Published in final edited form as:

Behav Med. 2008 ; 33(4): 125–135. doi:10.3200/BMED.33.4.125-136.

Direct and Indirect Links Between Childhood Maltreatment, Posttraumatic Stress Disorder, and Women's Health

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Abstract

The authors evaluated the relationships among childhood maltreatment, sexual trauma in adulthood, posttraumatic stress disorder (PTSD), and health functioning in women. Female Veterans' Affairs (VA) primary care patients (N = 200) completed self-report measures of childhood maltreatment, adult sexual trauma, PTSD symptoms, and current health functioning. The authors used structural equation modeling to test models of the relationship among these variables. Childhood nonsexual maltreatment and adult sexual assault were positively associated with PTSD. Childhood nonsexual maltreatment ($\beta = -.20$) and PTSD ($\beta = -.75$) were significantly associated with poorer physical and mental health functioning. Adult sexual assault negatively affected health functioning through its association with PTSD. Thus, poor health outcomes associated with childhood maltreatment in women may be conveyed through PTSD. These findings should strengthen efforts directed at identifying and treating PTSD in female victims of childhood maltreatment with the aim of preventing or attenuating poor health outcomes.

Index Terms

childhood maltreatment; health outcomes; posttraumatic stress disorder; sexual trauma

Researchers have found strong associations between adverse childhood experiences (including but not limited to sexual abuse)^{1,2} and poor physical and mental health functioning.³⁻¹⁰ This observation is particularly pertinent to understanding the health of women, a group that is at high risk for exposure to childhood maltreatment, in addition to subsequent exposure to sexual assault and intimate partner violence in adulthood.¹¹⁻¹⁴ Although criminal victimization of women has been identified as a frequent antecedent to poor health outcomes, the pathways through which these traumatic events are translated into such outcomes are unclear.¹⁵ Improved understanding of potential mediators of these outcomes could lead to better-targeted efforts at secondary prevention.

One likely mediator of the relationship between traumatic experiences in women and poor health-related functioning is posttraumatic stress disorder (PTSD).¹⁵ PTSD is a common mental disorder characterized by a set of maladaptive responses to serious, life-threatening

trauma.¹⁶ PTSD is more common in women than men, and this difference is partly—but not completely—explained by the higher rates of certain forms of maltreatment (eg, sexual abuse, emotional abuse) experienced by women.^{11,17,18} PTSD has been shown repeatedly to have a detrimental effect on health and well-being in a wide variety of contexts, including in general public communities,^{19,20} patients seeking mental health treatment,²¹ military veterans and active-duty personnel (including peacekeepers),²²⁻²⁹ and victims of trauma seen in primary care and specialty medical and surgical settings.³⁰⁻³⁵

Findings from several studies support the hypothesis that PTSD mediates the relationship between traumatic experiences and poor health outcomes. Taft et al³⁶ conducted a path analysis, using National Vietnam Veterans Readjustment Survey data, to test the relationship between mental and physical aspects of health to combat exposure in 1,632 male and female Vietnam War veterans and found that PTSD was a critical mediator of the relationship between combat exposure and physical and functional health status. In a separate study of 2,301 male and female Gulf War veterans, PTSD symptoms upon return from war were associated with self-reported health problems.³⁷ In another study, presence and severity of PTSD in 276 combat veterans was associated with greater physical health problems and conditions.²² In a study of 52 female Vietnam War-era veterans, PTSD symptoms accounted significantly for the variance associated with self-reported health problems.²⁵ In a large survey of 30,685 female veteran respondents, PTSD was associated with an elevated burden of medical illness, over and above that associated with comorbid major depression.²⁹

These data provide a rationale for expecting that PTSD will at least partially mediate the relationship between childhood maltreatment and poor health-related functioning in women. To our knowledge, this pathway has not been investigated but may have important implications for intervention. In the present study, we tested this hypothesis by examining, in a primary care sample of female military veterans, the associations among self-reported childhood maltreatment. We selected female military veterans because this group is known to have higher rates of sexual trauma than do civilian women.^{38,39} We used separate models to test paths from childhood sexual abuse and from other forms of childhood maltreatment, which are highly comorbid,^{1,2} to adult sexual assault, PTSD, and physical and mental health functioning.

METHODS

Participants

We mailed a consent form and a packet of questionnaires to all women seen in the Veterans' Affairs San Diego Healthcare System (VASDHS) primary care clinic in 1998. We used the questionnaires to gather information about demographics, exposure to traumatic stressors, and psychological and medical symptoms. We sent a second questionnaire packet to recipients who did not respond within 2 weeks; we sent one additional reminder 3 weeks later to those who still did not respond. Of the total 419 questionnaires that were mailed, 21 were sent back because of incorrect addresses and 4 were sent back because the veteran was deceased. The revised eligible sample of female veterans was therefore reduced to 394. Of these, 29 women indicated that they did not wish to participate in the study, and 221 completed the questionnaire, yielding an adjusted response rate of 56.1%. All participants provided informed, written consent to participate in the study, which the University of California San Diego (UCSD) Human Research Protection Program approved.

Using the clinic's computer system, we compared demographic characteristics of survey responders and nonresponders. Women who returned the questionnaire did not differ significantly in age ($M = 46.1$, $SD = 14.9$) from those who did not return the questionnaire ($N = 173$; $M = 46.2$, $SD = 14.2$). Women who returned the questionnaire were significantly

more likely to be never married than were women who did not return the questionnaire ($p < .01$). The groups were otherwise comparable in terms of ethnicity and marital status.

Researchers focusing on somatic symptoms and health anxiety⁴⁰ and health behaviors^{41,42} have described this demographic. The mean age of the women was 46.6 years ($SD = 14.6$; range 20–86). Most women were Caucasian (71%), followed by African American (15%) and Hispanic (6%) participants. Most women were divorced/separated/widowed (41%); the others were married/living with a partner (38%) or never married (21%). Most women (75%) had 12–15 years of education, with a minority (25%) having 16 or more years.

Measures

We assessed childhood maltreatment using the Childhood Trauma Questionnaire (CTQ).⁴³ The CTQ is a 28-item self-report retrospective instrument that assesses 5 areas of childhood maltreatment: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. It includes statements about childhood experiences followed by response options on a 5-point Likert scale (1 = *never true*, 5 = *very often true*), according to the frequency with which experiences occurred. The CTQ subscales, as well as the entire scale, have high test–retest reliability ($r = .80-.88$) and high levels of internal consistency (Cronbach's $\alpha = .79-.94$). Findings from validation studies of the CTQ support its convergent and discriminant validity with structured trauma interviews and corroboration with independent data, and researchers⁴⁴ have used the CTQ in studies of general healthcare use and costs in primary care settings. Wright et al⁴⁵ recently confirmed the factor structure of the CTQ, and normative community data with the CTQ are available.⁴⁶ The internal consistency of the CTQ was high ($\alpha = .91$) in our sample. Subscale scores on Emotional Abuse, Physical Abuse, Emotional Neglect, and Physical Neglect served as indicators of childhood maltreatment (nonsexual), and answers to each of the 5 questions that comprised the sexual abuse subscale served as indicators of childhood sexual abuse.

We assessed adult sexual assault using self-report questions from the list of stressful experiences from the Clinician-Administered PTSD Scale (CAPS) from the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (*DSM-IV*).⁴⁷ We considered exposure to adult sexual assault (questioned separately for military and extra-military time periods) present if the respondent said she experienced “rape, attempted rape, or made to perform any type of sexual act through force or threat of harm.”

We assessed PTSD symptoms using the Posttraumatic Stress Disorder Checklist-Civilian Version (PCL-C).⁴⁸ The PCL-C includes the 17 PTSD symptoms that correspond to the *DSM-IV*. Respondents use a Likert scale to rate the degree to which they have been bothered by each symptom during the past month on a scale from 1 (*not at all*) to 5 (*extremely*). The 17 PCL-C items can be scored to yield 4 subscales that correspond with dimensions of PTSD consistently extracted from factor analyses: reexperiencing symptoms, hyperarousal, emotional numbing, and avoidance.^{49,50} The scores of these 4 dimensions served as indicators of PTSD symptoms. The PCL-C has been extensively used in health outcomes research studies over the past decade.⁵¹ The internal consistency of the PCL-C was high in our sample ($\alpha = .95$).

We assessed physical and mental health functioning using the Medical Outcome Study Short Form (SF-36), a self-administered scale that is a reliable and valid measure of physical and mental health-related functioning.⁵² We used the mental health composite score (MCS) and a physical health composite score (PCS), which are obtained from the SF-36, as indicators of physical and mental health functioning. Scores on the composite scales range from 0 to 100, with lower scores indicating more impaired functioning.

Statistical Analysis

We computed scale scores by summing all items in a given scale and then computing the scale mean. We then used the mean scores as indicators for latent variables. We used structural equation modeling (SEM)⁵³ to test our hypotheses. We first tested a latent variable measurement model, then tested the full theoretical model, a mediation-only model, a direct-effects only model, and, finally, a trimmed model. We conducted model testing with the Mplus analytic program⁵⁴ and maximum likelihood estimation with robust standard errors and mean-adjusted chi-square statistic (ie, MLM). We used the MLM estimator to adjust for nonnormal distributional characteristics (skewness) found for some variables.^{55,56} The use of weighted least-squares estimation with robust standard errors and adjusted means and variances (WLSMV) is recommended for confirmatory factor analytic models with binary or ordered categorical (eg, Likert-scaled) data with nonnormality.⁵⁷ In the current set of analyses, we used no individual categorical items, but, rather, mean scale scores of multiple Likert-scaled items. However, to be conservative, we also tested all models using the WLSMV approach. The results were essentially equivalent for the MLM and WLSMV approaches, and we report the MLM analyses in this article.

We evaluated model fit by examining the fit indicators for maximum likelihood estimation, using criteria suggested by Hu and Bentler.⁵⁸ These include examination of the chi-square statistic, a comparative fit index (CFI) value near .95 or greater, a Tucker-Lewis (TLI) index value close to .95 or greater, a root mean square error of approximation (RMSEA) value close to .08 or less, and a standardized root mean residual (SRMR) value close to .06 or less. As recommended,⁵⁴ we examined the weighted root mean square residual (WRMR) for use with MLM estimation⁵⁹ where values close to 1.0 or lower indicated acceptable fit. We also report the χ^2/df ratio in which values greater than 3 indicated poor model fit⁶⁰ and chi-square difference tests using the Satorra and Bentler⁶¹ scaled correction factor. The chi-square difference tests allow for testing of relative fit for nested models.

We report standardized factor loadings and path coefficients so that each can be easily interpreted and the relative effect size of path coefficients can be interpreted and contrasted. Sample size for SEM analyses was slightly reduced ($n = 200$) because of missing data for some participants.

RESULTS

Characterization of Maltreatment Exposure and PTSD Symptoms

Total scores on the CTQ ranged from 25 to 114, with an average of 50.29 ($SD = 21.97$). Mean subscale scores on the CTQ were as follows: Sexual Abuse 9.49 ($SD = 6.58$), Physical Abuse 9.55 ($SD = 5.39$), Emotional Abuse 11.74 ($SD = 6.28$), and Physical Neglect 7.54 ($SD = 3.45$). These scores are characteristic of a sample with substantially more childhood maltreatment than community norms.^{18,46}

Ninety-seven participants (44.3%) reported experiencing sexual assault in adulthood. This rate of exposure to sexual assault, although higher than in other reported primary care samples,^{30,62} is compatible with rates of sexual trauma reported in other studies of female military veterans.³⁹

For the PCL-C, total scores ranged from 17 to 85, the range of the measure, with an average of 39.12 ($SD = 18.22$). Given that scores of 30 or above have been recommended as a threshold when screening for PTSD among women in primary care settings,⁶³ this mean score would indicate that this sample carried a heavy burden of PTSD symptoms.

Model Fitting

We examined a measurement model and primary hypothesized full model, and alternative models tested whether various direct and mediated effect models would better fit the data. To facilitate model comparison, Table 1 shows the fit indexes for all models.

Model 1 assessed the measurement model, which showed good fit between the hypothesized model and the data, $\chi^2(109, N = 200) = 215.08, p < .001$, CFI = .96, TLI = .95, RMSEA = .07, SRMR = .04, indicating that items loaded on the appropriate latent variables. All factor loadings were significant ($p < .001$) and ranged from .66 to .95. As shown in Figure 1, Model 2 assessed the full-hypothesized structural model, allowing exogenous independent variables to covary. We also specified structural paths between the dependent variable (health functioning) and the mediating variable (PTSD), and the dependent and exogenous independent latent variables (childhood maltreatment [nonsexual], childhood sexual abuse, adult sexual assault). The full structural model also showed good fit, $\chi^2(109, N = 200) = 215.08, p < .001$, CFI = .96, TLI = .95, RMSEA = .07, SRMR = .04; however, the path from child sexual abuse to PTSD and the path from adult sexual assault to health functioning were nonsignificant.

To evaluate whether a mediational model was supported, we estimated 2 additional models, Model 3 (with mediational paths from exogenous variables to PTSD but no direct paths to health functioning) and Model 4 (without these mediational paths but with direct paths from exogenous variables to health functioning). Both models also had a direct path from PTSD to health functioning. As shown in Figure 2, Model 3 demonstrated somewhat poorer fit than did Models 1 or 2, $\chi^2(112, N = 200) = 222.19, p < .001$, $\chi^2/df = 1.98$, CFI = .955, TLI = .945, RMSEA = .07, SRMR = .046, WRMR = .821. However, the chi-square difference tests were marginal in suggesting that the model differed significantly from the full structural model. These results suggest that although PTSD mediates effects of trauma on health functioning, there are likely to be significant direct effects. Model 4 (see Figure 3) fit poorly relative to the full structural model, $\chi^2(112, N = 200) = 273.38, p < .001$, $\chi^2/df = 2.44$, CFI = .933, TLI = .919, RMSEA = .085, SRMR = .181, WRMR = 3.27, and the chi-square difference test indicated that Model 4 differed significantly from the full model, $\Delta\chi^2(3) = 46.08, p < .001$. This indicates strong support for the mediational role of PTSD in health functioning.

To determine an optimal and more parsimonious model, we re-estimated a trimmed model with nonsignificant paths from Model 1 removed. As shown in Figure 4, this trimmed model demonstrated good fit, $\chi^2(112, N = 200) = 218.68, p < .001$, CFI = .96, TLI = .95, RMSEA = .07, SRMR = .05. The trimmed model was not significantly different from the full structural model, $\Delta\chi^2(3) = 3.72, p > .05$, and we accepted it as our final model on the basis of its more parsimonious structure.

In summary, we found that PTSD mediates the effects of trauma on health functioning and, as shown in Figure 4, that both childhood maltreatment (nonsexual) and adult sexual assault were positively associated with PTSD, with higher levels of childhood maltreatment and adult sexual abuse associated with greater levels of PTSD. Childhood maltreatment (nonsexual), childhood sexual abuse, and adult sexual assault were all significantly positively correlated with one another. Last, higher levels of childhood maltreatment (nonsexual) directly affected health functioning, and PTSD predicted poorer physical and mental health functioning.

COMMENT

Researchers have demonstrated and successfully replicated strong, negative relationships between childhood maltreatment and adverse health consequences in women. Likewise, relationships between PTSD and poor health-related quality of life in female veterans have been amply documented. These studies, in concert, leave little doubt that childhood

maltreatment and PTSD are associated with substantial functional adversity in women. Given these observations, an obvious question is to what extent PTSD mediates the relationship between childhood maltreatment and poor health functioning.

We found that the strongest path to physical and mental health functioning was a direct (negative) path from PTSD, indicating that more PTSD symptoms are associated with poorer health functioning. This path represented a large effect size with an additional, small effect size for the direct path from childhood nonsexual trauma to health functioning. PTSD symptoms were associated with childhood maltreatment and adult sexual assault by approximately equal moderate effect sizes. A number of researchers have shown that PTSD at least partially mediates the effects of trauma exposure (eg, combat) in adulthood on health outcomes. In the context of these earlier observations, our finding that PTSD largely mediated the effects of childhood maltreatment and fully mediated the effect of adult sexual assault on health functioning completes the picture of PTSD as a critical mediator of health outcomes to a wide spectrum of traumatic antecedents, both in childhood and adulthood. From a public health perspective, given its placement in the pathway from trauma to adverse health outcomes, a focus on aggressively identifying and treating PTSD may have substantial salutary effects on women's health functioning and use of health services.^{23,64} Researchers should test this hypothesis in randomized clinical trials in which they target women with PTSD in general medical settings. Given the high rates of trauma and PTSD among female veterans^{22,35,39} the VA system may be an ideal system of care in which to conduct such a study.

Our study has a number of limitations that must be considered when interpreting our results. First, this is a cross-sectional study, and although the SEM approach compares alternative models to enable inferences about causal pathways, definitive statements about causality can be made only with prospective, longitudinal data. Second, the model tested was not inclusive of all potentially relevant kinds of trauma (eg, domestic violence)⁶⁵ because our focus was on childhood maltreatment and sexual trauma; more inclusive models may lead to different results. Third, PTSD symptoms were not explicitly linked to a particular "worst" trauma, as is customarily done in epidemiologic surveys, and we did not include PTSD in the model as a diagnosis but, rather, as a continuous measure of posttraumatic stress-related symptoms. There is, however, considerable justification for this approach. Taxometric analyses of PTSD symptoms have yielded a dimensional solution, suggesting that PTSD reflects the upper end of a stress-response continuum rather than a discrete clinical syndrome.⁶⁶ Fourth, we did not include other potentially relevant symptom domains that may be part of the pathway from PTSD to poor health outcomes (eg, depression, substance abuse),⁶⁷⁻⁷⁰ which may have yielded different results had they been included. Fifth, current health state may bias retrospective reporting of trauma,⁷¹ although there is considerable evidence to suggest that individuals are highly reliable in their recollection of traumatic experiences.⁷² Sixth, although our response rate (56%) is respectable by survey standards—and similar to response rates were obtained in similar VA outpatient settings (eg, 65% in a recent survey³⁵)—a higher response rate would have provided greater reassurance about the generalizability of our findings. Last, self-reported physical and mental health functioning, as characterized by the SF-36, may provide different results than do more specific disease-related outcomes (eg, major depression, heart disease).^{8,73} Cloitre et al⁷⁴ found that exposure to trauma, but not PTSD, was related to the number of medical problems endorsed, whereas PTSD, but not trauma exposure, was associated with perceived health. However, the SF-36 is a robust, widely used summary measure of health-related functioning that transcends specific diagnoses.

In conclusion, much of the variance in poor health outcomes associated with childhood maltreatment and adult sexual assault in women may be conveyed through PTSD. Investigators must replicate these observations in prospective, longitudinal studies before definitive conclusions about causality can be made. Nonetheless, even at this juncture, these findings

should strengthen efforts directed at identifying and treating PTSD in female victims of childhood maltreatment with the aim of preventing or attenuating poor health outcomes and high healthcare costs.^{64,75} It is possible, for example, that better educating physicians about PTSD and its effects may lead to improved care and improved outcomes. Our findings also point to the need for future researchers to determine the mechanisms by which PTSD mediates the health impact of trauma and develop methods to enhance resilience to these injurious effects.

Acknowledgements

Dr Stein received a VA Merit Review Grant and NIMH Grant MH64122 and Dr Aarons received NIMH Grant MH01695 to support this study. The authors thank Rebecca Lenox for her assistance with data collection.

The authors dedicate this work to the memory of James Gearity, who always maintained a positive outlook in difficult circumstances.

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FIGURE 1.

Model 2: Full hypothesized structural model of the links between childhood maltreatment, adult sexual trauma and posttraumatic stress disorder and their relationship to health functioning in women in primary health care $\chi^2(109, N = 200) = 215.08, p < .001$, comparative fit index (CFI) = .96, Tucker-Lewis Index (TLI) = .95, root mean square error of approximation (RMSEA) = .07, standardized root mean residual (SRMR) = .04. All factor loadings and correlations were significant at $p < .001$; for path coefficients: ns = nonsignificant. $^{**}p < .01$. $^{***}p < .001$.

FIGURE 2.

Model 3: Mediation only model, $\chi^2(112, N = 200) = 222.19, p < .001, \chi^2/df = 1.98$, comparative fit index (CFI) = .955, Tucker-Lewis Index (TLI) = .945, root mean square error of approximation (RMSEA) = .07, standardized root mean residual (SRMR) = .046, weighted root mean square residual (WRMR) = .821. All factor loadings and correlations were significant at $p < .001$; for path coefficients: ns = nonsignificant. ** $p < .01$. *** $p < .001$.

FIGURE 3.

Model 4: No mediation model, $\chi^2(112, N = 200) = 273.38, p < .001, \chi^2/df = 2.44$, comparative fit index (CFI) = .933, Tucker-Lewis Index (TLI) = .919, root mean square error of approximation (RMSEA) = .085, standardized root mean residual (SRMR) = .181, weighted root mean square residual (WRMR) = 3.27. All factor loadings and correlations were significant at $p < .001$; for path coefficients: ns = nonsignificant. $^{**}p < .01$. $^{***}p < .001$.

FIGURE 4.

Model 5: Parsimony trimmed model, $\chi^2(112, N = 200) = 218.68, p < .001$, comparative fit index (CFI) = .96, Tucker-Lewis Index (TLI) = .95, root mean square error of approximation (RMSEA) = .07, standardized root mean residual (SRMR) = .05. All factor loadings and correlations were significant at $p < .001$; for path coefficients: ns = nonsignificant. ** $p < .01$. *** $p < .001$.