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# Trauma Exposure and Health: The Role of Depressive and Hyperarousal Symptoms

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Posttraumatic stress disorder (PTSD) and depressive symptoms have been theorized to mediate the relationship between trauma exposure and physical health symptoms. Although empirical evidence supports this premise, studies conducted to date have employed statistical mediation analyses that are now broadly criticized. Furthermore, the mediating roles of both PTSD and depressive symptoms have seldom been examined concurrently, and it remains unclear which PTSD symptom clusters uniquely mediate this relationship. The aim of the present study was to examine the mediating role of reexperiencing, avoidance/numbing, hyperarousal, and depressive symptoms in the relationship between trauma exposure and physical health symptoms. Participants were 516 Spanish female undergraduate students. Physical health symptoms were compared between those who reported trauma exposure ($n = 266$) and those who did not ($n = 250$). Data from trauma-exposed participants were analyzed using regression models with bootstrapping to test mediation. Results of the analyses showed that the trauma-exposed group reported significantly more physical health symptoms ($r^2 = .035$). Hyperarousal and depressive symptoms uniquely mediated the relationship between trauma exposure and physical health symptoms. Our findings clarify some of the mechanisms by which negative health consequences occur subsequent to trauma exposure.

Numerous studies provide evidence for a positive relationship between trauma exposure and physical health symptoms (Ruiz-Pérez, Plazaola-Castaño, & del Río-Lozano, 2007; Sachs-Ericsson, Cromer, Hernandez, & Kendall-Tackett, 2009). Schnurr and Green (2004) proposed an integrative model in which posttraumatic stress disorder (PTSD) may be the primary pathway by which trauma leads to negative health outcomes. They note depressive symptoms may also be an important pathway to poor physical health subsequent to trauma exposure. Several studies have tested the postulates of this model.

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The role of PTSD symptoms as a mediator of the relationship between trauma and health symptoms has been examined, with findings supporting both full (Campbell, Greeson, Bybee, & Raja, 2008; Kimerling, Clum, & Wolfe, 2000; Taft, Vogt, & Mechanic, 2007) and partial (Del Gaizo, Elhai, & Weaver, 2011; Eadie, Runtz, & Spencer-Rodgers, 2008; Spitzer, Barnow, Völzke, Freyberger, & Grabe, 2009) mediation. Results, however, remain equivocal, with some studies failing to replicate evidence of a mediating role for PTSD symptoms (Norman et al., 2006; Sledjeski, Speisman, & Dierker, 2008).

Although previous research suggests PTSD symptom clusters (i.e., reexperiencing, avoidance/numbing, hyperarousal) *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev; *DSM-IV-TR;* American Psychiatric Association, 2000) are differentially related to physical health symptoms, it remains unclear which symptoms uniquely predict health symptoms. It has been hypothesized that the chronic physiological stress that accompanies PTSD—particularly hyperarousal symptoms—is responsible for increased disease risk (Boals, Riggs, & Kraha, 2012; Litz, Keane, Fisher, Marx, & Monaco, 1992). Results of studies examining the relationships between the specific PTSD symptom clusters and physical health symptoms, however, have been mixed. Some researchers have found that only hyperarousal accounted for unique variance in physical health symptoms (Clum, Nishith, & Resick, 2001; Kimerling et al.,

2000), even when controlling for depressive symptoms (Clum et al., 2001). Others have found avoidance/numbing (Polusny et al., 2008; Woods, Hall, Campbell, & Angott, 2008) and re-experiencing (Zoellner, Goodwin, & Foa, 2000) to uniquely predict physical health symptoms. Collectively, these results suggest relationships between specific PTSD symptoms and physical health symptoms remain poorly understood.

Two recent studies that have concurrently examined depression and PTSD as mediators of the relationship between trauma and physical health symptoms (Baker, Norris, Jones, & Murphy, 2009; Eadie et al., 2008) have reported contradictory results. Whereas Baker at al. (2009) found that depression mediated the relationship between childhood sexual violence and physical health symptoms, the results found by Eadie et al. (2008) did not support the mediating role of depression in the relationship between sexual assault experiences and adverse physical health outcomes. Moreover, mediation was tested using Baron and Kenny's (1986) procedure, a method now critiqued as problematic (Preacher & Hayes, 2008; Zhao, Lynch, & Chen, 2010). According to Kraemer (2008), the use of classic methods to assess mediation based on statistical significance in linear models often produces inconsistent results that are difficult to interpret in terms of clinical significance. Instead, nonparametric techniques that assess population side effects rather than statistical significance in a sample are recommended. Furthermore, Zhao et al. (2010) maintain that, contrary to Baron and Kenny's suggestions, the test of the relationship between the predictor and the outcome is never relevant to establishing mediation, as it is possible to establish an indirect effect despite no total effect. Therefore, researchers should not give up on a mediation hypothesis when they fail to find an effect to be mediated—in so doing they may be erroneously abandoning significant research projects and impeding theoretical development. There is a need for research in this area that applies more recent statistical procedures for evaluating mediation (i.e., bootstrapping methods).

Other methodological issues may also account for the mixed results of studies assessing the mediational role of PTSD and depressive symptoms. First, most studies have employed medical or other treatment-seeking samples, which, due to range restriction in physical health symptoms, may inflate the strength of observed relationships (Tabachnick & Fiddell, 2007). Second, with some exceptions (e.g., Sledjeski et al., 2008), most studies have been conducted with victims of a single traumatic exposure type (e.g., military combat, sexual assault), rather than considering the full spectrum of traumatic exposure over the lifespan. This issue not only limits the generalizability of results but may impact the reliability of trauma exposure assessment (Finkelhor, Ormrod, & Turner, 2007). Finally, the suggestion that traumatic exposure has a cumulative impact on health (Sledjeski et al., 2008) needs to be considered.

Given the equivocal nature of the extant literature, this study sought to clarify the nature of the relationships between traumatic exposure and self-reported health symptoms in a sample of undergraduate women who reported heterogeneous trauma. We examined differences in self-reported physical health symptoms between participants who reported trauma exposure (as defined in the *DSM-IV-TR*) and those who did not. We also concurrently examined the roles of both *DSM-IV-TR* PTSD symptom clusters and depressive symptoms as potential mediators of the relationship between lifetime traumatic exposure (i.e., number of reported lifetime traumatic events) and physical health symptoms. We hypothesized that only PTSD hyperarousal (Clum et al., 2001; Kimerling et al., 2000) and depressive symptoms (Baker et al., 2009; Schnurr & Green, 2004) would uniquely mediate the relationship between trauma exposure and physical health symptoms. We caution that due to the cross-sectional nature of our data, our analyses can offer only provisional findings regarding mediation. To conclusively evaluate mediation, the use of longitudinal research methodologies is required (Kraemer, 2008).

## Method

### Participants and Procedure

Participants were 516 female undergraduate students whose average age was 20.51 years ($SD = 3.66$) from the University of Málaga (Spain) who had participated in a larger study. Participants were recruited from undergraduate classes in October 2008 and May 2009. They received no compensation for participating. The full sample was divided into (a) individuals reporting no trauma exposure ($n = 250$) whose average age was 19.86 ($SD = 2.69$), and (b) individuals reporting trauma exposure ($n = 266$) whose average age was 20.15 ($SD = 3.20$). See Table 1 for traumatic event types. Participants completed a battery of questionnaires prior to or after scheduled

Table 1
*Frequency of Traumatic Events Reported by Those Reporting Exposure*

| Traumatic event | Frequency | % |
|---|---|---|
| Life-threatening illness | 25 | 9.4 |
| Life-threatening accident | 57 | 21.4 |
| Robbery with physical force or use of weapon | 32 | 12.0 |
| Death of close person due to accident, homicide, suicide | 64 | 24.0 |
| Sexual abuse | 52 | 19.6 |
| Physical abuse | 65 | 24.5 |
| Emotional abuse | 89 | 33.5 |
| Being threatened with a weapon | 18 | 6.8 |
| Witness to violence | 47 | 17.7 |
| Exposure to other life-threatening situations | 2 | 0.8 |
| Miscarriage | 16 | 6.0 |
| Other traumatic situations | 45 | 16.5 |

*Note*. $n = 265$. Total exceeds 265 as participants could endorse more than one event.

undergraduate psychology classes. Participants were informed that the research focused on aspects of stress and health. Informed consent was obtained from all participants, and the study was approved by the University of Malaga's Research Ethics Board.

## Measures

The Stressful Life Events Screening Questionnaire-Revised (SLESQ-R; Green, Chung, Daroowalla, Kaltman, & DeBedenedictis, 2006) was designed to identify *DSM-IV-TR* PTSD Criterion A traumatic events in nontreatment-seeking individuals, and to minimize reporting of subthreshold events (Goodman, Corcoran, Turner, Yuan, & Green, 1998). The SLESQ-R has good test-retest reliability and convergent validity, and has been found to discriminate between Criterion A and non-Criterion A events (Goodman et al., 1998). As this questionnaire does not assesses PTSD Criterion A2 (i.e., subjective reaction to a traumatic event), participants were also asked to rate the level of fear and helplessness experienced on an 11-point scale (i.e., 0 = *Not at all intense* to 10 = *Extreme intensity*).

The Davidson Trauma Scale (DTS; Davidson, 1996) was developed to diagnose PTSD and to assess symptom severity and treatment outcome. The DTA was adapted for use among Spanish speakers by Bobes and colleagues (2000). Its 17 items assess both frequency and severity of PTSD symptoms as described in *DSM-IV* (American Psychiatric Association, 1994) and can be used to compute PTSD symptom cluster scores (i.e., reexperiencing: range = 0–20, avoidance/numbing: range = 0–44, and hyperarousal: range = 0–20). The DTS is a reliable and valid measure of PTSD symptoms and has demonstrated diagnostic accuracy comparable to the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1990). Alpha coefficients were .92, .93, and .95 for the reexperiencing, avoidance/numbing, and hyperarousal subscales, respectively. The DTS was administered immediately after the SLESQ-R (participants completed the DTS only if they endorsed at least one SLESQ-R traumatic event). If participants endorsed multiple SLEQ-R events they were instructed to respond to DTS items with reference to the most distressing event experienced.

The Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977) is a 20-item self-report measure designed to assess depressive symptoms experienced during the past week. Items are endorsed on a 4-point scale ranging from 0 = *Never or few* to 3 = *Usually*. The CES-D scores range from 0 to 60. The original version of the CES-D exhibited good psychometric properties (Roberts & Vernon, 1983). The Spanish version has good psychometric properties and has been validated for use in nonclinical populations (Vázquez, Blanco, & López, 2007). The α coefficient for the CES-D was .91.

The Patient Health Questionnaire-15 (PHQ-15; Kroenke, Spitzer, & Williams, 2002) was used to assess physical health symptoms. Items inquire about 15 somatic symptoms accounting for more than 90% of the symptoms reported in outpatient settings. Respondents rate the severity of each symptom on a 3-point scale (i.e., 0 = *Not bothered at all*, 1 = *Bothered a little*, 2 = *Bothered a lot*). The PHQ-15 enables classification of participants into four categories according to the reported severity of their symptoms: *minimal* (scores = 0–4), *low* (scores = 5–9), *medium* (scores = 10–14), and *high* (scores = 15–30). The PHQ-15 has excellent internal reliability and adequate convergent validity. It has been strongly associated with functional status, disability days, and symptom-related difficulties. Moreover, it has been demonstrated to have discriminant validity with respect to depressive symptoms (Kroenke et al., 2002). For the current sample the α coefficient was .73.

## Data Analysis

Criteria for trauma-exposure were endorsement of at least one item on the SLESQ-R (Green et al., 2006; *DSM-IV-TR* Criterion A1) together with reported high levels of fear, helplessness, or horror (i.e., scores $\geq$ 8/10 on the numeric rating scale; Vázquez, Pérez-Sales, & Matt, 2006; *DSM-IV-TR* Criterion A2). We conducted a *t* test to assess whether participants who reported exposure to one or more traumatic events ($n$ = 266) would also report more physical health symptoms than participants reporting no trauma exposure ($n$ = 250). The role of *DSM-IV-TR* PTSD symptoms clusters (i.e., reexperiencing, avoidance/numbing, hyperarousal) and depressive symptoms in the relationship between trauma exposure and physical health symptoms was assessed in a series of regression models which used bootstrapping to test mediation. These analyses were conducted with only the sample of individuals reporting exposure to at least one traumatic event. Specifically, we employed an SPSS macro developed for testing mediation in multiple mediator models (Preacher & Hayes, 2008), which generated 5,000 bootstrap samples to calculate 95% bias-corrected confidence intervals (see Figure 1 for a representation of the tested model). Figure 1A represents the total effect of trauma exposure on physical health symptoms (Path C). Figure 1B represents both the direct effect of trauma exposure on physical health symptoms (Path C') and the indirect effects of trauma exposure on physical health symptoms via hyperarousal, reexperiencing, avoidance/numbing, and depressive symptoms. The specific indirect effect of trauma exposure on physical health symptoms via a particular mediator is defined as the product of the two unstandardized paths (ab) linking trauma exposure and physical health via that mediator. For example, the specific indirect effect of trauma exposure on physical health symptoms via hyperarousal is quantified as a1b1. The total indirect effect of trauma exposure on physical health symptoms is the sum of the specific indirect effect of each mediator, and the total effect of trauma exposure on physical health symptoms is the sum of the direct effect and the total indirect effect. To bootstrap the sampling distribution of the specific and total indirect effects, a sample of size $n$ cases is taken from the original sample. Using this new resample size ($n$) a and b values for each mediator are estimated, and ab (the IE) for each mediator, as well as the total IE, are calculated. This process is repeated $k$ times (5,000 in our

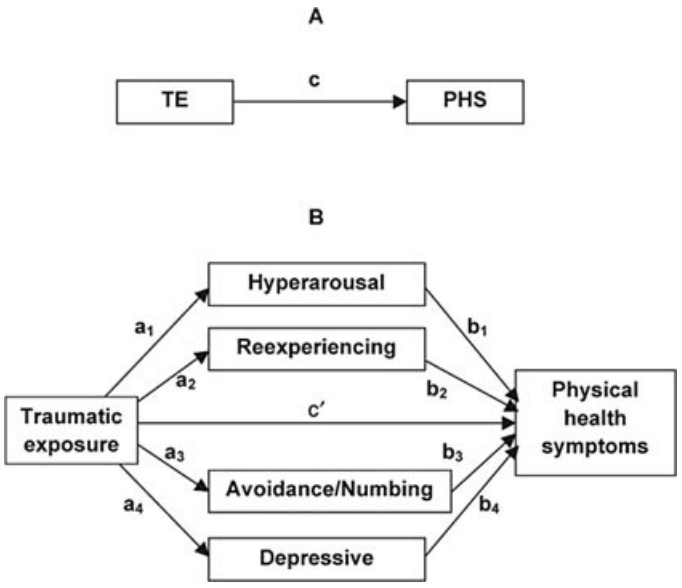


*Figure 1.* Graphic representation of the multiple mediation model with four mediators (i.e., hyperarousal, reexperiencing, avoidance/numbing, and depressive symptoms. A: Traumatic exposure affects physical health symptoms. B: Trauma exposure is hypothesized to exert effects on physical health symptoms through the four mediators.

analyses) yielding 5,000 estimates of the total and specific indirect effects of trauma exposure on physical health symptoms. The distributions of these 5,000 estimates serve as empirical, nonparametric approximations of the sampling distributions of the indirect effects of interest. The bootstrap confidence interval (CI) for the population-specific indirect effect through each mediator is derived by sorting the 5,000 values of each indirect effect from low to high. Values defining the lower and upper 100% (α/2) of the distribution of each indirect effect are then found and taken as the lower and upper limits of the 100% CI $(1 - \alpha)$ for the population indirect effect, where α is the desired nominal Type I error rate. When CIs of the indirect effect of a mediator do not include 0, this effect is considered statistically significant (for a more thorough explanation of these methods, see Preacher & Hayes, 2008).

Prior to analyses, data were assessed for normality by inspection of histograms and probability-probability plots as well as statistical indicators in SPSS. All indicators suggested data were within recommended limits. We also assessed for and found no violation of assumptions concerning multicollinearity among depression and PTSD predictor variables.

## Results

As expected, participants who reported at least one traumatic event also reported significantly more physical health symptoms than participants reporting no trauma exposure, $t(513) = 4.30$, $p < .001$, two-tailed, $r^2 = .035$. Mean physical health symptoms for those reporting at least one traumatic event was 10.34 ($SD = 4.46$). Mean physical health symptoms for those reporting no traumatic events was 8.73 ($SD = 4.01$).

Among the trauma-exposed participants, 80 reported DTS (Davidson et al., 1997) scores ≥ 40, suggesting probable PTSD (Davidson et al., 1997); 70 trauma-exposed participants reported CES-D scores ≥ 26 (Radloff, 1977), suggesting probable major depression (Vázquez et al., 2007). Descriptive statistics and zero-order correlations among variables included in the mediation models are presented in Table 2, respectively. Trauma exposure was significantly and positively correlated with the three PTSD symptom clusters (i.e., reexperiencing, avoidance/numbing, hyperarousal) as well as with depressive symptoms and physical health symptoms. All PTSD symptom clusters were positively and significantly correlated with depressive and physical health symptoms. Depressive symptoms were positively correlated with physical health symptoms.

Results of mediational analyses indicated the total indirect effect of trauma exposure on physical health symptoms through the three PTSD symptom clusters and depressive symptoms was statistically significant (Table 3). The direct effect of trauma exposure on physical health symptoms was not significant ($\beta = .23$, $SE = .19$, $t = 1.18$, $p > .05$). These results are consistent

Table 2
*Descriptive Statistics and Correlations for Trauma, PTSD and Depressive Symptoms, and Physical Symptoms*

| Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|---|---|
| 1. Trauma frequency[a] | 1.92 | 1.32 | – | | | | |
| 2. DTS-RXP | 10.41 | 8.83 | .31*** | – | | | |
| 3. DTS-AVN | 10.45 | 11.28 | .36*** | .75*** | – | | |
| 4. DTS-HA | 9.61 | 10.12 | .33*** | .75*** | .79*** | – | |
| 5. CES-D | 19.21 | 10.84 | .14* | .24*** | .37*** | .39*** | – |
| 6. PHQ-15 | 10.34 | 4.46 | .18** | .25*** | .30*** | .36*** | .48*** |

*Note.* $n = 266$. PTSD = Posttraumatic stress disorder; DTS = Davidson Trauma Scale; RXP = reexperiencing; AVN = avoidance/numbing; HA = hyperarousal; PHQ-15 = Patient Health Questionnaire-15; CES-D = Center for Epidemiologic Studies-Depression Scale.

[a]Frequency refers to traumatic events that met *DSM-IV-TR* PTSD A2 criteria.

*$p < .05$. **$p < .01$. ***$p < .001$, two-tailed.

Table 3
*Indirect Effect of Trauma Exposure on Self-Reported Physical Health Symptoms Through PTSD and Depressive Symptoms (Bootstrapping)*

| Variable | β | *SE* | 95% CI[a] |
|---|---|---|---|
| 1. Depressive symptoms | .19 | .09 | [.04, .39] |
| 2. Reexperiencing | .00 | .09 | [-.18, .20] |
| 3. Avoidance/numbing | -.07 | .13 | [-.33, .21] |
| 4. Hyperarousal | .26 | .13 | [.07, .61] |
| Total | .39 | .13 | [.14, .65] |
| Indirect effect differences | | | |
| 1. vs. 2. | .19 | .12 | [-.01, .44] |
| 1. vs. 3. | .26 | .17 | [-.07, .62] |
| 1. vs. 4. | -.06 | .15 | [-.37, .22] |
| 2. vs. 3. | .07 | .19 | [-.33, .45] |
| 2. vs. 4. | -.26 | .17 | [-.70, .05] |
| 3. vs. 4. | -.33 | .23 | [-.87, .08] |

*Note*. $n = 265$; 5,000 bootstrap samples. PTSD = posttraumatic stress disorder.
[a]Bias corrected and accelerated confidence interval (CI).

with indirect-only mediation (Zhao et al., 2010) of the effect of trauma exposure through PTSD and depressive symptoms on physical health symptoms. Closer examination showed the specific indirect effects of trauma exposure on physical health symptoms through hyperarousal and depressive symptoms were statistically significant, whereas the indirect effects of trauma exposure through reexperiencing and avoidance/numbing were not (Table 3).

Examination of indirect effect pair-wise contrasts (i.e., C1, C2, C3, C4, C5, and C6 reported in Table 3) revealed that although the specific indirect effect of trauma exposure on self-reported physical health symptoms through hyperarousal was higher than the specific indirect effect through depressive symptoms, the magnitude of this difference (C3) was not statistically significant. Despite finding that the indirect effects of trauma exposure on physical health symptoms for both hyperarousal and depressive symptoms were statistically significant, the magnitudes of these indirect effects did not statistically differ from those found for reexperiencing and avoidance/numbing.

## Discussion

The purpose of this study was to clarify the nature of the relationship between trauma exposure and physical health symptoms by concurrently examining the mediational roles of PTSD and depressive symptoms in this relationship. As predicted, participants who experienced at least one traumatic event reported significantly more physical health symptoms than those who did not report traumatic exposure. Also consistent with expectations, the relationship between trauma exposure and physical health symptoms was completely and uniquely mediated by hyperarousal and depressive symptoms.

These results are in line with previous findings regarding the relationship between trauma exposure and physical health (Ruiz-Pérez et al., 2007; Sachs-Ericsson et al., 2009). Reported exposure to at least one traumatic event accounted for 3.5% of the variance in physical health symptoms. Following PHQ-15 classification based on the symptom severity, the nontrauma-exposed individuals fell into the low severity category, whereas trauma-exposed individuals fell into the medium severity category, suggesting that these differences in physical health symptoms were not only statistically significant, but also clinically significant. Moreover, trauma exposure (i.e., number of lifetime traumatic events) was positively correlated with PTSD, depressive, and physical health symptoms, supporting the suggestion traumatic exposure may have a cumulative impact on health (Sledjeski et al., 2008).

Our results are also consistent with findings supporting the unique role of hyperarousal in relation to physical health symptoms when controlling both for depressive (Clum et al., 2001) as well as reexperiencing and avoidance/numbing (Clum et al., 2001; Kimerling et al., 2000) symptoms. Chronic autonomic arousal, common among persons with PTSD (Stam, 2007), may lead to the development of organic and functional physical health problems via different mechanisms. First, hyperarousal may be responsible for neuroimmunoendocrine system alterations recently found in persons diagnosed with PTSD. Posited to arise, in part, via epigenetic processes, these alterations are reported to include changes in cortisol, insulin, and glucose levels; elevations in cytokines and enzymes associated with inflammation; and altered genetic signatures reflecting compromised immune function (Bauer, Wieck, Lopes, Teixeira, & Grassi-Oliveira, 2011; Gill, Saligan, Woods, & Page, 2009; Uddin et al., 2010). In fact, hyperarousal symptoms have been shown to correlate with thyroid hormone levels (i.e., T3) in veterans with PTSD (Wang et al., 1995). Although these findings are intriguing, whether hyperarousal symptoms are specifically related to biological alterations awaits empirical investigation. It is also possible that hyperarousal impairs health via risk behaviours (e.g., substance use; Schnurr & Green, 2004) or psychological mechanisms (e.g., hypervigilance for bodily sensations; Asmundson & Katz, 2009).

Our findings support the model proposed by Schnurr and Green (2004) in that symptoms of both PTSD and depression were found to mediate the relationship between trauma and physical health symptoms. Consistent with Baker and colleagues (2009), depressive symptoms accounted for unique variance in physical health symptoms beyond that accounted for by all PTSD symptom clusters. This finding suggests that individuals who experience depressive symptoms subsequent to trauma, but do not present prominent PTSD symptoms, are also at risk for poor physical health. One mechanism potentially linking depressive symptoms with poor physical health may be glucocorticoid dysregulation (Rohleder, Wolf, & Wolf, 2010). Depressive symptoms could also influence physical health via other factors such as physical inactivity, smoking, or sleep disturbances (Goral, Lipsitz, Muhsen, & Gross, 2011).

Recent findings collectively indicate that the mechanisms by which PTSD influences health may differ from those associated with depression. Evidence points to decreased glucocorticoid sensitivity of inflammatory systems in depression versus increased glucocorticoid sensitivity in PTSD (Rohleder et al., 2010). It may be that differing biological alterations develop in persons for whom depression arises subsequent to traumatic exposure. Alternatively, given the high comorbidity of depressive and PTSD symptoms (Kessler, Sonnega, Bromet, Hughes, & Nelson, 2004), it is possible these symptoms interact in ways that lead to different patient subtypes. Further research employing clinical samples should be directed toward the biological and psychological mechanisms by which depressive symptoms lead to poor physical health.

Neither reexperiencing or avoidance/numbing symptoms were found to uniquely mediate the trauma exposure-physical health symptoms relationship. Nonetheless, the magnitudes of their effects on physical health symptoms were comparable to those of hyperarousal and depression. This may suggest that reexperiencing and avoidance/numbing symptoms also mediate the relationship between trauma exposure and physical health symptoms, but not uniquely.

Our findings shed light on some of the mediational mechanisms by which negative health occurs subsequent to trauma, while improving some methodological issues. Although Schnurr and Green (2004) posited that both PTSD and depressive symptoms were potential mediators of the trauma-health relationship, few studies have attempted to evaluate both variables concurrently; moreover, to our knowledge depressive symptoms and the three PTSD symptom clusters have previously not been assessed together as mediators of physical health symptoms. Unlike previous studies, we examined relationships between trauma and physical health symptoms in a sample of relatively healthy young trauma-exposed females in whom the relationships found are less likely to be inflated by inclusion of treatment-seeking individuals. Our use of a nonclinical sample permitted us to conclude that relationships among trauma exposure, PTSD, and depressive symptoms influence health in normative samples. In light of our findings, monitoring and perhaps treating subclinical symptoms of PTSD and major depressive disorder might be considered to prevent the development of physical health symptoms. In addition, our study considered the full spectrum of traumatic event exposure over the lifespan, which improves the reliability of trauma assessment and the generalizability of our results (Finkelhor et al., 2007). Finally, by employing currently recommended best statistical procedures, we addressed some of the methodological limitations of previous studies.

This study had several limitations that suggest future research directions. First, self-report scales were employed to assess all variables. Future studies would be strengthened by clinician assessment of trauma exposure, PTSD, and depression. Self-reported physical health is important insofar as it reflects subjective perceptions of health and well-being. Although perceived health can motivate individuals to seek medical attention and physicians importantly consider symptom reports in making patient care decisions (Sareen, Stein, Campbell, Hassard, & Menec, 2005), research methodologies would be strengthened by the inclusion of objective health measures. Second, we did not distinguish among the different types of health complaints (e.g., gastrointestinal, cardiovascular). Some authors have suggested the influence of PTSD and depression on physical health may vary depending on the type of health problems examined (Feztner, McMillan, & Asmundson, 2012). Studies in which this distinction is considered are needed. Third, it is possible that some of the traumatic situations reported did not meet *DSM-IV-TR* traumatic event criteria, as the SLEQ contains an item allowing individuals to indicate whether they have experienced a traumatic situation not described in the scale. Nonetheless, recent findings suggest that situations perceived as traumatic, but not meeting *DSM-IV-TR* A1 criteria have an equivalent impact on health and can even be more pervasive, especially when characterized by high levels of betrayal (Brown & Freyd, 2008). Fourth, we employed a cross-sectional design; therefore, our findings should be considered preliminary, as proper mediation can only be tested with longitudinal designs. Fifth, we instructed participants to respond to the DTS items in reference to their most distressing traumatic event, but we did not directly ask for the event participants were referencing when completing this questionnaire. Furthermore, information regarding some of the sociodemographic characteristics of the sample (e.g., socioeconomic or marital status, race) was not gathered. These data should be collected in future investigations. Finally, because only women participated in the study, the results need to be replicated with samples of men.

In conclusion, our findings suggest that even in normative samples trauma-exposed individuals appear to be at increased risk for physical health problems. Early treatment of hyperarousal and depressive symptoms after trauma exposure may mitigate the development of physical health problems. We suggest that primary care patients who report idiopathic physical health symptoms be assessed for PTSD and depressive symptomatology, as identifying and treating these symptoms (particularly hyperarousal) may reduce physical symptoms. Reductions in PTSD and depressive symptoms via psychological intervention accounted for 29% of the variance in reduction of reported health problems and 30% of the variance in improvement of general social functioning (Rauch et al., 2009); thus, it seems probable that treatments that target hyperarousal and depressive symptoms may improve outcomes.

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