Soc Psychiatry Psychiatr Epidemiol (2013) 48:1743–1754
DOI 10.1007/s00127-013-0709-8

ORIGINAL PAPER

The role of stress sensitization in progression of posttraumatic distress following deployment

Geert E. Smid · Rolf J. Kleber · Arthur R. Rademaker · Mirjam van Zuiden · Eric Vermetten

Received: 29 December 2012 / Accepted: 9 May 2013 / Published online: 29 May 2013

Abstract

Purpose Military personnel exposed to combat are at risk for experiencing post-traumatic distress that can progress over time following deployment. We hypothesized that progression of post-traumatic distress may be related to enhanced susceptibility to post-deployment stressors. This study aimed at examining the concept of stress sensitization prospectively in a sample of Dutch military personnel deployed in support of the conflicts in Afghanistan.

Method In a cohort of soldiers ($N = 814$), symptoms of post-traumatic stress disorder (PTSD) were assessed before deployment as well as 2, 7, 14, and 26 months ($N = 433$; 53 %) after their return. Data were analyzed using latent growth modeling. Using multiple group analysis, we examined whether high combat stress exposure during deployment moderated the relation between post-deployment stressors and linear change in post-traumatic distress after deployment.

Results A higher baseline level of post-traumatic distress was associated with more early life stressors (standardized regression coefficient = 0.30, $p < 0.001$). In addition, a stronger increase in posttraumatic distress during deployment was associated with more deployment stressors (standardized coefficient = 0.21, $p < 0.001$). A steeper linear increase in posttraumatic distress post-deployment (from 2 to 26 months) was predicted by more post-deployment stressors (standardized coefficient = 0.29, $p < 0.001$) in high combat stress exposed soldiers, but not in a less combat stress exposed group. The group difference in the predictive effect of post-deployment stressors on progression of post-traumatic distress was significant ($\chi^2(1) = 7.85$, $p = 0.005$).

Conclusions Progression of post-traumatic distress following combat exposure may be related to sensitization to the effects of post-deployment stressors during the first year following return from deployment.

Keywords Post-traumatic stress disorder · Delayed onset · Stress sensitization · Military deployment

Paper presented at The International Society for Traumatic Stress Studies 28th Annual Meeting, Los Angeles, November 1–3, 2012.

Electronic supplementary material The online version of this article (doi:10.1007/s00127-013-0709-8) contains supplementary material, which is available to authorized users.

G. E. Smid (✉) · R. J. Kleber
Foundation Centrum'45/Arq, Nienoord 5,
1112 XE Diemen, The Netherlands
e-mail: g.smid@centrum45.nl

R. J. Kleber
Department of Clinical and Health Psychology,
Utrecht University, Utrecht, The Netherlands

A. R. Rademaker · M. van Zuiden · E. Vermetten
Research Centre-Military Mental Health,
Ministry of Defense, Utrecht, The Netherlands

M. van Zuiden
Department of Psychiatry, Academic Medical Center,
University of Amsterdam, Amsterdam, The Netherlands

M. van Zuiden
Laboratory of Neuroimmunology and Developmental Origins
of Disease, University Medical Center Utrecht, Utrecht,
The Netherlands

E. Vermetten
Rudolf Magnus Institute of Neurosciences, University Medical
Center Utrecht, Utrecht, The Netherlands

Introduction

Deployed military personnel are at risk of developing symptoms of posttraumatic stress disorder (PTSD) following deployment. Among Dutch soldiers, prevalence of PTSD 5 months after deployment to Iraq was estimated at 3–4 % [1]. Among U.S. soldiers, PTSD prevalence 3–4 months after deployment to Afghanistan was estimated at 6 % using strict criteria [2], and PTSD prevalence with serious functional impairment 3 and 12 months after deployment to Iraq at 6–12 % [3]. Among UK soldiers, PTSD prevalence after deployment to Iraq and Afghanistan was estimated at 4 % [4].

During the past decades, numerous studies have documented progression of PTSD symptoms following return from deployment. In soldiers and veterans initially reporting subthreshold levels of post-traumatic distress, progression of post-traumatic distress may lead to delayed-onset PTSD. The estimated prevalence of delayed-onset PTSD in military populations varies across prospective studies, equaling 3 % with onset between 3 and 9 months after deployment [5], 6.5 % with onset between 4 and 18 months [6], 7 % with onset between 1 and 7 months [7], and 3.5 % with onset between 1 and 6 years [8]. In a prospective study covering 20 years following the 1982 Lebanon war [9], veterans endorsing combat-related PTSD with delayed onsets 3–20 years following combat exposure were identified. In this study, delayed-onset PTSD was endorsed by 23.8 % of veterans who did not have stress reactions during the war and who did not meet PTSD criteria 1 year after the war.

A large cross-sectional population study [10] evidenced an increased likelihood of delayed onset of PTSD following exposure to military combat as compared to other potentially traumatic events. The association between military combat and delayed-onset PTSD was subsequently confirmed in a meta-analysis [11]. Consistent with these findings, some prospective studies noted an overall trend of symptom increase after combat exposure, i.e., an increase of point prevalence of PTSD across subsequent assessments. Following deployment to Iraq, a large sample ($N = 88{,}235$) of US soldiers were screened immediately after return and 3–6 months later [12]. Prevalence of screening positive for PTSD increased across occasions from 12 to 18 % in active soldiers, and from 13 to 25 % in reserve component soldiers returning to civilian life. Prospective studies of Gulf War veterans reported increases in PTSD symptom levels over the course of 2 years following return from deployment [13] as well as between 4 and 14 years following deployment [14]. Likewise, in UK regular soldiers after deployment to Iraq and Afghanistan, an increase in probable PTSD prevalence from 3.0 to 5.2 % was reported between 1 and 6.5 years post-deployment [4].

Post-traumatic distress following deployment has been shown to be related to several predictive factors. These markers of risk can be categorized according to their temporal origin in relation to the time of deployment. Thus, pre-deployment (e.g., demographic), deployment-related, and post-deployment risk markers can be outlined. Combat exposure has been reported as the strongest deployment-related predictor of subsequent posttraumatic distress [15]. Pre-deployment factors such as prior traumatic events [16] and early life trauma [17] have also been shown to predict posttraumatic distress. Recently, disciplinary offences during military service, preceding any exposure to trauma, were found to predict later delayed-onset PTSD [18].

Post-deployment stressors may additionally influence the course of post-traumatic distress. In a sample of soldiers deployed to Iraq who were followed up between 2 and 6 months after deployment, post-deployment stressors such as unemployment, broken relationships or illness of significant others were independently associated with PTSD symptom increases when controlling for baseline (pre-deployment) symptoms and deployment stressors [19]. Also, 77 % of veterans endorsing delayed-onset PTSD reported severe life stressors in the year preceding delayed PTSD onset, against 32 % in veterans who reported no PTSD [20]. Finally, in a prospective 20-year study in veterans from the 1982 Lebanon war, post-war negative life events were associated with delayed PTSD onset [21].

The effects of new stressors on posttraumatic distress may operate in two different ways. First, the effect may be simply additive, such that distress related to new stressors adds to the posttraumatic distress. Second, an interactive effect may occur if extreme traumatic exposure influences the intensity with which survivors respond to subsequent stressors. Indeed, exposure to extreme stressors may enhance an individual's reactivity to subsequent stressors, a process that has been labeled sensitization to stress [22]. Sensitization refers to the situation in which an organism responds more strongly to a variety of stimuli after exposure to a potentially threatening or noxious stimulus. It represents a form of non-associative learning that is likely to cover a repertoire of mechanisms [23]. Sensitized reactions may be both non-specific (e.g., depressed mood) and specific to the stimulus that caused the sensitization (e.g., trauma-related symptoms in PTSD). A role of stress sensitization in delayed-onset PTSD has been suggested [24]. A recent study among residents affected by a fireworks disaster [25] found that residents whose home was completely destroyed responded with greater distress to stressful life events reported 18–20 months following the disaster than residents whose home was less damaged, using a stratified analysis. These results suggest that stress sensitization effects are likely to manifest in groups characterized by considerable stressor exposure.

Stress sensitization can be described as a three–variable relationship in which change in posttraumatic distress over time constitutes the dependent variable, recent stressors (e.g., post-deployment stressful life events) represent a direct causal variable predicting change in distress, and prior stressors (e.g., high combat stress exposure) represent a temporally preceding interaction variable moderating the direct effects of recent stressors on change in distress. Simultaneous modeling of longitudinal change over time based on repeated assessments as well as causal and interaction effects can be achieved using structural equation modeling.

The aim of the current study was to examine whether progression of post-traumatic distress after return from military deployment could be explained with the stress sensitization hypothesis. Specifically, our research questions were (1) to what extent are high combat stress exposed (HCSE) soldiers more likely to manifest stress sensitization, i.e., increased reactivity to post-deployment stressors reported 1 year following deployment compared with low combat stress exposed (LCSE) soldiers? (2) Is stress sensitization likely to be involved in progression of post-traumatic distress following deployment?

We hypothesized that pre-deployment levels of post-traumatic distress and change in post-traumatic distress during deployment would be predicted by early life trauma and deployment stressors, respectively. We also hypothesized that change in posttraumatic distress (slope) over 2 years following deployment would be predicted by life stressors reported 1 year following return from deployment in HCSE soldiers, but not in LCSE soldiers. Thus, we hypothesized a sensitization effect in the HCSE group.

Methods

Participants and Procedures

This study is part of a prospective cohort study in the Dutch Department of Defense. From 2006 to 2010, The Netherlands deployed approx. 20,000 soldiers to Afghanistan as part of an International Security Assistance Force (ISAF). Participants in this study volunteered to participate prior to a 4-month deployment to Afghanistan between 2006 and 2008. Duties during deployment consisted of combat patrols, clearing or searching buildings, participation in demining operations, and transportation across enemy territory. Combat experiences included exposure to enemy fire, participation in armed combat, seeing seriously injured comrades and civilians, and witnessing the death of fellow soldiers and civilians. The study comprised 5 assessments: approximately 2 months prior to deployment (T1) and approximately 2 (T2), 7 (T3), 14 (T4), and 26 months (T5) following return from deployment. The first three assessments took place at military bases, and the last two assessments were mailed-in. At T1, $N = 814$ constituted the initial sample. The number of participants at T2 was $N = 693$ (85.1 % of the initial sample); at T3: $N = 644$ (79.1 %); at T4: $N = 465$ (57.1 %); and at T5: $N = 433$ (53.2 %). Of the 814 participants at T1, $N = 345$ (42.4 %) participated in all 5 assessments; $N = 146$ (17.9 %) participated in 4, $N = 167$ (20.5 %) in 3, $N = 82$ (10.1 %) in 2, and $N = 74$ (9.1 %) in 1 assessment. The study was approved by the Institutional Review Board of the University Medical Center Utrecht, The Netherlands. Written consent was obtained after a written and verbal description of the study.

Measures

Socio-demographic characteristics

Participants were asked about number of previous deployments, rank during deployment, age during deployment, gender, and education level. In addition, participants indicated whether their function during deployment was inside the military base or compound only or outside the compound as well. Participants whose function during deployment was outside the compound comprised the HCSE group, and those having worked inside the compound the LCSE group.

Early life trauma

Exposure to potential traumatic experiences before the age of 18 was assessed at T1 using the Early Trauma Inventory (ETI) self-report short form [26], Dutch version [27]. The ETI is designed to assess exposure to potential traumatic experiences before the age of 18 years (general trauma, physical abuse, emotional abuse and sexual abuse) and consists of 27 dichotomous items. The total score represents the number of experienced events.

Deployment stressors

At T2, exposure to potentially traumatic deployment stressors was assessed with a 13-item Deployment Stressors Checklist specifically developed for this study [28]. Items refer to specific events, for example, "Exposure to enemy fire (yes/no)", "Being the target of enemy fire (yes/no)", and "Being held at gunpoint (yes/no)" (the full text of the checklist was published previously [28]). A total score was obtained by summing affirmative responses.

Post-deployment stressors

Exposure to stressful life events in the first year after return from deployment was assessed at T4 using a 10-item yes/no checklist specifically developed for this study (see the data supplement). The checklist comprised items about divorce or broken relationship, accident or assault to self or close other, severe illness to self or close other, death of a close other, burglary or fire in own home, financial problems, and being dismissed. A total score was obtained representing the number of endorsed stressors.

Self-report inventory for PTSD

PTSD symptom level over the past 4 weeks was assessed with the 22-item Self-Report Inventory for PTSD (SRIP) [29, 30]. A higher score indicates more PTSD symptoms (range 22–88), i.e., higher levels of post-traumatic distress. The SRIP has good concurrent validity with other PTSD measures such as the Clinician Administered PTSD Scale and Mississippi Scale for PTSD [30]. The SRIP does not assess symptoms of PTSD with reference to a specific event. Measurement invariance testing revealed that no substantial differences in PTSD factor structure exist if PTSD is assessed in trauma-exposed participants with vs. without reference to a single traumatic event [31]. Participants were assigned a probable PTSD diagnosis when their score on the SRIP was $\geq$ 38 [28]. This cutoff score corresponds to the mean plus two standard deviations, which coincides with the 95th percentile of scores before deployment within a population of 704 soldiers from the Dutch Armed Forces [(mean: 26.91 SD = 5.34)]. The validity of this cutoff score is supported by a study [29] that compared SRIP scores with ratings from a diagnostic interview within a community population of older adults, showing adequate sensitivity and specificity.

Analyses

Analyses were performed using SPSS/Amos software versions 20.0 and 17.0, respectively. For our structural equation modeling (SEM) analyses, missing data due to attrition were handled using the full information maximum likelihood (FIML) procedure. It has been shown that under ignorable missing data conditions, FIML estimates are unbiased, and the bias in FIML parameter estimates is relatively unaffected by the amount of missing data [32].

Descriptive analyses

Using Chi-square tests for categorical variables and *t* tests for continuous variables, we evaluated differences between the sample completing all assessments and the dropout sample and between the HCSE and LCSE groups. Data screening revealed moderate to severe non-normality in PTSD symptom scores, early trauma, and number of previous deployments. This non-normality was expected because low scores on these variables were the most common, and higher scores increasingly rare. We, therefore, replicated our SEM analyses using Bayesian estimation (see below).

Latent growth modeling (LGM)

Progression of post-traumatic distress may be flexibly modeled using latent growth models. Interest of the present analysis centered on changes during two distinct time periods, specifically during deployment (from T1 to T2) and following deployment (from T2 to T5). Piecewise growth models can be used to subdivide a series of measurements of PTSD into meaningful segments and to summarize important aspects of change in each segment [33]. We specified a piecewise growth model comprising three factors: (1) pre-deployment (baseline) posttraumatic stress symptom level (intercept); (2) linear change (slope) in posttraumatic distress during deployment (pre-to-post); and (3) linear change (slope) in posttraumatic distress after deployment. The average posttraumatic stress symptom level can be expressed using this model as the sum of the pre-deployment level, the change during deployment, and the change after deployment (slope * time after deployment). Therefore, the baseline level factor loaded on all five indicators (T1–T5) with factor loadings set to 1 (see Fig. 1). The slope during deployment factor loaded on the four post-deployment indicators (T2–T5) with factor loadings set to one to capture the change during the period from pre- to post-deployment. The slope post-deployment factor loaded on the indicators assessed at the 7–26-month follow-up (T3 to T5). Factor loadings were set to 0.42, 1, and 2, respectively, to capture the mean time in years since the T2 assessment. Residual variances associated with the T1 and T2 assessments were constrained to be equal to enable the model to be identified. We evaluated model fit using the discrepancy χ^2, comparative fit index (CFI), non-normed fit index (NNFI/TLI), root-mean-square error of approximation (RMSEA), and Akaike information criterion (AIC). Models that fit very well are indicated by CFIs and NNFIs $\geq$ 0.95 and RMSEAs $\leq$ 0.06 [34].

Construction of a MIMIC model

To explore predictor effects on levels and course of post-traumatic distress, we constructed a multiple indicators, multiple causes (MIMIC) model [25]. MIMIC models are a broad class of structural equation models where exogenous

observed variables influence latent variables that in turn have multiple indicators. We hypothesized that the early trauma would predict pre-deployment distress; deployment stressors would predict linear change during deployment; and post-deployment stressors would predict linear change post-deployment. To adjust for possible confounding effects, we included gender, age, education, rank, and number of previous deployments as covariates in the model with paths to all factors.

Multiple group analysis

We applied multiple group analysis to test whether high combat stress exposure moderated the relation between post-deployment stressors and linear change in posttraumatic stress post-deployment. In multiple group analysis, a model is estimated simultaneously across groups. Through the specification of cross-group equality constraints, group differences on any individual parameter or set of parameters can be tested [35]. The fit of the model with parameters constrained to be equal across groups is compared with that of the unrestricted model with the $\Delta\chi^2$ test [35]. Thus, we examined differences in paths and factor means between the HCSE and LCSE groups by applying the MIMIC model to these groups simultaneously and subsequently testing models corresponding to increasingly restricted hypotheses. Specifically, we subsequently applied cross-group equality constraints corresponding to the following nil-hypotheses: equality of early trauma effects on baseline posttraumatic distress level; equality of deployment stressor effects on slope during deployment; equality of post-deployment stressor effects on slope post-deployment; equality of adjusted baseline levels; equality of adjusted slopes during deployment; and equality of adjusted slopes post-deployment (see Table 1).

Bayesian replication

Following construction of our final models, we tested robustness of the maximum likelihood (ML) estimation method against normality violations in our data. Therefore, we repeated all analyses using Bayesian estimation, which is not based on normality assumptions [36]. For our Bayesian implementation of SEM, we used the default settings in Amos [36], including an uninformative flat prior ranging from $-3.4 * 10^{38}$ to $3.4 * 10^{38}$. The Markov Chain Monte Carlo (MCMC) sampling for the Bayesian estimation was continued until subsequent runs were sufficiently uncorrelated, i.e., when the value of the Gelman–Carlin–Stern–Rubin convergence statistic was less than the conservative default value of 1.002. Because the Bayesian and FIML estimates were essentially identical, we concluded that ML estimation was robust against normality violations in our data and chose to present FIML estimates. (The Bayesian estimates are presented in Supplementary Table 4.)

Results

Attrition and descriptive analyses

Study attrition was associated with the following baseline characteristics: younger age (25.16 vs. 30.25, $p < 0.001$), lower (soldier or corporal) rank (76.9 vs. 49.1 %, $p < 0.001$), lower education (51.2 vs. 28.6 %, $p < 0.001$), and fewer previous deployments (0.67 vs. 1.07, $p < 0.001$). In addition, study attrition was associated with working outside the compound (79.2 % vs. 57.8 %, $p < 0.001$), more deployment stressors (3.87 vs. 3.40, $p = 0.030$), and higher PTSD symptoms at T3 (29.16 vs. 27.16, $p < 0.001$)

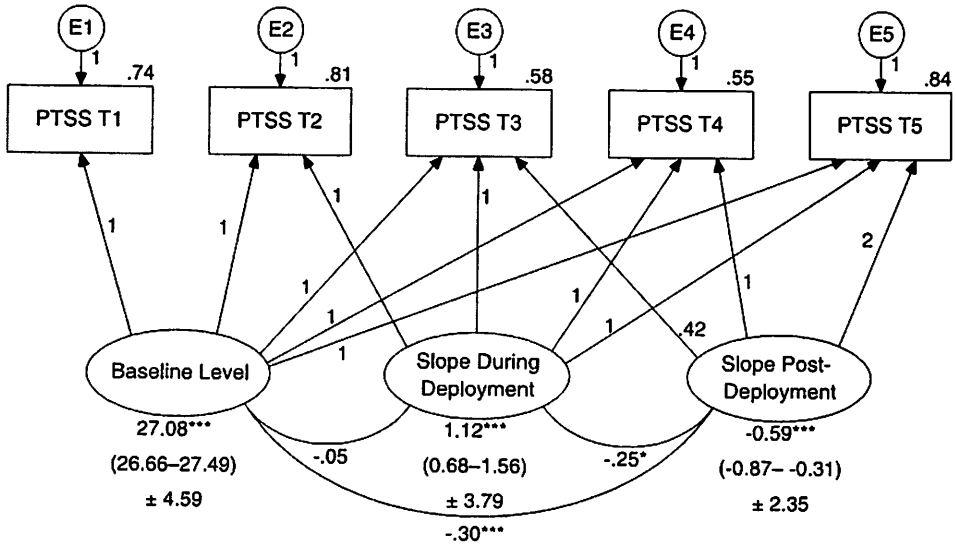

Fig. 1 *Piecewise Growth Model.* For the model: $\chi^2(7) = 14.26$, $p = 0.047$, CFI = 0.99, NNFI = 0.98, RMSEA = 0.04, AIC = 40.26. Factors (*ovals*) are shown with unadjusted means (95 % confidence intervals) ± SD, factor loadings (*arrows*) with fixed regression weights, covariances (*curved lines*) with correlations, and indicators (*squares*) with explained variance (R^2). *PTSS* posttraumatic stress symptoms. E1 to E5 represent residual error variances. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 1 Multiple group model selection

Nr.	Ref.[a]	Cross-group equality constraints[b,c]	*df*	χ^2	*p*	Δdf	$\Delta\chi^2$	*p*	CFI	NNFI	RMSEA	AIC
1	–	Unconstrained	38	67.03	0.003				0.97	0.95	0.03	167.03
2	1	Early trauma effects equal across groups	39	67.03	0.003	1	0.00	0.953	0.98	0.95	0.03	165.03
3	2	Deployment stressor effects equal across groups	40	67.16	0.005	1	0.13	0.723	0.98	0.96	0.03	163.16
4	**3**	**Post-deployment stressor effects equal across groups**	**41**	**75.01**	**0.001**	**1**	**7.85**	**0.005**	**0.97**	**0.95**	**0.03**	**169.01**
5	3	Adjusted baseline level equal across groups	41	67.62	0.006	1	0.46	0.496	0.98	0.96	0.03	161.62
6	5	Adjusted slope during deployment equal across groups (Final)	42	68.48	0.006	1	0.86	0.353	0.98	0.96	0.03	160.48
7	**6**	**Adjusted slope post-deployment equal across groups**	**43**	**72.54**	**0.003**	**1**	**4.06**	**0.044**	**0.97**	**0.96**	**0.03**	**162.54**

Estimates from the final model are presented in Table 3

[a] Reference model nr

[b] Models represent the reference model with equality constraint(s) added; the models shown in bold were rejected because of a significant worsening of the model fit

[c] Groups: 1. Low combat stress exposed ($N = 227$), 2. High combat stress exposed ($N = 520$)

as well as T4 (28.33 vs. 26.67, $p = 0.036$). Study participants who dropped out from one or more study assessments did not differ from those who completed all assessments in gender, early trauma, post-deployment stressors, and PTSD symptoms at T1, T2, and T5. Missing data due to attrition were handled using FIML, which is bias-free under ignorable missing data conditions [32]. Because demographic variables (age, rank, education), number of previous deployments, and exposure variables associated with study attrition were correlated, and analyses corrected for demographic variables and number of previous deployments (see below) only showed negligible differences from the unadjusted results, and because PTSD symptoms at baseline did not predict study attrition, we concluded that there were no indications of meaningful nonresponse bias.

Descriptive analyses are reported in Table 2. Of $N = 455$ participants providing sufficient data to establish a diagnosis of probable deployment-related PTSD across at least four assessments, $n = 22$ were excluded because they endorsed PTSD before deployment. Participants who did not endorse PTSD at any follow-up assessment ($n = 368$) constituted 85.0 % of the sample. Thirty-five participants (8.1 %) endorsed probable PTSD at 2 months post-deployment. Probable delayed-onset PTSD, defined as endorsing probable PTSD for the first time 7, 14, and/or 26 months after deployment, was found in 30 participants (6.9 %).

Differences between high vs. low combat stress exposed groups are also shown in Table 2. The HCSE group was younger, more often male, lower educated and ranked than the LCSE group. In addition, the HCSE group reported fewer previous deployments, more deployment stressors, and more post-deployment stressors than the LCSE group. We, therefore, corrected for these variables in separate analyses (see below). As expected, the HCSE group reported higher PTSD symptoms at 1 and 6 months after deployment, more probable PTSD at T2, and more probable delayed-onset PTSD (see Table 2). The percentage of participants meeting probable PTSD (including delayed-onset PTSD) threshold levels in the HCSE group (19.0 %) was over twice the percentage in the LCSE group (9.1 %).

Change in post-traumatic distress during and after deployment

Figure 1 depicts the piecewise growth model for the whole sample. As shown in Fig. 1, the mean increase in post-traumatic distress during deployment was modest, equaling 1.12 points on the SRIP (95 % CI = 0.68–1.56, $p < 0.001$). Following deployment, there was a significant linear decrease in mean distress, equaling −0.59 points on the SRIP each year (95 % CI = −0.87 to −0.31, $p < 0.001$). The model fits the data well (see model fit indices in Fig. 1). The three factors pre-deployment level, slope during deployment, and slope post-deployment explained large proportions of the variance in PTSD symptoms at all assessments (55–85 %; see Fig. 1). Not withstanding, all residual variances were significant (data not shown), indicating that the longitudinal course of symptoms over time departed from linearity in some individuals.

Stressor effects on post-traumatic distress

To examine effects of stressor exposure on pre-deployment level of posttraumatic distress, slope during deployment, and

Table 2 Descriptive analyses

	Full sample ($N = 814$ at T1)			Low combat stress exposed ($N = 227$)		High combat stress exposed ($N = 520$)		
	Mean	SD	Range	Mean	SD	Mean	SD	Diff.[b]
Age (years) during deployment	27.33	8.16	18–60	32.98	9.68	24.82	6.09	***
Number of previous deployments	0.84	1.15	0–6	1.20	1.28	0.66	1.06	***
Early Trauma Inventory score	3.22	2.91	0–17	3.12	3.12	3.22	2.78	
Exposure to deployment stressors	3.99	2.58	0–10	2.37	1.95	4.68	2.52	***
Stressful life events 1 year post-deployment	0.76	0.96	0–4	0.64	0.91	0.88	0.99	***
Self-Rating Inventory for PTSD total score								
Before deployment	27.05	5.48	22–74	26.96	5.58	27.12	5.52	
2 months after deployment	28.08	6.43	22–61	27.37	5.80	28.34	6.53	
7 months after deployment	28.09	7.36	22–82	26.90	6.71	28.70	7.67	**
14 months after deployment	27.20	7.08	22–86	26.28	6.72	27.80	7.31	*
26 months after deployment	26.75	6.19	22–73	26.24	5.89	27.14	6.51	
	N	%		*N*	%	*N*	%	Diff.[b]
Gender								
Male	740	90.1		185	81.5	495	95.2	***
Female	74	9.9		42	18.5	25	4.8	
Education								
Lower	302	41.37		58	28.6	224	46.6	***
Middle	360	49.32		103	50.7	237	49.3	
Higher	68	9.32		42	20.7	20	4.2	
Rank during deployment								
Soldier	348	43.77		34	15.0	299	57.7	***
Corporal	168	21.13		46	20.4	110	21.2	
Noncommissioned officer	181	22.77		89	39.4	76	14.7	
Officer	98	12.33		57	25.2	33	6.4	
Trajectory[a]								
No PTSD	368	85.0		149	90.9	209	81.0	*
Probable PTSD at T2	35	8.1		7	4.3	27	10.5	
Probable delayed-onset PTSD	30	6.9		8	4.9	22	8.5	

Total numbers vary due to missing responses

[a] Participants providing sufficient data to establish a diagnosis of probable deployment-related PTSD across at least four assessments, excluding $N = 22$ who endorsed PTSD before deployment

[b] Differences between high vs. low combat stress exposed groups

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

slope post-deployment, we created a MIMIC model, i.e., the piecewise growth model with added predictor variables. This model fits the data well. A path diagram is shown in Fig. 2. As expected, baseline level and slope during deployment of post-traumatic distress were significantly associated with early trauma (standardized regression weight = 0.30, $p < 0.001$) and exposure to deployment stressors (standardized regression weight = 0.21, $p < 0.001$), respectively. For the sample as a whole, post-deployment stressors had no significant effects on slope of PTSD symptoms post-deployment ($p = 0.114$). Estimates from this model as well as model fit indices are presented in Table 3.

Stress sensitization in high combat stress exposed soldiers

To examine whether high combat stress exposure moderated the relation between post-deployment stressors and

linear change in post-traumatic distress post-deployment, we applied the MIMIC model to the HCSE and LCSE groups simultaneously and subsequently tested models corresponding to increasingly restricted hypotheses. The multiple group model selection is shown in Table 1. The unconstrained model (i.e., paths and factors not set to be equal across groups; model nr. 1 in Table 1) showed adequate model fit. In model nr. 2, early trauma effects were constrained to be equal across groups. The $\Delta\chi^2$ test showed that the model fit did not worsen significantly, indicating that early trauma effects on pre-deployment level were equal across groups. Thus, this constraint was maintained in subsequent models. The model constraining deployment stressor effects on slope during deployment to be equal across groups (nr. 3) again did not show worsening of model fit, compared with the preceding model. However, the model constraining post-deployment stressor effects on slope post-deployment to be equal across groups (nr. 4) showed a significant worsening of the model fit compared with the preceding model. Therefore, model nr. 4 was rejected and the constraint was not maintained in subsequent models. The model constraining adjusted baseline levels to be equal across groups (nr. 5) and the model constraining adjusted slopes during deployment to be equal across groups (nr. 6) again did not show a worsening of model fit, compared with the respective preceding models. The model constraining adjusted slopes post-deployment to be equal across groups (nr. 7) showed a significant worsening of the model fit compared with the preceding model. Therefore, model nr. 7 was rejected, and model nr. 6 became the final most parsimonious model.

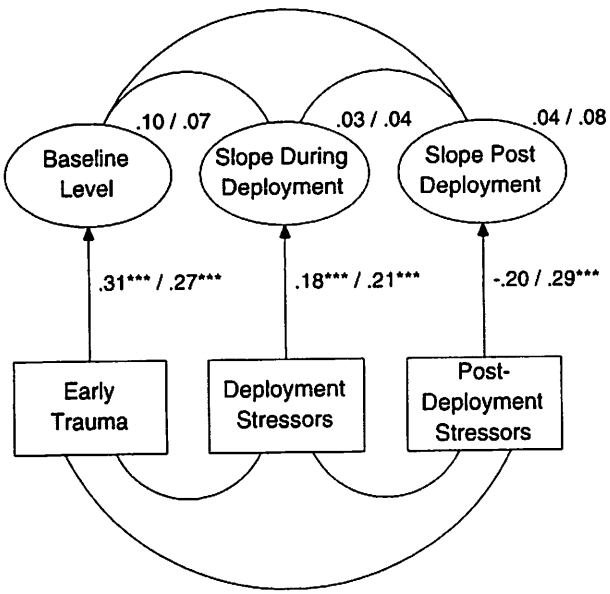

Fig. 2 *Final Multiple Group Model.* For the model: $\chi^2(42) = 68.48$, $p = 0.006$, CFI = 0.98, NNFI = 0.96, RMSEA = 0.03, AIC = 160.48. Factors (*ovals*) are shown with explained variance (R^2), paths with standardized regression weights for the LCSE/HCSE groups, respectively. *Curved lines* represent covariances. Indicators PTSS T1 to T5 (see Fig. 1) as well as residual error variances are not shown. See Table 3 for factor intercepts and unstandardized regression weights

Estimates from the final multiple group model (model nr. 6 in Table 1) are presented in Table 3. Early trauma effects on baseline level and deployment stressor effects on slope during deployment were equal across groups. Early trauma explained 10 and 7 % of variance in the LCSE and HCSE groups, respectively. Deployment stressors explained 3 and 4 % of variance in slope during deployment in the LCSE and HCSE groups, respectively. Post-deployment stressors effects on slope post-deployment were not significant in the LCSE group. In contrast, in the HCSE group, slope post-deployment was strongly predicted by post-deployment stressors (standardized regression weight = 0.29, $p < 0.001$). Within the HCSE group, for each reported post-deployment stressor, the mean SRIP score increased by 0.88 points (95 % CI = 0.37–1.38, $p < 0.001$) per year. Post-deployment stressors explained 4 % of variance in slope post-deployment in the LCSE group, against 8 % in the HCSE group.

Factor means and confidence intervals are also reported in Table 3. The factors representing baseline level of post-traumatic distress (adjusted for early trauma) and slope during deployment (adjusted for deployment stressors) were equal across groups. The factor representing the slope post-deployment (adjusted for post-deployment stressors) differed between the LCSE and HCSE groups. Within the HCSE group, the adjusted mean SRIP score decreased 1.22 points per year (95 % CI = −1.84 to −0.60, $p < 0.001$). (Note that adjusted in this context means: when post-deployment stressor effects equal zero.) This decrease was steeper than in the LCSE group, where the adjusted mean SRIP score decreased 0.44 points per year (95 % CI = −0.77 to −0.06, $p = 0.023$). This difference appears to represent regression of elevated PTSD scores to the mean, reflecting an overall tendency towards recovery and resilience. Thus, HCSE soldiers were likely to recover from combat-related distress in the absence of post-deployment stressors. In the presence of post-deployment stressors, they were likely to experience persistence or progression of post-traumatic distress during the 2 years following return from deployment.

Analyses adjusted for demographic variables and previous deployments

To adjust for possible confounding effects, we included gender, age, education, rank, and number of previous deployments as covariates in the MIMIC and multiple group models with paths to all factors. Because the relevant results of adjusted analyses only showed negligible differences from

Table 3 Model estimates

Parameter	Mean/coeff.	(95 % CI)	Std. coeff.	*p*
	Full sample ($N = 814$)			
	Piecewise growth model[a]			
Baseline level	27.08	(26.66 to 27.49)	–	0.000
Slope during deployment	1.12	(0.68 to 1.56)	–	0.000
Slope post-deployment	−0.59	(−0.87 to −0.31)	–	0.000
	MIMIC model[b]			
Early trauma → baseline level	0.48	(0.36 to 0.60)	0.30	0.000
Deployment stressors → slope during deployment	0.31	(0.16 to 0.45)	0.21	0.000
Post-deployment stressors → slope post-deployment	0.27	(−0.06 to 0.60)	0.11	0.114
Baseline level	25.55	(24.99 to 26.11)	–	0.000
Slope during deployment	−0.12	(−0.85 to 0.61)	–	0.745
Slope post-deployment	−0.78	(−1.16 to −0.39)	–	0.000
	Multiple group model[c]			
	LCSE group ($N = 227$)			
Early trauma → baseline level	0.46	(0.34 to 0.59)	0.31	0.000
Deployment stressors → slope during deployment	0.33	(0.18 to 0.48)	0.18	0.000
Post-deployment stressors → slope post-deployment	**−0.19**	**(−0.53 to 0.14)**	**−0.20**	**0.114**
Baseline level	25.60	(25.02 to 26.19)	–	0.000
Slope during deployment	−0.29	(−1.01 to 0.43)	–	0.424
Slope post-deployment	**−0.41**	**(−0.77 to −0.06)**	–	**0.023**
	HCSE group ($N = 520$)			
Early trauma → Baseline level	0.46	(0.34 to 0.59)	0.27	0.000
Deployment stressors → Slope during deployment	0.33	(0.18 to 0.48)	0.21	0.000
Post-deployment stressors → Slope post-deployment	**0.88**	**(0.37 to 1.38)**	**0.29**	**0.000**
Baseline level	25.60	(25.02 to 26.19)	–	0.000
Slope during deployment	−0.29	(−1.01 to 0.43)	–	0.424
Slope post-deployment	**−1.22**	**(−1.84 to −0.60)**	–	**0.000**

Coeff coefficient, *std* standardized

Means indicate mean adjusted self-rating inventory for PTSD total scores

[a] For the model: $\chi^2(7) = 14.26$, $p = 0.047$, CFI = 0.99, NNFI = 0.98, RMSEA = 0.04, AIC = 40.26. See path diagram in Fig. 1

[b] For the model: $\chi^2(19) = 43.44$, $p = 0.001$, CFI = 0.98, NNFI = 0.96, RMSEA = 0.04, AIC = 93.44. See path diagram in Fig. 2

[c] For the model: $\chi^2(42) = 68.48$, $p = 0.006$, CFI = 0.98, NNFI = 0.96, RMSEA = 0.03, AIC = 160.48. Parameters displayed in **bold** were **not** constrained to be equal across groups. See model selection in Table 1 (model nr. 6)

the unadjusted results, we concluded there were no confounding effects of demographic variables and previous deployments. The results of the adjusted analyses are presented in Supplementary Table 3. The multiple group model selection is presented in Supplementary Table 2.

Discussion

Our results demonstrate that high combat stress exposed (HCSE) soldiers responded more strongly to stressful life events during the first year after deployment compared with low combat stress exposed (LCSE) soldiers. Specifically, post-deployment stressors predicted persistence or progression of post-traumatic distress in HCSE soldiers, whereas no such predictive effects were found in LCSE soldiers. Consistent with earlier findings, more early life trauma was associated with higher baseline levels of post-traumatic distress [17], and more exposure to deployment stressors was associated with stronger increases in post-traumatic distress during deployment [15].

To the best of our knowledge, our study is the first to provide direct evidence that stress sensitization serves well as a model explaining progression of post-traumatic distress. In addition, our findings provide prospective support for the concept of the stress sensitization. At present, most of the evidence in support of sensitization is based on studies of trauma-exposed populations, showing elevated

risk of PTSD in individuals reporting prior trauma exposure [37]. A recent prospective study highlighted the strongly increased risk of PTSD following repeated sexual victimization [38]. A prospective study in disaster survivors found direct evidence for stress sensitization during the first years following a disaster in survivors reporting total home destruction due to the disaster [25]. In a prospective study in young children, trauma-exposed children with current life stressors had elevated internalizing and externalizing problems compared with trauma-exposed children without current stress and non-trauma-exposed children with and without current stressors [39], consistent with stress sensitization.

Stress sensitization may involve a repertoire of neurobiological mechanisms [40]. Changes in functioning of the hypothalamus–pituitary–adrenal axis, autonomic nervous system, as well as brain regions associated with threat detection and fear expression (e.g., amygdala) may be implicated in sensitized (behavioral) responses after stressor exposure. The functioning of these systems and circuits can be altered by prolonged, severe or traumatic stress [41]. In addition, the functioning of these systems is dysregulated in individuals who developed PTSD [42]. Moreover, several recent studies have shown that vulnerabilities in the functioning of these systems, as assessed prior to or shortly after the trauma exposure leading to PTSD, are associated with subsequent development of PTSD [28, 43]. Future research is needed to indicate whether identification of risk of stress sensitization based on stressor exposure may be complemented by neurobiological markers of stress sensitization.

Stress sensitization also involves cognitive processes used for meaning attribution that may be understood within Conservation of Resources (COR) theory [25]. COR theory states that people strive to retain, protect, and build resources and that what constitutes a stressor to them is the potential or actual loss of these resources [44]. According to this theory, resource loss is disproportionally more salient than resource gain. Therefore, those who already lack resources are more vulnerable to resource loss [44]. Soldiers exposed to direct combat stressors may be at increased risk of resource loss, for example, due to negative perceptions of the mission [6] or deployment-related distress or impairment. Progression of post-traumatic distress may thus reflect increased vulnerability to further resource loss in combat-exposed soldiers.

The clinical relevance of the differences in stress sensitivity between the LCSE and HCSE groups may be illustrated by the finding that the percentage of participants meeting probable PTSD (including delayed-onset PTSD) threshold levels in the HCSE group (20.1 %) was over twice the percentage in the LCSE group (9.3 %). In addition, in our sample, HCSE soldiers reported more stressors post-deployment than LCSE soldiers. Higher levels of posttraumatic distress in the HCSE group may in turn confer an increased risk of interpersonal stressors due to conflicts or violence [10]. Notably, more post-deployment stressors do not by themselves explain their stronger effect (i.e., a stronger effect of each reported stressor) on slope post-deployment in the HCSE group.

Strengths of the current study include the true prospective design, the inclusion of a pre-deployment assessment, as well as the duration of follow-up covering 2 years following return from deployment. However, we also acknowledge some methodological limitations to this study. First, the distress assessments were restricted to participant self-report. It has been suggested that military personnel may be reluctant to endorse genuine distress from fear of stigma in the military context [6]. Therefore, estimates of distress based on these data may be conservative. Notwithstanding, mean PTSD scores in our sample were far below recommended cutoff scores for probable PTSD, indicating generally mild distress. Second, stressor exposure was assessed by self-report questionnaire. Use of interview assessment of exposure to stressful life events could be considered more valid. Given the large size of our sample, questionnaire assessments were more feasible than interviews. Third, differential attrition in our study represents another potential limitation. Given frequently high rates of attrition in trauma research [45], our completion rates are acceptable. Importantly, symptoms of posttraumatic distress at the initial assessment did not predict attrition, and our adjusted analyses suggested that there were no meaningful effects of differential attrition on our results. Fourth, there were important differences between the HCSE vs. LCSE groups in gender, age, education, rank, and number of previous deployments. To prevent confounding, we corrected for these differences in our analyses.

Our findings have several implications for practice. In addition to levels of distress, levels of stressor exposure during and following deployment play a central role in explaining the trajectory of deployment-related distress. We feel that these should, therefore, be routinely assessed by clinicians dealing with soldiers as well as veterans. Our results suggest that the absence of prominent signs and symptoms of distress immediately following deployment does not preclude distress in the long term that may in part still be related to the deployment. Early interventions may effectively be targeted at high risk groups based on combat exposure [46]. When soldiers and veterans are seeking mental health care at later stages following deployment, progression of posttraumatic distress should seriously be considered.

The duration of the sensitization effects may depend on the intensity as well as the recency of combat stress

exposure [25]; this would be an important avenue for further research. Such research could provide arguments to evaluate the length between deployments to reduce mental health risks for combat-exposed soldiers. Foreseeable stressors and resource losses, including unemployment and physical impairments, may be an effective target for secondary prevention of psychological distress in military personnel. Availability of practical assistance following return from deployment tailored to individual concerns related to the deployment and reintegration is, therefore, essential. Health care providers, social workers, chaplains, commanders, and peer supporters dealing with recently combat-exposed soldiers should be aware that they may show increased responsiveness to subsequent stressful life events. They may signal possible signs of posttraumatic distress, for example, mood instability [47], social withdrawal, increased alcohol use, indifference, disciplinary measures, and thrill-seeking behavior, and motivate individuals to seek appropriate care.

Acknowledgments Funded by a grant from the Dutch Ministry of Defense. The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript. Geert E. Smid, MD, PhD had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The authors thank Col. Kees IJzerman, MD, MPH for his valuable comments and the commanders and troops for their time and effort. The authors also thank Kim Kroezen, Anne Muilwijk, Capt. Maurits Baatenburg de Jong, Capt. Jessie Smulders, Lt. Martijn Derks, and Sgt. Loes van den Boomen for organizing the data acquisition.

Conflict of interest None.

References

1. Engelhard IM, van den Hout MA, Weerts J, Arntz A, Hox JJCM, McNally RJ (2007) Deployment-related stress and trauma in Dutch soldiers returning from Iraq: prospective study. Br J Psychiatry 191:140–145
2. Hoge CW, Castro CA, Messer SC, McGurk D, Cotting DI, Koffman RL (2004) Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. N Engl J Med 351:13–22
3. Thomas JL, Wilk JE, Riviere LA, McGurk D, Castro CA, Hoge CW (2010) Prevalence of mental health problems and functional impairment among active component and National Guard soldiers 3 and 12 months following combat in Iraq. Arch Gen Psychiatry 67:614–623
4. Fear NT, Jones M, Murphy D, Hull L, Iversen AC, Coker B, Machell L, Sundin J, Woodhead C, Jones N, Greenberg N, Landau S, Dandeker C, Rona RJ, Hotopf M, Wessely S (2010) What are the consequences of deployment to Iraq and Afghanistan on the mental health of the UK armed forces? A cohort study. Lancet 375:1783–1797
5. Dickstein BD, Suvak M, Litz BT, Amy BA (2010) Heterogeneity in the course of posttraumatic stress disorder: trajectories of symptomatology. J Trauma Stress 23:331–339
6. Gray MJ, Bolton EE, Litz BT (2004) A longitudinal analysis of PTSD symptom course: delayed-onset PTSD in Somalia peacekeepers. J Consult Clin Psychol 72:909–913
7. Grieger TA, Cozza SJ, Ursano RJ, Hoge C, Martinez PE, Engel CC, Wain HJ (2006) Posttraumatic stress disorder and depression in battle-injured soldiers. Am J Psychiatry 163:1777–1783
8. Goodwin L, Jones M, Rona RJ, Sundin J, Wessely S, Fear NT (2012) Prevalence of delayed-onset posttraumatic stress disorder in military personnel: is there evidence for this disorder?: results of a prospective UK cohort study. J Nerv Ment Dis 200:429–437
9. Solomon Z, Mikulincer M (2006) Trajectories of PTSD: a 20-year longitudinal study. Am J Psychiatry 163:659–666
10. Prigerson HG, Maciejewski PK, Rosenheck RA (2001) Combat trauma: trauma with highest risk of delayed onset and unresolved post-traumatic stress disorder symptoms, unemployment, and abuse among men. J Nerv Ment Dis 189:99–108
11. Smid GE, Mooren TTM, Van der Mast RC, Gersons BPR, Kleber RJ (2009) Delayed post-traumatic stress disorder: systematic review, meta-analysis, and metaregression analysis of prospective studies. J Clin Psychiatry 70:1572–1582
12. Milliken CS, Auchterlonie JL, Hoge CW (2007) Longitudinal assessment of mental health problems among active and reserve component soldiers returning from the Iraq war. JAMA 298:2141–2148
13. Thompson KE, Vasterling JJ, Benotsch EG, Brailey K, Constans J, Uddo M, Sutker PB (2004) Early symptom predictors of chronic distress in Gulf War veterans. J Nerv Ment Dis 192:146–152
14. Li B, Mahan CM, Kang HK, Eisen SA, Engel CC (2011) Longitudinal health study of US 1991 Gulf War Veterans: changes in health status at 10-year follow-up. Am J Epidemiol 174:761–768
15. Kang HK, Natelson BH, Mahan CM, Lee KY, Murphy FM (2003) Post-traumatic stress disorder and chronic fatigue syndrome-like illness among Gulf war veterans: a population-based survey of 30,000 veterans. Am J Epidemiol 157:141–148
16. Smith TC, Wingard DL, Ryan MAK, Kritz-Silverstein D, Slymen DJ, Sallis JF, For the Millennium Cohort Study Team (2008) Prior assault and posttraumatic stress disorder after combat deployment. Epidemiology 19:505–512
17. Smith TC, Ryan MAK, Wingard DL, Slymen DJ, Sallis JF, Kritz-Silverstein D (2008) New onset and persistent symptoms of posttraumatic stress disorder self reported after deployment and combat exposures: prospective population based US military cohort study. BMJ 336:366–371
18. Brewin CR, Andrews B, Hejdenberg J, Stewart L (2012) Objective predictors of delayed-onset post-traumatic stress disorder occurring after military discharge. Psychol Med 42:2119–2126
19. Vasterling JJ, Proctor SP, Friedman MJ, Hoge CW, Heeren T, King LA, King DW (2010) PTSD symptom increases in Iraq-deployed soldiers: comparison with non-deployed soldiers and associations with baseline symptoms, deployment experiences, and post-deployment stress. J Trauma Stress 23:41–51
20. Andrews B, Brewin CR, Stewart L, Philpott R, Hejdenberg J (2009) Comparison of immediate-onset and delayed-onset posttraumatic stress disorder in military veterans. J Abnorm Psychol 118:767–777
21. Horesh D, Solomon Z, Zerach G, Ein-Dor T (2011) Delayed-onset PTSD among war veterans: the role of life events throughout the life cycle. Soc Psychiatry Psychiatr Epidemiol 46:863–870
22. Post RM, Weiss SRB (1998) Sensitization and kindling phenomena in mood, anxiety, and obsessive- compulsive disorders: the role of serotonergic mechanisms in illness progression. Biol Psychiatry 44:193–206

23. Kandel ER, Schwartz JH (1982) Molecular biology of learning: modulation of transmitter release. Science 218:433–443
24. McFarlane A (2010) The long-term costs of traumatic stress: intertwined physical and psychological consequences. World Psychiatry 9:3–10
25. Smid GE, Van der Velden PG, Lensvelt-Mulders GJLM, Knipscheer JW, Gersons BPR, Kleber RJ (2012) Stress sensitization following a disaster: a prospective study. Psychol Med 42:1675–1686
26. Bremner JDM, Bolus RP, Mayer EAM (2007) Psychometric properties of the early trauma inventory-self report. J Nerv Ment Dis 195:211–218
27. Rademaker AR, Vermetten E, Geuze E, Muilwijk A, Kleber RJ (2008) Self-reported early trauma as a predictor of adult personality: a study in a military sample. J Clin Psychol 64:863–875
28. van Zuiden M, Geuze E, Willemen HLDM, Vermetten E, Maas M, Heijnen CJ, Kavelaars A (2011) Pre-existing high glucocorticoid receptor number predicting development of post-traumatic stress symptoms after military deployment. Am J Psychiatry 168:89–96
29. van Zelst WH, de Beurs E, Beekman ATF, Deeg DJH, Bramsen I, van Dyck R (2003) Criterion validity of the self-rating inventory for post-traumatic stress disorder (SRIP) in the community of older adults. J Affect Disord 76:229–235
30. Hovens JE, Bramsen I, Van der Ploeg HM (2002) Self-rating inventory for post-traumatic stress disorder: review of the psychometric properties of a new brief Dutch screening instrument. Percept Mot Skills 94:996–1008
31. Elhai JD, Engdahl RM, Palmieri PA, Naifeh JA, Schweinle A, Jacobs GA (2009) Assessing posttraumatic stress disorder with or without reference to a single, worst traumatic event: examining differences in factor structure. Psychol Assess 21:629–634
32. Enders CK, Bandalos DL (2001) The relative performance of full information maximum likelihood estimation for missing data in structural equation models. Struct Equ Model 8:430–457
33. Duncan TE, Duncan SC, Strycker LA (2006) An Introduction to Latent Variable Growth Curve Modeling: Concepts, Issues, and Applications. Lawrence Erlbaum Associates, Mahwah (NJ)
34. Hu LT, Bentler PM (1999) Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. Struct Equ Model 6:1–55
35. Kaplan DW (2008) Structural Equation Modeling: Foundations and Extensions. Sage, Thousand Oaks (CA)
36. Arbuckle JL (2009) Amos 18.0 User's Guide. Amos Development Corporation, Crawfordville, Florida
37. Breslau N, Chilcoat HD, Kessler RC, Davis GC (1999) Previous exposure to trauma and PTSD effects of subsequent trauma: results from the Detroit area survey of trauma. Am J Psychiatry 156:902–907
38. Walsh K (2012) National prevalence of posttraumatic stress disorder among sexually revictimized adolescent, college, and adult household-residing women. Arch Gen Psychiatry 69:935–942
39. Grasso DJ, Ford JD, Briggs-Gowan MJ (2012) Early life trauma exposure and stress sensitivity in young children. J Pediatr Psychol 38:94–103
40. Stam R (2007) PTSD and stress sensitisation: a tale of brain and body: part 1: human studies. Neurosci Biobehav Rev 31:530–557
41. Van Wingen GA, Geuze E, Vermetten E, Fernández G (2011) Consequences of combat stress on brain functioning. Mol Psychiatry 16:583
42. Morris MC, Compas BE, Garber J (2012) Relations among posttraumatic stress disorder, comorbid major depression, and HPA function: a systematic review and meta-analysis. Clin Psychol Rev 32:301–315
43. van Zuiden M, Geuze E, Willemen HLDM, Vermetten E, Maas M, Amarouchi K, Kavelaars A, Heijnen CJ (2012) Glucocorticoid receptor pathway components predict post-traumatic stress disorder symptom development: a prospective study. Biol Psychiatry 71:309–316
44. Hobfoll SE (2001) The influence of culture, community, and the nested-self in the stress process: advancing conservation of resources theory. Appl Psychol 50:337–421
45. Scott CK, Sonis J, Creamer M, Dennis ML (2006) Maximizing follow-up in longitudinal studies of traumatized populations. J Trauma Stress 19:757–769
46. Adler AB, Bliese PD, McGurk D, Hoge CW, Castro CA (2009) Battlemind debriefing and battlemind training as early interventions with soldiers returning from Iraq: randomization by platoon. J Consult Clin Psychol 77:928–940
47. Marwaha S, Parsons N, Broome M (2013) Mood instability, mental illness and suicidal ideas: results from a household survey. Soc Psychiatry Psychiatr Epidemiol (Online) 1–7