskal–Wallis H test if residuals were nonnormally distributed. All reported p values are two-tailed tests.
[b,c,d]Global comparisons of the continuous variables for the three groups were followed by parametric or nonparametric post hoc tests, if the ANOVA F test or Kruskal–Wallis H test revealed statistical significant differences between the groups. Different letters indicate a statistical significant difference in the post hoc test between the respective groups.

Siegel & Castellan, 1988) was employed as a nonparametric alternative.

We further employed regression models to investigate whether mental defeat contributes to the prediction of PTSD diagnosis and posttraumatic psychopathology. In particular, regression models were calculated to investigate the additional predictive power of mental defeat in predicting PTSD over and above the effect of cumulative trauma exposure as well as the control variables age, sex, and age at worst event. We also tested whether mental defeat and trauma load would interact in the prediction of trauma-related psychopathology. To investigate the categorical outcomes current and lifetime PTSD, as well as spontaneous remission from PTSD, we conducted logistic regression analyses. For the continuous variables PTSD symptom severity (assessed by the PDS sum score) and dissociative symptom severity (Shut-D sum score), we calculated negative binomial regression models because the assumptions of linear regression were violated due to an excess of small values and overdispersed data in these variables.

Nested models of varying complexity were compared based on the model fit criterion Akaike's information criterion (AIC). Furthermore, Nagelkerke's R^2 was calculated for all models as a pseudo-R^2 measurement of explanatory power. Finally, for the logistic regression models predicting PTSD diagnosis the coefficient of discrimination (D) was computed indexing the ability of the model to differentiate the two possible outcomes (i.e., PTSD vs. no PTSD). A coefficient of 0 would refer to no discriminatory power, whereas a coefficient of 1 would refer to a perfect classification of cases and controls (Tjur, 2009).

To determine statistical significance of traumatic load and mental defeat in the prediction of the outcome variables, we calculated likelihood ratio (LR) tests, which have the advantage that they can be performed for both logistic and negative binomial regression models (Harrell, 2001). The strength of the influence of the predictors was evaluated by calculating odds ratios (ORs) and incidence rate ratios (IRRs) for logistic and negative binomial regression models, respectively. To allow for comparisons between the ORs and IRRs for the differently scaled predictors, we z-standardized predictor variables. Finally, we evaluated the ability of the models with and without mental defeat to correctly predict PTSD status by means of 1,000 repeats of tenfold cross-validation. All statistical analyses were performed using the statistical environment R version 3.3.2 (R Core Team, 2016).

Results

Prediction of PTSD diagnosis

Logistic regression models showed a strong association between traumatic load and the likelihood of lifetime PTSD development. However, the prediction of lifetime PTSD was decisively enhanced by also including mental defeat in a main effects model, as illustrated by the model fit statistics in Table 2. In a model including only

Table 2. Model Fit Statistics for the Prediction of Trauma-Related Psychopathology

Predictor	AIC	D	Nagelkerke's R^2
Prediction of Lifetime PTSD			
Traumatic Load	225.28	0.18	.24
MDQ	202.09	0.29	.36
Traumatic Load** + MDQ****	**186.36**	**0.36**	**.44**
Traumatic Load × MDQ	187.26	0.37	.45
Age + Sex + Age at Worst Event	264.25	0.03	.04
Age + Sex + Age at Worst Event + Traumatic Load	221.80	0.22	.29
Age + Sex + Age at Worst Event + Traumatic Load + MDQ	190.00	0.37	.45
Age + Sex + Age at Worst Event + Traumatic Load × MDQ	190.96	0.38	.46
Prediction of Current PTSD			
Traumatic Load	192.67	0.22	.30
MDQ	223.72	0.07	.12
Traumatic Load ** + MDQ**	**192.45**	**0.23**	**.31**
Traumatic Load × MDQ	193.99	0.22	.32
Age + Sex + Age at Worst Event	243.75	0.01	.01
Age + Sex + Age at Worst Event + Traumatic Load	196.19	0.23	.31
Age + Sex + Age at Worst Event + Traumatic Load + MDQ	196.07	0.24	.33
Age + Sex + Age at Worst Event + Traumatic Load × MDQ	197.38	0.23	.33
Prediction of Spontaneous Remission from PTSD			
Traumatic Load**	**174.67**	**0.18**	**.24**
MDQ	201.25	0.02	.03
Traumatic Load + MDQ	176.61	0.18	.24
Traumatic Load × MDQ	178.60	0.18	.24
Age + Sex + Age at Worst Event	205.79	0.02	.03
Age + Sex + Age at Worst Event + Traumatic Load	178.99	0.18	.25
Age + Sex + Age at Worst Event + Traumatic Load + MDQ	180.86	0.18	.25
Age + Sex + Age at Worst Event + Traumatic Load × MDQ	182.82	0.18	.25
Prediction of Current PTSD Symptom Severity			
Traumatic Load	1094.93	—	.30
MDQ	1121.37	—	.14
Traumatic Load + MDQ	1092.42	—	.32
Traumatic Load × MDQ	1094.38	—	.32
Age + Sex + Age at Worst Event	1143.28	—	.02
Age + Sex + Age at Worst Event + Traumatic Load	1095.43	—	.33
Age + Sex + Age at Worst Event* + Traumatic Load** + MDQ***	**1092.36**	**—**	**.36**
Age + Sex + Age at Worst Event + Traumatic Load × MDQ	1094.28	—	.36
Prediction of Current Dissociative Symptom Severity			
Traumatic Load	1007.54	—	.17
MDQ	995.70	—	.24
Traumatic Load + MDQ****	**987.87**	**—**	**.30**
Traumatic Load × MDQ	988.60	—	.31
Age + Sex + Age at Worst Event	1035.39	—	.00
Age + Sex + Age at Worst Event + Traumatic Load	1009.55	—	.19
Age + Sex + Age at Worst Event + Traumatic Load + MDQ	990.40	—	.32
Age + Sex + Age at Worst Event + Traumatic Load × MDQ	989.91	—	.33

Note: AIC = Akaike's information criterion; D = coefficient of discrimination; MDQ = Mental Defeat Questionnaire. The best model, according to the Akaike's information criterion, is bold. Asterisks denote the regression items that were significant in the selected regression model.

*$p < .05$. **$p < .01$. ***$p < .001$. ****$p < .0001$.

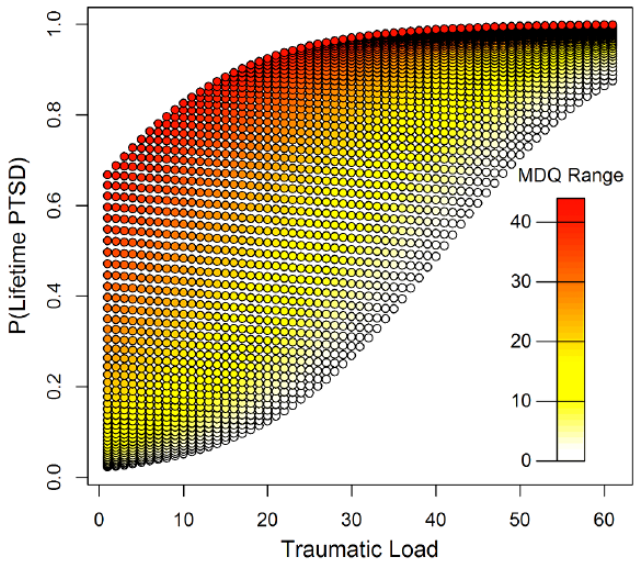

Fig. 1. Fitted values of probability for a diagnosis of lifetime PTSD derived from a logistic regression model with traumatic load and mental defeat as predictor variables. The range of the Mental Defeat Questionnaire (MDQ) is represented by heat colors, with white representing a score of 0, yellow and orange representing low to medium values, and red representing the highest values on the MDQ.

traumatic load and mental defat, both traumatic load (OR = 2.46 [1.60–3.91], LR = 17.83, $p < .0001$) and mental defeat (OR = 3.48 [2.35–5.39], LR = 44.84, $p < .0001$) showed a strong relationship with lifetime PTSD (see Fig. 1). Furthermore, mental defeat still predicted lifetime PTSD risk above and beyond traumatic load after controlling for age, sex, and age at worst event (traumatic load: OR = 2.77 [1.74–4.64], LR = 19.76, $p < .0001$; MDQ: OR = 3.21 [2.12–5.06], LR = 33.81, $p < .0001$). The inclusion of the interaction of mental defeat and trauma load did not improve model fit in models with and without considering age, sex, and age at worst event. In 1,000 repeats of tenfold cross validation, the mean prediction accuracy for lifetime PTSD from a model including traumatic load and mental defeat was 82% (sensitivity = 90%, specificity = 62%). In comparison, prediction accuracy from traumatic load alone was 77% (sensitivity = 93%, specificity = 34%).

Traumatic load was also strongly associated with the likelihood of a diagnosis of current PTSD. Furthermore, also considering mental defeat in the regression model improved model fit statistics, whereas the inclusion of a trauma load × mental defeat interaction effect worsened model fit as indicated by the AIC (see Table 2). However, in a main effects model including trauma exposure and mental defeat, only trauma exposure was a strong and significant predictor of current PTSD (OR = 3.44 [2.17–5.77], LR = 32.30, $p < .0001$), whereas mental defeat did not reach statistical significance (OR = 1.42 [0.92–2.27], LR = 2.41, $p = .12$). These results were mirrored when also controlling for age, sex, and age at worst event. We further investigated whether spontaneous remission from PTSD was predicted by traumatic load and mental defeat. Although the probability of spontaneous remission decreased with increasing traumatic load (OR = 0.32 [0.19–0.51], LR = 26.63, $p < .0001$), mental defeat did not contribute to the prediction of spontaneous remission (OR = 0.94 [0.56–1.55], LR = .06, $p = .81$). Similar results were obtained when also controlling for age, sex, and age at worst event.

Prediction of PTSD symptoms

The consideration of a main effect of mental defeat improved the prediction of current PTSD symptom severity in negative binomial regression models, whereas including trauma load × mental defeat interaction effects increased the AIC, indicating a worse model fit (see Table 2). Furthermore, in a main effect model accounting for the effect of trauma exposure (IRR = 1.88 [1.52–2.32], LR = 31.78, $p < .0001$), mental defeat was a significant predictor of PTSD symptom severity (IRR = 1.27 [1.03–1.56], LR = 5.01, $p = .025$). The effect remained stable when age, sex, and age at worst event were controlled for (trauma load: IRR = 1.92 [1.54–2.41], LR = 31.53, $p < .0001$; MDQ: IRR = 1.28 [1.03–1.57], LR = 5.07, $p = .024$).

Prediction of dissociative symptoms

In negative binomial regression models predicting dissociative symptoms (assessed by the Shut-D), the inclusion of a main effect of mental defeat improved model fit criteria compared to a model only including traumatic load, whereas the inclusion of a potential interaction effect displayed a worse model fit according to the AIC (Table 2). Furthermore, mental defeat was a strong and significant predictor of the Shut-D score (IRR = 1.95 [1.51–2.52], LR = 23.61, $p < .0001$; see also Fig. 2) above and beyond the effect of traumatic load (IRR = 1.48 [1.16–1.90], LR = 9.88, $p = .002$). The same pattern was observed when also adjusting for age, sex, and age at worst event (traumatic load: IRR = 1.58 [1.21–2.08], LR = 11.17, $p = .0008$; MDQ: IRR = 1.91 [1.47–2.48], LR = 21.14, $p < .0001$).

Discussion

In line with previous investigations (Catani et al., 2010; Kolassa et al., 2010; Mollica et al., 1998; Neugebauer et al., 2009; Neuner et al., 2004; Schauer et al., 2003; Wilker et al., 2015), this study highlights the cumulative

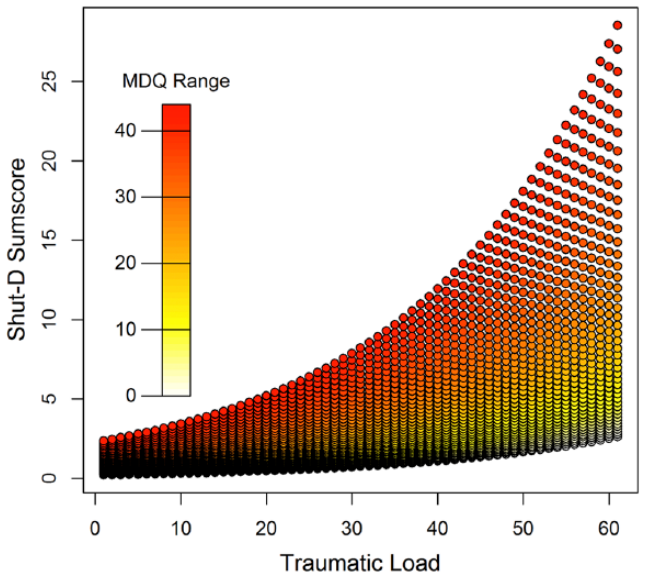

Fig. 2. Fitted values of the Shutdown Dissociation Questionnaire (Shut-D) derived from a negative binomial regression model with traumatic load and mental defeat as predictor variables. The range of the Mental Defeat Questionnaire (MDQ) is represented by heat colors, with white representing a score of 0, yellow and orange representing low to medium values, and red representing the highest values on the MDQ.

effect of trauma exposure on the risk of PTSD development and symptomatology.

Yet, despite this strong effect of traumatic load on lifetime PTSD, mental defeat still decisively contributed to the explanation in individual differences in PTSD susceptibility across all levels of trauma load. Severity of peritraumatic mental defeat in individuals with a diagnosis of lifetime PTSD was more than twice as high compared to individuals who never met diagnostic criteria for PTSD. In a regression model accounting for main effects of traumatic load and mental defeat, an increase of one *SD* on the MDQ increased the odds of a lifetime PTSD diagnosis by more than three. Including mental defeat in cross-validation procedures increased the specificity of lifetime PTSD prediction from 34% in a model including only traumatic load to 62% if mental defeat was also considered. Hence, mental defeat significantly contributed to a better prediction of lifetime PTSD prevalence (see also Fig. 1 for an illustration of this effect).

Regarding the prediction of current PTSD, the inclusion of mental defeat in regression models accounting for traumatic load improved model fit criteria, but mental defeat did not reach statistical significance. This might be partly explained by the relatively low prevalence rate (22%) of current PTSD, as many survivors had recovered during the eight years that had passed since the cease-fire agreement. At the time of the assessment, the majority of the study participants who once fulfilled a diagnosis of PTSD already showed spontaneous remission. Several factors apart from mental defeat have been shown to influence remission from PTSD, including negative appraisals of self (Dunmore et al., 2001), maladaptive coping styles such as rumination (e.g., Ehring & Ehlers, 2014; Kleim et al., 2007), thought suppression (e.g., Ehlers, Mayou, & Bryant, 1998; Kleim et al., 2012), or safety behaviors (Dunmore et al., 2001). Even more, adaptive factors and processes related to PTSD remission may have played a crucial role in this process, like, for instance, social support or aspects of emotion regulation (Brewin & Holmes, 2003; Ozer, Best, Lipsey, & Weiss, 2003). Indeed, spontaneous remission from PTSD occurred independently of peritraumatic mental defeat in our sample.

However, mental defeat significantly predicted current PTSD symptom severity in regression models accounting for the cumulative effect of trauma exposure. Finally, it was remarkable that the effect of mental defeat on shutdown dissociation was particularly strong. In regression models accounting for the effect of trauma exposure, an increase of mental defeat by one *SD* almost doubled the predicted shutdown dissociation. Events with particular proximity to the perpetrator—which include severe forms of sexual and physical violence—are more likely to cause symptoms of shutdown dissociation compared to traumatic events without direct perpetrator contact (Schalinski et al., 2011; Schalinski et al., 2015; Schauer & Elbert, 2010). Hence, one might assume that similar types of traumatic events could be associated with both mental defeat and dissociative responding. On the other hand, similar individual predispositions might influence whether an individual responds with mental or physical resistance or mental and bodily defeat and dissociation (for a further discussion of the overlap of mental defeat and shutdown dissociation, see below).

Hence, similar to findings from industrialized countries (Dunmore et al., 1997, 1999, 2001; Ehlers et al., 2000; Freeman et al., 2013; Kleim et al., 2007; Kleim et al., 2012), our study showed that peritraumatic mental defeat also contributes to posttraumatic symptomatology in survivors of multiple traumatic experiences from a postconflict setting. This is in line with findings of Ehlers et al. (2000), who showed that mental defeat predicted PTSD symptom severity beyond the effect of maltreatment severity in political prisoners who survived torture. In contrast to previous studies, however, we assessed trauma exposure over the entire lifespan with a detailed life event checklist, comprising 62 different traumatic experiences. Because we found evidence of strong main effects of trauma load and mental defeat on posttraumatic symptomatology in the absence

of significant interaction effects, mental defeat seems to influence posttraumatic symptomatology across all levels of trauma load.

Thus, our findings correspond well with prominent theories on PTSD development. First, the cognitive model of PTSD (Ehlers & Clark, 2000) assumes that PTSD symptoms result if trauma survivors process the experienced past traumatic events in a way that the events or their consequences lead to the feeling of a current threat. This sense of an ongoing threat is based on characteristics of the trauma memory and on negative assumptions and beliefs about oneself and the world, such as "I am a loser" or "There is no place which is safe" (Dunmore et al., 1999, p. 825). Experiences of mental defeat increase the likelihood of the development of such negative beliefs about oneself, such as "I am destroyed as a person" or "I have no power to protect myself," and thereby increase the risk of PTSD symptom development (Dunmore et al., 2001). Furthermore, mental defeat might enhance maladaptive coping strategies such as avoidance of thinking and talking about the traumatic event, and thereby perpetuate PTSD symptoms, as it prevents a modification of the poorly elaborated and contextualized trauma memories (Dunmore et al., 2001).

Second, the fear network model (Elbert et al., 2006; Elbert & Schauer, 2002; Wilker et al., 2014; Wilker & Kolassa, 2013) also describes peritraumatic cognitions as important elements of the trauma memory network, which will be remembered as part of the trauma. Therefore, trauma reminders will activate not only the emotions and sensory memories associated with the trauma but also, in the case of an experience of mental defeat, feelings of lost dignity and a lost sense of humanness. Therefore, this model can also provide an explanation on how mental defeat might increase the likelihood of the development of persisting PTSD.

Finally, from a neurobiological perspective, the peritraumatic reactions to traumatic stress can be described by the defense cascade model (Schauer & Elbert, 2010; see also Hagenaars et al., 2014; Kozlowska et al., 2015; Lang et al., 1998). With increasing danger and decreasing perceived individual defense possibilities, the defense cascade escalates from a state where fight or flight is still possible (accompanied by an activation of the sympathetic nervous system) to states of peritraumatic shutdown dissociation, which are characterized by parasympathetic dominance (Schauer & Elbert, 2010). The results of this study revealed an association between peritraumatic mental defeat and states of persistent dissociative responding in survivors of the LRA war. Persistent dissociative reactions are associated with severe PTSD symptoms (Schalinski et al., 2011), which might be due to increased difficulties to integrate the fragmented trauma memories into autobiographical memory in case of ongoing dissociative responses (Schauer & Elbert, 2010). One possible interpretation of the results of our study might hence be that mental defeat represents a cognitive counterpart to the biological processes that accompany the escalation of the defense cascade and result in shutdown dissociation.

Strengths and Limitations

Strengths of this study include the relatively large sample size, the systematic assessment via structured clinical interviews as well as the detailed assessment of trauma exposure, and the simultaneous investigation of a cognitive risk factor and dose-dependent effects of trauma exposure. The main limitation is the cross-sectional assessment, which required a retrospective recall of mental defeat during the trauma, which might be biased by current symptoms. However, one finding that speaks against this hypothesis is that mental defeat showed a stronger association with lifetime as opposed to current PTSD. In contrast to previous studies, we employed the MDQ in the form of a structured interview. Although this was necessary to ensure the comprehension of the questions by all study participants, it also limits comparability to previous investigations on cognitive risk factors for PTSD. Furthermore, the prevalence of current PTSD was relatively low; as many survivors showed spontaneous remission in the time interval since their worst traumatic experiences during the LRA conflict. This high remission rate is in accordance with findings of a recent meta-analysis on PTSD remission (Morina, Wicherts, Lobbrecht, & Priebe, 2014). Accordingly, the mean PDS score was also quite low. This might have limited the power of the analyses regarding the influence on mental defat on the diagnosis of current PTSD as well as on current PTSD symptom severity. Finally, the MDQ was assessed in respect to only one, the worst event. The possibility that the defeat can be high in some but low during other traumatic experiences deserves further investigation.

Conclusions and Implications

In sum, this study showed that in a severely traumatized sample of rebel war survivors, mental defeat contributed to the prediction of PTSD and shutdown dissociation. Hence, our results indicate that individual cognitive risk factors and the associated biological cascades play an important role after multiple traumatization. This underscores a pivotal role of mental defeat in the development of PTSD and a high clinical relevance of assessing cognitive processes and addressing them in trauma-focused psychotherapy. In particular, mental

defeat might impair the recovery process during exposure-based psychotherapy, as it might be difficult for the trauma survivor to appraise the traumatic experience as an event in the past which no longer signals danger if the negative self-beliefs persist. Therefore, therapists should assess mental defeat and directly target those cognitions during the therapeutic process (Dunmore et al., 2001; Ehlers, Clark et al., 1998; Schauer, Neuner, & Elbert, 2011).

Author Contributions

S. Wilker, B. Kleim, and I.-T. Kolassa developed the study concept. S. Wilker, A. Geiling, and A. Pfeiffer performed the study setup and data collection, with general support from T. Elbert and I.-T. Kolassa. S. Wilker performed the data analysis and interpretation under the supervision of B. Kleim and I.-T. Kolassa. S. Wilker drafted the manuscript, and B. Kleim, T. Elbert, and I.-T. Kolassa provided critical revisions. All authors approved the final version of the manuscript for submission.

Acknowledgments

This research was supported by the German Research Foundation (Deutsche Forschungsgemeinschaft). We are very grateful to the team of Ugandan interviewers who conducted the diagnostic interviews with the highest empathy and professionalism.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Supplemental Material

Additional supporting information may be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702616719946

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