The Impact of Repression, Hostility, and Post-Traumatic Stress Disorder on All-Cause Mortality

A Prospective 16-Year Follow-up Study

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Abstract: A common assumption is that repression of traumatic memories is harmful to health. To assess this, we examined all-cause mortality among a national random sample of 4462 male US Army veterans evaluated in 1985 and followed up in 2000. Our hypothesis was that repression on the Welsh R scale would be associated with increased future mortality. We also expected to find a repression × post-traumatic stress disorder (PTSD) interaction effect. Multivariate Cox regression results for all veterans and for theater veterans (Vietnam service) and era veterans (no Vietnam service) separately, revealed that while PTSD was significant in all models, no main or interaction effect was found for repression. In addition, for era veterans, higher repression symptoms were protective for future mortality (HR = 0.95, $p = 0.03$). For hostility symptoms, although no interaction effect was found by PTSD, a positive main effect was detected for hostility, but only for theater veterans (HR = 1.04, $p = 0.034$). Disease-specific results were nonsignificant. Similar to a recent study, we also found that repression symptoms were negatively correlated with PTSD symptoms ($r = -0.109$, $p < 0.001$), suggesting repression might be protective. Our study found no evidence that repression had an adverse health impact on men exposed to psychological trauma.

Key Words: Repression, hostility, mortality, post-traumatic stress disorder, survival analysis.

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Studies suggest that having post-traumatic stress disorder (PTSD) is associated with increased morbidity and mortality (Boscarino, 2008a; 2008b; 2006a; 2006b; Felitti et al., 1998; Schnurr and Green, 2004). Several etiologic pathways have been advanced to explain this association, including alterations in the hypothalamic-pituitary-adrenocortical stress axis (Boscarino, 2004), the onset of harmful health behaviors brought on by PTSD psychopathology (Schnurr & Green, 2004), confounding by genetic and personality factors (Boscarino, 2006b; Boscarino and Hoffman, 2007), and other plausible psychobiological mechanisms (Boscarino, 2008a; 2008b).

It is clinically significant that trauma exposures are often associated with an intense emotional response (American Psychiatric Association, 2000; Boscarino & Hoffman, 2007). The role of these emotional responses in survival and adaptation has been noted (Horowitz et al., 1990; Hung et al, 1999; Izard, 1993; Kiecolt-Glaser et al, 2002; Wastell, 2002). Emotional responsiveness to environmental events is thought to generate adaptive behaviors that enable adjustment to environmental demands (Horowitz et al., 1990; Hung et al., 1999; Izard, 1993; Wastell, 2002). Those with PTSD commonly report not only that traumatic memories result in re-experiencing of past traumatic events, but also that this occurs with little diminution of emotional intensity (Boscarino, 1996). It has been noted that during traumatic exposures many persons appear to use automated control processes characterized by cognitive disassociation that seem to assist in functioning during traumatic events (Horowitz et al., 1990; Jones, 1993; Wastell, 2002). Thus, emotional response during psychological trauma exposure is not only central during the event, but it is also connected to development of traumatic memories of the event (Brewin, 2005; van der Kolk, 1996).

Concurrent with these clinical observations, it is suggested that cognitions that disrupt processing of trauma memories may create future stress-related illnesses (Wastell, 2002). This conceptualization asserts that if emotional reactions remain cognitively unintegrated and emotion-suppressing defensives become established, the long-term physical effects will be detrimental (Jones, 1993). This repressed emotional state is thought to prevent psychological recovery, increase levels of anxiety and depression, and to result in adverse health outcomes (Clohessy and Ehlers, 1999; Weihs et al., 2008). Consequently, it is typically suggested that it is important to achieve emotional expression and cognitive reintegration after traumatic exposures to avoid untoward health effects (Horowitz et al., 1990; Pennebaker and Seagal, 1999). This conceptualization appears consistent with PTSD symptom manifestation, which often includes disturbed sleep, avoidance symptoms, intrusive images, hyperalertness, and social withdrawal (American Psychiatric Association, 2000). In summary, a central hypothesis related to the concept of repression in psychology and psychiatry is that traumatic stress exposures are associated with greater use of cognition-suppressing defenses and that this type of cognitive defense is potentially harmful to future health (Horowitz et al., 1990; Schwartz, 1990).

The objective of the current study is to assess the health consequences of psychological repression as this relates to PTSD. Our hypothesis is that a repressive personality trait, as measured by the Welsh R scale, concurrent with PTSD would be a significant predictor of poorer health outcomes, as reflected in increased mortality at follow-up (Bryant and Harvey, 1995). In particular, we expected that the impact of repression would be observed both as a main effect and as an interaction effect between PTSD and a repressive personality trait. In our study, we also assessed the impact of hostility and PTSD on these same outcomes (Ouimette et al., 2004). Researchers have often reported a positive association between higher hostility and adverse health outcomes (Boyle et al., 2005; Bunde & Suls, 2006; Iribarren et al., 2005). However, in the current study we hypothesized that there would be no interaction effect between hostility and PTSD in predicting mortality, because the latter represented more of an externalizing trait, rather than an internalizing one as this relates to repression (Brewin, 2005; Holmes, 1990; Horowitz et al, 1990; Wastell, 2002). Contrary to repression, we hypothesized that hostility concurrent with PTSD

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would be protective for adverse health outcomes, as this might provide a release for internalized negative emotional states (Barefoot, 1992).

DATA AND METHODS

Study Population

The current study was based on a random sample of all men who served in the US Army during the Vietnam War and identified through the National Personnel Records Center (St. Louis, MO). Altogether, 18,581 men were randomly selected by a computer program, using data tapes containing virtually all US Army service personnel for this period (Centers for Disease Control, 1989a; 1989b; 1989c). Participants were classified as theater veterans (TVs), if they served in Vietnam or as era veterans (EVs), if they served elsewhere. Starting in January 1985, attempts were made to complete telephone interviews with these men. From these efforts, 87% of TVs (7924) and 84% of the EVs (7364) were interviewed (overall completion rate = 86%). Among these men, a random sample was selected for personal interviews and examinations. Altogether, 75% of the TVs ($N = 2490$) and 63% of the EVs ($N = 1972$) participated in this examination phase. More detailed reports regarding this study have been published and are available elsewhere (Centers for Disease Control, 1989a; 1989b; 1989c; 1989d). The CDC's Human Subject Review Committee approved the study protocols.

Ascertainment of All-Cause Mortality

For the current study, mortality status was assessed from the date of completion of the telephone interviews starting in January 1985 until the end of the mortality follow-up on December 31, 2000. Mortality was ascertained using 3 databases: the Department of Veterans Affairs Beneficiary Identification Record Locator Death File, Social Security Administration Death Master File, and the National Death Index (NDI) file (Boehmer et al., 2004). Veterans not found in these databases were assumed to be living on December 31, 2000. In the current study, the primary outcome of interest included all-cause mortality. During the follow-up period, a total of 250 deaths occurred due to all causes.

Assessment of PTSD

The PTSD measure used in this study was based on the original nomenclature that became part of the DSM-III criteria for PTSD (Boscarino, 2006a). This assessment was administered using telephone surveys just prior to the physical examination (Centers for Disease Control, 1989a). For this scale, veterans were asked to report on the presence of 15 PTSD-related symptoms that could have occurred in the past 6 months (recorded as never, sometimes, often, very often). Consistent with DSM-III criteria, a veteran was classified as having current PTSD if he reported at least one criterion B symptom (re-experiencing), at least 1 criterion C symptom (avoidance), and at least 2 criterion D symptoms (hyper-arousal). This PTSD scale also provided a PTSD symptom severity measure, based on summation of the 15 PTSD symptoms (mean = 9.6, $SD = 8.1$, range: 0–45). For the current study, we classified veterans as having PTSD on this scale, if they met the DSM-III criteria described above. We also used this PTSD scale as a continuous symptom-level measure, as described.

Assessment of Repression, Hostility and Anxiety

To assess psychological repression, we used the Welsh R Scale (WRS). This scale was developed from factor analysis with items from the MMPI (Dahlstrom et al., 1972; Greene, 1991; Welsh, 1965). Although this measure represents an early MMPI scale, it has been use in recent studies and has general face, concurrent, and predictive validity for the presence of repression (Archer et al., 1989; Goldwater and Collis, 1985; Persky et al., 1987). Higher scores on the Welsh R scale are generally associated with increased internalization, denial, and use of repressive coping styles in dealing with emotional stressors (Dahlstrom et al., 1972; Greene, 1991; Welsh, 1965). In our current study, it is noted that the WRS was negatively correlated with antisocial personality disorder ($r = -0.157$, $p < 0.001$), alcohol abuse/dependence ($r = -0.130$, $p < 0.001$), the Welch Anxiety Scale ($r = -0.178$, $p < 0.001$), and the Wiggins Hostility Scale ($r = -0.473$, $p < 0.001$). The case classification for this trait was based on the standard MMPI cut-off for cases (i.e., a T-score of 70 or higher). For symptom-level assessment, we used the raw MMPI score for this measure.

Since recent studies of repression also involve assessment of this trait within the context of anxiety (Frasure-Smith et al., 2002; Weinberger., 1990), we also include assessment of this domain, based on the Welsh A scale (WAS), also developed with the MMPI (Dahlstrom et al., 1972; Greene, 1991; Welsh, 1965). Although this scale is also an early MMPI measure, it has been used in recent studies and appears to have good face, concurrent, and predictive validity for anxiety (Archer et al., 1989; Colligan & Offord, 1988a; Greene, 1991). Higher scores on the WAS are associated with increased trait anxiety and related characteristics (Dahlstrom et al., 1972; 1975; Greene, 1991; Welsh, 1965). In the current study, we used both a case- and a symptom-level measure for this trait, as discussed above for the WRS.

In our study, hostility was assessed by the Wiggins Hostility Scale (WHS), also developed from the MMPI (Dahlstrom et al., 1972; 1975; Greene, 1991; Wiggins et al., 1971). The WHS is reported to measure resentment, feelings of hostility, and poor impression management (Greene, 1991; Wiggins et al., 1971). Assessment of hostility has been common in recent behavioral science research (Boyle et al., 2005; Bunde and Suls, 2006; Friedman, 1992; Iribarren et al., 2005). Since its development, the WHS has been widely used in different studies (Colligan & Offord, 1988b; Dahlstrom et al., 1972; 1975; Greene, 1991) and appears to have strong face, concurrent, and predictive validity for this trait (Colligan & Offord, 1988b; Greene, 1991; Richards, 1986). For measurement of hostility in the current study, we also used a case- and symptom-level measure as discussed above for the WRS and WAS.

Assessment of Confounding Factors

Since this was an observational study, assessment of our hypotheses is contingent upon controlling potential confounders (Boscarino, 2008a; 2008b). In our study this included age, race, and veteran status as demographic variables. Also included were variables related to possible confounding character traits, including intelligence, volunteer status, and history of antisocial personality disorder. We also included history of other mental health disorders that could affect health outcomes, such as alcohol abuse/dependence and major depressive disorder. Confounding health behavior variables were also assessed, including pack-years of smoking and body mass index (BMI). Age was based on the veteran's age at the interview. Race was based on reported race (White, 82%; Black, 11%; Hispanic, 5%; other, 2%) and coded as an indicator variable (white vs. nonwhite). Volunteer status was taken from the military record and coded as a binary variable to indicate if the veteran volunteered for military service or was drafted. Since we combined the TVs and EVs in some multivariate analyses, we also included a binary measure to control for theater status. Intelligence was taken from the military record and based on the General Technical (GT) examination at induction, a measure considered a valid and reliable measure of adult intelligence (Centers for Disease Control, 1989b). Antisocial personality disorder, lifetime depression, and lifetime alcohol abuse/dependence were based on having met the DSM-III

criteria for these disorders and using the Diagnostic Interview Schedule administered during the baseline examination (Centers for Disease Control, 1989b; Robins et al., 1987). BMI was used to classify obesity as present at baseline, based on the subject's weight divided by height squared. Measurement of pack-years of cigarette smoking was based on the average number of reported cigarette packs smoked per day and the number of years smoked at baseline.

Statistical Methods

First, we describe the differences found for PTSD, repression, and mortality by key study variables. Next we use Cox proportional hazard regressions to calculate the adjusted main effects for PTSD, repression, and hostility, respectively, expressed as hazards ratios (HRs) predicting all-cause mortality. In the final step, we add the interaction effects for PTSD × repression, and PTSD × hostility, respectively. Since prior studies suggested that anxiety interacted with repression (Frasure-Smith et al., 2002; Weinberger, 1990), we assessed for this effect as well. For the Cox regressions, we analyzed the results for veterans combined and for TVs and EVs, separately. Since PTSD was assessed in the 1985 survey, we only included those who were alive and completed the 1985 telephone interviews. Thus, our analyses examined survival time from interview completion starting in January 1985 through to December 31, 2000, a period of about 16 years. For these analyses, we evaluated the main proportional hazard assumption of our statistical models (Cleves et al, 2002; Hosmer & Lemeshow, 1999). Statistical analyses were performed using *Stata, version 9.2* (College Station, TX). All *p*-values presented were based on the 2-tail test.

TABLE 1. Profile of Deceased Versus Nondeceased Veterans by Study Variables (N=4462)

Variable	% Total (N)	% Alive	% Deceased	p*
Current PTSD	7.2 (323)	6.6	17.6	<0.001
Welsh repression	4.3 (193)	4.8	4.3	0.704
Wiggins hostility	2.1 (94)	5.2	1.9	<0.001
History of antisocial personality disorder	22.1 (988)	21.5	32.4	<0.001
History of depression	10.5 (468)	10.0	19.2	<0.001
History of alcohol abuse/ dependence	46.7 (2084)	45.9	59.6	<0.001
Age 40+ at interview	19.8 (883)	19.5	25.2	0.027
Non-white race	18.1 (808)	17.3	31.6	<0.001
Intelligence–lowest quintile	18.7 (836)	18.1	29.2	<0.001
Volunteered for military service	36.9 (1645)	36.8	37.6	0.805
Pack-years of cigarette smoking: 19 + years	32.9 (1470)	32.2	45.2	<0.001
Body mass index–highest quintile	16.8 (750)	16.5	21.6	0.037
(N)	(4462)	(4212)	(250)	—

Percents based on column, percents for variable shown. For example, of those who were deceased at follow-up in Table 1, 17.6% had PTSD at baseline, vs. 6.6% who were alive at follow-up.

*2-sided χ^2 *test, df* = 1.

RESULTS

In the current study, 250 men were deceased from all causes at follow-up, including deaths from cardiovascular diseases (n = 65), cancer (n = 52), infectious diseases (n = 19), digestive conditions (n = 19), external causes, including suicides, homicides, and accidents (n = 55), and all other causes (n = 40). At follow-up, as shown in Table 1, mortality was associated with current PTSD, nonhostility, antisocial personality, lifetime depression, history of alcohol abuse/dependence, nonwhite race, lower intelligence, and with higher pack-years of cigarette smoking (all $p < 0.001$). Mortality was also associated with older age ($p = 0.027$) and with a higher BMI at baseline ($p = 0.037$) (Table 1). At baseline, TVs were more likely to have current PTSD, lifetime depression, history of alcohol abuse/dependence, and to be younger (all $p < 0.001$). TVs were also more likely to have volunteered for military service ($p < 0.029$) (table not shown, but available upon request).

The adjusted multivariate Cox regression results for PTSD, repression, and hostility are presented in Table 2. In the PTSD diagnostic model for repression (top panel), it is noted that main effects for repression were not significant, whereas those for PTSD were all significant regardless of veteran cohort status ($p < 0.01$). No interaction effects were detected for repression; that is, none of the interaction effects for PTSD × repression were significant. The Cox regression results for PTSD symptoms and repression were similar, with one exception. For the EVs, having higher repression symptoms at baseline were protective for mortality at follow-up (HR = 0.95, $p = 0.03$). The interaction effects for the PTSD symptom × repression symptom models were not significant for either of the veteran cohorts, however.

Results in panel 2 shows the findings for PTSD and hostility. As can be seen, the main effects for PTSD in the hostility models were all significant ($p < 0.05$), regardless of being measured on a diagnostic or a symptom level. However, for the TVs, a higher hostility symptom score at baseline was associated with higher mortality risk at follow-up (HR = 1.04, $p = 0.034$) and approached significance for a diagnostic case definition for hostility at baseline (HR = 1.93, $p = 0.052$). No interactions were detected for PTSD × hostility regardless of the measurement levels used, however. As suggested, since other research indicated that trait anxiety interacted with repression, we also added the Welsh A (anxiety) scale to our models and assessed both the main and the interaction effects for this measure. Both of these effects were nonsignificant, however (available upon request). For a final step in evaluating our models, we removed the covariate for alcohol abuse/dependence, a behavior often identified as a repressive coping style, but our results were essentially the same. It should also be noted that we examined these results by specific mortality outcomes for cancer, cardiovascular diseases, and for external-cause mortality. However, neither repression nor hostility was significant in any of these models (available upon request).

To assess Cox proportional hazard assumptions, we used Schoenfeld residuals and the "staph test" procedure in Stata to assess model fit. The results indicated that the final models used were adequate, with p generally > 0.05. We also plotted the multivariate survival functional curves for our final models and these indicated adequate model fit as well.

DISCUSSION

Repression has been characterized in the behavioral sciences as the mental process of excluding aversive cognitions from consciousness and as detrimental to future health (Brewin, 2005; Horowitz et al, 1990; Jones, 1993). However, in spite of the use of this concept over the years, this construct has been diffucult to validate (Brewin, 2005; Holmes, 1990). Repression is said to exclude disturbing impulses and their proximate derivatives by means of keeping them unconscious (Jones, 1993). It has been assumed that repression of these stimuli comes at a psychological price, since traumatic emotional states were not cognitively integrated, resulting

TABLE 2. Cox Regressions: Adjusted Hazard Ratio Models Predicting All-Cause Mortality for Veterans by Repression, Hostility, and PTSD at Baseline

	All Veteran Person Years at Risk ~ 71,382; Theater Veterans Person Years at Risk ~ 39,840; Era Veterans Person Years at Risk ~ 31,552								
	All Veterans 250 Deaths (N = 4462)			Theater Veterans 154 Deaths (N = 2490)			Era Veterans 96 Deaths (N = 1972)		
Repression Models*	**HR**	**95% CI**	***p***	**HR**	**95% CI**	***p***	**HR**	**95% CI**	***p***
Main effects for diagnosis									
Repression diagnosis	1.28	0.72–2.29	0.404	1.85	0.97–3.53	0.061	0.53	0.13–2.16	0.376
PTSD diagnosis	1.93	1.36–2.75	<0.001	1.71	1.14–2.55	0.010	2.93	1.44–5.98	0.003
Interaction effect added to main effect									
Repression × PTSD diagnosis	1.99	0.51–7.73	0.320	2.11	0.51–8.77	0.303	†	†	†
Main effects for symptoms									
Repression symptoms	1.00	0.97–1.03	0.816	1.03	0.99–1.06	0.157	0.95	0.91–1.00	0.030
PTSD symptoms	1.05	1.03–1.06	<0.001	1.04	1.02–1.06	<0.001	1.05	1.02–1.08	<0.001
Interaction effect added to main effect									
Repression × PTSD symptoms	1.00	1.00–1.01	0.118	1.00	1.00–1.00	0.715	1.00	1.00–1.01	0.263
	All Veterans			**Theater Veterans**			**Era Veterans**		
Hostility Models*	**HR**	**95% CI**	***p***	**HR**	**95% CI**	***p***	**HR**	**95% CI**	***p***
Main effects for diagnosis									
Hostility diagnosis	1.72	0.97–3.06	0.064	1.93	1.00–3.75	0.052	1.08	0.33–3.55	0.899
PTSD diagnosis	1.87	1.31–2.67	0.001	1.64	1.09–2.47	0.017	2.93	1.42–6.05	0.004
Interaction effect added to main effect									
Hostility × PTSD diagnosis	0.66	0.20–2.13	0.488	0.68	0.18–2.59	0.573	0.50	0.04–6.06	0.582
Main effects for symptoms									
Hostility symptoms	1.03	1.00–1.06	0.030	1.04	1.00–1.08	0.034	1.02	0.97–1.06	0.450
PTSD symptoms	1.04	1.02–1.06	<0.001	1.03	1.01–1.06	0.001	1.05	1.02–1.08	0.001
Interaction effect added to main effect									
Hostility × PTSD symptoms	1.00	0.99–1.00	0.241	1.00	0.99–1.00	0.103	1.00	1.00–1.00	0.659

*Results shown are for the adjusted main effects for repression and hostility, respectively, followed by the adjusted interaction effects for each main effect. All model results shown include age, race, intelligence, pack-years of cigarette smoking, history of alcohol abuse, and history of depression. The "all-veterans" models also controlled for theater status by inclusion of a binary variable for theater status.

†Not possible to calculated these results due to empty cells for this model.

HR indicates hazard ratio; CI, confidence interval; PTSD, posttraumatic stress disorder.

in negative health impacts over time (Horowitz et al, 1990; Brewin, 2005; Pennebaker and Seagal, 1999).

Our study suggests that psychological repression, as measured on the Welsh R scale, had no negative impact on mortality. We assessed the hypothesis that PTSD at baseline concurrent with a repressive personality trait would be associated with an increase in adverse health outcomes. As we have seen, this was not the case. In addition, for the EVs the main effect for repression appeared protective. The main effect for repression, although not significant, was in the opposite direction for TVs, suggesting an increased mortality risk for this trait for them (HR = 1.85, p = 0.061). Furthermore, at least for the TVs, the main effect for hostility symptoms was associated with poorer health outcomes (p = 0.034). However, no interaction effects were detected for either repression or hostility traits in our study. We also assessed for an interaction effect for repression × anxiety, since this had been previously suggested (Frasure-Smith et al, 2002; Weinberger, 1990), but this was not significant in our study. Given these findings, we accept the null hypothesis that there was no association between a repressive personality trait and PTSD (or hostility) in our study.

As also noted, no disease-specific results were found either. Given these findings and recent reports that repressive coping styles might be protective for PTSD symptoms (Solomon et al, 2007; Ginzburg et al, 2002; Coifman et al, 2007), we also assessed this association in the current study. Our findings were consistent with these recent reports: repression symptoms were negatively associated with PTSD symptoms ($r = -0.11$, $p < 0.001$). In addition, we found that hostility symptoms were positively associated with PTSD symptoms ($r = 0.21$, $p < 0.001$). However, unlike the results reported above, these respective correlations were similar, regardless of theater or era veteran status.

This study has strengths and limitations. Use of multiple sources of mortality allowed for a more complete post-service assessment and is a study strength. However, although our PTSD measure appeared to have concurrent validity, this scale was based on early version of this nomenclature (Centers for Disease Control, 1989b; Kulka et al, 1990). Another limitation stems from the measure of repression we used—the Welch R scale. This MMPI-derived measure is described as capturing character traits related to repression and denial across multiple domains, including health and physical symptoms, emotionality, and personal interests (Green, 1991). However, this is an older MMPI scale and it may have lacked specificity. In this regard, it has been noted that the repression literature has suffered from a lack of conceptual clarity (Horowitz

et al, 1990; Singer and Sincoff, 1990). Although the WRS has been widely used in research (Archer et al, 1989; Dahlstrom et al, 1972; 1975; Goldwater & Collis, 1985; Greene, 1991; Persky et al, 1987; Welsh, 1965), it is possible that this measure may have misclassified this conceptual domain (Colligan & Offord, 1988a). Other scales measuring this may have resulted in different findings.

An additional limitation was that our study included only men. Furthermore, we only examined mortality outcomes at follow-up, not illness onset or physical symptoms, so our health outcome measure is limited. Also, the results for EVs differed somewhat from TVs, perhaps reflecting different types of trauma exposures between these cohorts. In particular, while our PTSD measure was associated with all-cause mortality among those in both groups, higher repression symptoms were protective for EVs. Although not quite significant ($p = 0.061$), the opposite was found for TVs. In addition, TVs with higher hostility symptoms were at greater risk of death ($p = 0.034$). For specific mortality related to cancer, external mortality or cardiovascular diseases, no differences were apparent, however.

Our study suggested that a repressive personality trait did not adversely impact long-term health outcomes among those with PTSD. Recent research suggesting that repression was protective for PTSD is noteworthy and warrants further investigation (Ginzburg et al, 2002; Solomon et al, 2007). This finding has also been confirmed in the current study, as noted. Human behavior is complex and for some persons, contrary to current clinical thinking, it is possible that repression of traumatic memories may be beneficial to long-term health (Coifman et al, 2007). Nevertheless, consistent with the original concept of repression, it has been suggested that the psychopathology involved with PTSD is likely related to physiologic processes to some degree inaccessible through normal cognitive processes (Izard, 1993; Depue et al, 2007). As has been previously suggested (Boscarino, 2008a; 1996), understanding the neurophysiological aspects of traumatic stress exposure seems to be warranted to describe the sequelae associated with PTSD onset and course. Future research is strongly urged to further investigate these findings.

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