PTSD as a Mediator of Sexual Revictimization: The Role of Reexperiencing, Avoidance, and Arousal Symptoms

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Theory and research suggest that posttraumatic stress disorder (PTSD) may mediate the relationship between child sexual abuse and adult sexual assault. However, little empirical research has examined the mediational role of PTSD. In the present study, the authors use structural equation modeling to examine the degree to which the three symptom clusters that define PTSD (reexperiencing, avoidance, and hyperarousal) contribute to sexual revictimization. To assess PTSD symptomatology, undergraduate women completed questionnaires (N = 1,449), which detailed the history and severity of childhood and adult sexual assault experiences. Results indicated that PTSD mediated sexual revictimization. When PTSD symptom clusters were examined individually, only the hyperarousal cluster was a significant mediator. Results are discussed in terms of information-processing mechanisms that may underlie sexual revictimization.

Women who experience childhood sexual abuse (CSA), compared to those who do not, are at least twice as likely to experience adult sexual victimization (e.g., Gidycz, Coble, Latham, & Layman, 1993; Mayall & Gold, 1995; Risser, Rabenhorst, & McCanne, 2002; Russell, 1986; Wyatt, Guthrie, & Notgrass, 1992). Moreover, among women who experienced CSA, the likelihood of adult sexual assault (ASA) is related to the severity of CSA experiences. Specifically, more intrusive types of CSA (e.g., noncontact vs. contact; Arata, 2000; Fergusson, Horwood, & Lynskey, 1997; Haskell, 2000; Koverola, Proulx, Battle, & Hanna, 1996), multiple CSA experiences (Haskell, 2000; Koverola et al., 1996), longer duration of CSA (Arata, 2000), and CSA involving physical force (Arata, 2000; West, Williams, & Siegel, 2000) are associated with a heightened risk of sexual revictimization.

Several explanations have been proposed for the heightened risk of ASA among women who experienced CSA, and especially women who experienced severe forms of CSA (for a review, see Messman-Moore & Long, 2003). One possible mediator of the CSA–ASA relationship that has garnered increased attention is the symptoms associated with posttraumatic stress disorder (PTSD; Acierio, Resnick, Kilpatrick, Saunders, & Best, 1999; Arata, 2000;
Bolstad & Zinbarg, 1997; Cloitre, Scarvalone, & Difede, 1997). According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revised (DSM-IV-TR; American Psychiatric Association, 2000), PTSD is a disorder that results from exposure to a traumatic event and that is marked by three symptom clusters: Reexperiencing aspects of the trauma (reexperiencing), avoidance of reminders of the trauma (avoidance), and persistent symptoms of hyperarousal (hyperarousal).

The primary purpose of the present study is to evaluate the role of PTSD as a potential mediator of the association between CSA and ASA. To capture the broad range of experiences that may constitute CSA and ASA, we assessed each of these constructs in terms of several indicators of severity, rather than using dichotomous measures of sexual victimization. In addition, we examined the possibility that the PTSD symptom clusters are differentially important in mediating sexual revictimization, by examining each symptom cluster as a potential mediating factor.

Theorists have suggested several ways in which PTSD might contribute to an increased likelihood of ASA. Posttraumatic stress disorder symptoms may impair a woman's ability to accurately detect danger cues (Arata, 2000; Becker-Lausen, Sanders, & Chinsky, 1995; Chu, 1992; Elliot, 1997; Koss, Figueredo, Bell, Tharan, & Tromp, 1996; McFarlane, 1988; McNally, 1998; Orcutt, Erikson, & Wolfe, 2002; Palmer, 2002), impair responsiveness to trauma-related cues (Elliot, 1997), or inhibit self-protective behaviors (Chu, 1992; Becker-Lausen et al., 1995; Ginsberg, Wright, Harrell, & Hill, 1989; van der Kolk, 1996). Theoretically, these outcomes could occur because PTSD symptoms may cause difficulties in concentrating (McFarlane, 1988), attending to new information (Orcutt et al., 2002), remembering trauma-related cues relevant to risk detection (Koss et al., 1996), and differentiating relevant from irrelevant information (Palmer, 2002). Posttraumatic stress disorder symptoms may also increase the risk of ASA by leading to increases in high-risk behaviors such as substance use (Kilpatrick et al., 2000) and increased sexual activity (Arata, 2000).

Theorists have not commonly framed their discussions of the mechanisms through which PTSD may mediate sexual revictimization effects in terms of specific PTSD symptom clusters. Nonetheless, different accounts of how PTSD might increase the risk of ASA implicate specific symptom clusters to varying degrees. For instance, it has been suggested that the hyperarousal component of PTSD may increase a woman's risk of ASA victimization by impairing her ability to recognize dangerous situations (Becker-Lausen et al., 1995). If a woman experiences high levels of hyperarousal on a consistent basis, even in the absence of external causes, she may lose her ability to discriminate between real threats and false alarms, and she may therefore begin to disregard legitimate warning cues. In turn, this could increase her risk of future sexual victimization. However, one study showed that, among multiply victimized women, greater risk was perceived by those who were high, rather than low, in hyperarousal symptoms (Wilson, Calhoun, & Bernat, 1999).

Another formulation implicates the avoidance symptom cluster of PTSD as a mediator of sexual revictimization. If a woman avoids thoughts, feelings, and behaviors associated with a previous traumatic experience, she may fail to learn from the experience. As a result, she may fail to recognize similar threats present in subsequently encountered situations (Arata, 2000). In addition, Chu (1992) suggested that the numbing component of the avoidance symptom cluster is likely to mediate revictimization by reducing the victim's capacity to engage in self-protective behaviors. In support of the avoidance hypothesis, research has shown that dissociation—another component of the avoidance cluster—mediates the relationship between general childhood maltreatment and subsequent victimization (Cloitre et al., 1997; Kluft, 1990).

According to another formulation, the PTSD symptoms of reexperiencing and hyperarousal, coupled with avoidance and numbing, constitute cognitively demanding tasks that increase cognitive load, thereby reducing the ability to efficiently and thoroughly process new information (Orcutt et al., 2002). This line of thinking suggests that PTSD symptoms resulting from CSA may impair attention to one's environment, making the victim less effective at noticing, recognizing, and processing danger cues present in his or her surroundings, and thereby
resulting in increased risk of ASA victimization. Although any of the symptom clusters might deplete cognitive resources, Orcutt et al. (2002) proposed that the reexperiencing symptom cluster is most responsible for interfering with self-protective behaviors.

Despite frequent speculation regarding a mediating role for PTSD in the CSA–ASA relationship, evidence supporting this hypothesis is primarily indirect. Considerable research documents associations between CSA, ASA, and PTSD, lending credence to the mediational hypothesis. We have already reviewed evidence that CSA is associated with heightened risk of ASA, and that more severe CSA increases the risk for ASA. It has also been well established that CSA can constitute a traumatic stressor that produces PTSD (e.g., see Beitchman et al., 1992; Boney-McCoy & Finkelhor, 1996; Rodriguez, Ryan, Kemp, & Foy, 1997). Moreover, just as more severe CSA is associated with increased risk of revictimization, more severe forms of CSA are also more likely to produce PTSD (e.g., Arata, 2000; Browne & Finkelhor, 1986; Fromuth, 1986; O’Neill & Gupta, 1991; Russell, 1986; Saunders, Villeponteaux, Lipovsky, Kilpatrick, & Veronen, 1992; Williams, 1993). More severe CSA experiences predict higher levels of PTSD symptoms as well (e.g., Williams, 1993).

There is also considerable evidence that PTSD and ASA are associated, although PTSD has typically been regarded as a consequence (rather than a cause) of ASA, and PTSD has generally been assessed only after ASA occurred (Arata, 1999; Foa, Riggs, & Gershuny, 1995; see Messman & Long, 1996; Resnick, Kilpatrick, Darnsky, Saunders, & Best, 1993; Sandberg, Matorin, & Lynn, 1999). However, at least two studies have provided evidence that PTSD may precede ASA (although neither examined the relationship between PTSD symptoms and the severity of subsequent ASA). First, in a 2-year, three-wave longitudinal study of a national probability sample of American women, Acierino and colleagues (1999) found that PTSD predicted ASA victimization during the subsequent 2 years, albeit only when previous victimization was not in the model. Second, in a prospective study of college women, Messman-Moore, Brown, & Koelsch (2005) found that posttraumatic symptoms assessed at Time 1 were marginally higher among previous abuse victims who were sexually revictimized within the following 8 months than among previous abuse victims who were not.

Although evidence of bivariate associations between CSA, PTSD, and ASA is abundant, we could locate only three studies that directly tested the hypothesis that PTSD mediates sexual revictimization. Arata (2000) tested a path model in which the association between CSA severity and sexual revictimization was mediated by PTSD, self-blame, and consensual sexual behavior. In her sample of 221 college women who had experienced CSA, the model provided a good fit to the data, and all mediators were statistically significant. Similarly, in an 8-month prospective study of 339 college women, Messman-Moore and colleagues (2005) reported that PTSD and self-dysfunction mediated the relationship between CSA and college rape. However, in a 10-week prospective study examining whether PTSD and dissociation mediated sexual revictimization effects among 323 college women, Sandberg et al. (1999) did not find support for the mediational model. Although previous victimization was significantly associated with both PTSD and ASA, PTSD did not significantly predict ASA.

One problem with these studies as tests of the hypothesis that PTSD mediates sexual revictimization effects lies in the manner in which PTSD was assessed. The Arata (2000) and Sandberg et al. (1999) studies both used the Revised Impact of Events Scale (IES; Horowitz, Wilner, & Alvarez, 1979), and the Messman-Moore et al. (2005) study used several subscales from the Trauma Symptom Inventory (TSI; Briere, 1995). Both measures assess only two of the symptom clusters that currently define PTSD (intrusion and avoidance); neither assesses the third (hyperarousal) cluster. As a result, the measures of PTSD used in these studies may constitute incomplete measures of PTSD symptomatology, and these studies consequently may provide an incomplete test of the role of PTSD in sexual revictimization. As we discussed previously, the assessment of all three symptom clusters that comprise PTSD is particularly important because different proposals about the manner in which PTSD influences revictimization have different implications regarding the specific cluster or clusters that may be most responsible for sexual victimization.
In the present study, we used structural equation modeling (SEM) to evaluate the role of PTSD in mediating the association between CSA and ASA. In addition to employing a measure of PTSD that assesses all three symptom clusters, we examined the independent and joint contributions of each cluster to the mediation of sexual revictimization effects. Explicating the role of each PTSD symptom cluster in sexual revictimization effects may help to illuminate possible mechanisms through which PTSD mediates revictimization. This model was tested in a sample of female college students. Although it is also important to study sexual revictimization among men, the paucity of previous research on these effects among men make it difficult to build complex models of male sexual revictimization (e.g., Roodman & Clum, 2001).

**Method**

**Participants**

Undergraduate women enrolled in an introductory psychology course at a large Midwestern university were recruited to participate in a survey of child and adult histories. Participants received course credit for their participation. The study sample consisted of 1,449 women (\(N = 1,464\)) who provided complete data on the measures described below. Most participants (71%) were first-year students, with a mean age of 19.0 years (\(SD = 3.10\)). The majority was White (61%); the remainder was African American (24%), Latina (7%), Asian American (6%), or of other descent (2%).

**Measures**

**Childhood sexual experiences questionnaire.** Childhood sexual abuse was measured using a self-report adaptation of Finkelhor’s Survey of Childhood Sexual Experiences (CSEQ; 1979). Participants were asked to indicate how frequently they had experienced each of 15 sexual acts before the age of 15 with someone at least 5 years older. Acts ranged from an invitation for sexual contact to oral, anal, and vaginal intercourse. Participants who indicated childhood sexual experiences were asked to provide details about the experience, including age at onset, frequency, duration (months), and level of force.

Participants’ CSEQ responses were used to derive the four indicators of CSA severity: frequency, duration, type of abuse, and level of force used. Frequency was rated on a 5-point scale (0 = never; 4 = very often). Duration was coded as the number of months (or portions of a month) for which abuse occurred. An index of the most extreme type of abuse experienced was created based on the most intrusive type of abuse reported (0 = no childhood sexual assault experience; 8 = completed oral, anal, or vaginal intercourse). Level of force was rated on a 6-point scale (0 = none; 5 = threat with/ use of a weapon).

**Adult sexual experiences questionnaire.** Adult sexual assault was measured using a self-report adaptation of Finkelhor Survey of Childhood Sexual Experiences (Finkelhor, 1979). The Adult Sexual Experiences Questionnaire (ASEQ) was identical to the CSEQ except in two respects. First, the participants were asked to report on forced or nonconsensual sexual experiences that happened after (rather than before) their 15th birthday. Second, for each of the 15 questions on sexual experiences, two additional questions were included. These questions assessed whether the experience felt forced and whether the experience was nonconsensual (yes or no). An adult sexual experience was considered to be ASA only if these two questions were answered in the affirmative. Just as for CSA, frequency, duration, type of assault, and level of force served as indicators of ASA severity.

**Posttraumatic stress disorder questionnaire.** A self-report version of the PTSD Interview (PTSD-Q; Watson, Juba, Manifold, Kucala, & Anderson, 1991) was used to assess PTSD symptoms as defined in the *DSM-IV-TR*. Participants were first asked to describe the most horrible or frightening event they had ever experienced. They were then asked to rate how often they had experienced each of the 17 symptoms listed in criteria B, C, and D for the *DSM-IV-TR* PTSD diagnosis since the occurrence of the trauma, and to report the month and year of symptom
onset. Ratings of each symptom were made on a 7-point scale (1 = no/never; 7 = extremely/always). The reexperiencing scale (criteria B) included five items assessing recurrent and intrusive recollections of the event. The avoidance scale (criteria C) included seven items assessing efforts to avoid situations related to the trauma. The hyperarousal scale (criteria D) included five items assessing symptoms such as irritability and difficulty sleeping or concentrating. Scores on each scale were computed by summing the relevant items.

Cross and McCanne (2001) validated the PTSD-Q using the Structured Clinical Interview for the DSM-IV (First, Spitzer, Gibbon, & Williams, 1995) with a sample of 76 college women. Using a cutoff score of 60, the PTSD-Q correctly classified 82% of the participants with high levels of specificity (.82) and sensitivity (.81). For the current sample, the internal consistencies of the reexperiencing, avoidance, and hyperarousal scales were .88, .85, and .84, respectively. Intercorrelations among the PTSD-Q subscales in this sample ranged from .68 to .75.

Procedure

The measures used in the current study were embedded in a larger battery of questionnaires. Surveys were administered to groups of no more than 25 participants by female experimenters. Participants were informed that all information provided would be confidential.

RESULTS

In the study sample, 14% (n = 205) of women reported some type of CSA. Of these, 80% reported at least contact sexual abuse and 22% reported completed rape in childhood. The average frequency of CSA was 1.9 (2 = occasionally; SD = 1.00). Most CSA victims reported that CSA occurred rarely (41%) or occasionally (31%; often = 17%; very often = 11%). Twenty-five percent of respondents reported that physical force was used during CSA. In terms of duration, 42% of those reporting CSA characterized the experience as lasting for days, 25% characterized it as lasting for months, and 33% characterized it as lasting for years.

The average score on the PTSD-Q was 32.0 (SD = 17.6). Using Cross and McCanne’s (2001) suggested cutoff score of 60, 10% of participants were classified as exhibiting PTSD. The average scores for the three PTSD symptom clusters were 10.5 (SD = 6.3) for PTSD reexperiencing, 12.8 (SD = 7.6) for PTSD avoidance, and 8.8 (SD = 5.5) for PTSD hyperarousal.

Adult sexual abuse was reported by 31% of the sample (n = 454). Of these, 83% reported at least contact sexual assault and 34% reported completed adult rape. The average frequency of ASA was 2.00 (2 = occasionally; SD = 0.90). Most ASA victims reported that ASA occurred rarely (43%) or occasionally (34%; often = 18%; very often = 5%). Thirty-three percent of respondents reported that physical force was used during the ASA. In terms of duration, 45% of those with ASA characterized the experience as lasting for days, 26% characterized it as lasting for months, and 29% characterized it as lasting for years. Only 2.1% of the sample reported continuous abuse from before age 15 to after age 15.

Structural Equation Modeling

We tested the proposed model using structural equation modeling (SEM), conducted using LISREL 8.3 (Jöreskog & Sörbom, 2001). Correlations between the indicators of CSA severity, ASA severity, and the three PTSD symptom clusters are provided in Table 1. To set the scale of the latent variables, for each latent variable the path of one indicator was set to 1. The chi-square goodness of fit test is highly influenced by sample size; χ² is likely to be significant when N is large, suggesting a lack of fit between the data and the hypothesized model, even when the model provides a good fit (Kline, 1998). Therefore, we examined several additional fit indices (root mean square error of approximation [RMSEA], non-normed fit index [NNFI], and the goodness of fit index [GFI]) to determine model superiority. Although there are no set criteria for evaluating these indices, conventional guidelines suggest that the
Table 1. Correlation Matrix, Means, and Standard Deviations of Observed Indicators

<table>
<thead>
<tr>
<th></th>
<th>CSA Severity</th>
<th>Reexperiencing</th>
<th>Avoidance</th>
<th>Hyperarousal</th>
<th>ASA Severity</th>
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<tr>
<td></td>
<td>Frequency</td>
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<td>Type</td>
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<tr>
<td>Force</td>
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<td>.54</td>
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<tr>
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<tr>
<td>Even</td>
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<td>.13</td>
<td>.13</td>
<td>.84</td>
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<tr>
<td>Avoidance</td>
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<tr>
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<tr>
<td>Even</td>
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<td>.08</td>
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<td>.10</td>
<td>.63</td>
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<td>ASA severity</td>
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<td>0.09</td>
<td>0.72</td>
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<td>0.3</td>
<td>1.96</td>
<td>0.84</td>
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</tr>
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</table>

Note. CSA = childhood sexual abuse; ASA = adult sexual abuse; Reexperiencing, Avoidance, and Hyperarousal—symptoms of posttraumatic stress disorder (PSTD). N = 1,449. All correlations are significant at $p < .01$. 

RMSEA be \( \leq .08 \), and the NNFI and GFI be \( \geq .90 \) (Kline, 1998).

The measurement model included five latent variables: one exogenous variable (CSA severity), three partially endogenous variables (PTSD reexperiencing, PTSD avoidance, and PTSD hyperarousal), and one fully endogenous variable (ASA severity). As described above, CSA severity and ASA severity each had four indicators: frequency, duration, type of abuse or assault, and level of force. Each of the three PTSD symptom clusters (PTSD reexperiencing, PTSD avoidance, and PTSD hyperarousal) had two observed indicators created by arbitrarily splitting each symptom cluster scale into two scales using odd versus even items. For both CSA and ASA, duration showed moderate positive skew and was therefore subjected to a logarithmic transformation. The data were otherwise univariate and multivariate normal.

The initial measurement model showed statistically significant loadings of each observed indicator on the specified latent construct, \( \chi^2 (1,448) = 5.20, p < .001 \), and adequate model fit, \( \chi^2 (67, N = 1,449) = 577.38, p < .001 \), RMSEA = .073, NNFI = .958, GFI = .946. However, examination of the residuals and modification indices suggested that we allow two pairs of indicator variables to covary (ASA frequency and duration, and ASA type and force). It is not surprising that frequency and duration were positively associated because abuse that occurs over a longer duration is likely to occur more times. Similarly, more extreme types of sexual abuse were more likely to involve force than less-extreme types. The modified model provided a good fit to the data, \( \chi^2 (65, N = 1,449) = 430.85, p < .001 \), RMSEA = .062, NNFI = .969, GFI = .959.

We first tested a simple, unmediated structural model that included only a direct effect of CSA severity on ASA severity. The model fit was adequate, \( \chi^2 (71, N = 1,449) = 585.57, p < .001 \), RMSEA = .071, NNFI = .960, GFI = .945. Moreover, the CSA–ASA path coefficient was statistically significant, confirming the existence of the revictimization effect, \( b (SE) = .11 (.04) \), \( t(1,448) = 3.92, p < .01 \). Next, we tested the proposed mediational structural model, which included paths from CSA severity to each of the three PTSD symptom clusters, and from each PTSD symptom cluster to ASA severity (see Figure 1). In addition, to allow for the possibility that CSA severity has effects on ASA severity that are not mediated by PTSD, we included a direct path from CSA severity to ASA severity. Because they constitute elements of a unified syndrome and because previous research (e.g., Cross & McCanne, 2001) has shown that the three PTSD symptom clusters are strongly related, the latent variables representing the PTSD symptom clusters were allowed to correlate.

The structural model provided a good fit to the data, \( \chi^2 (65, N = 1,449) = 430.85, p < .001 \), RMSEA = .062, NNFI = .969, GFI = .959. Moreover, the fit was significantly better than that of the simple, unmediated model, \( \chi^2 \Delta (6, N = 1,449) = 154.72, p < .001 \). The path coefficients for the measurement model were all statistically significant (\( p < .001 \); see Table 2). Examination of the structural path coefficients indicated that CSA severity was positively associated with all three PTSD symptom

<table>
<thead>
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<th>Latent construct and indicator variable</th>
<th>Path coefficient</th>
<th>Error</th>
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<tbody>
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<td>Childhood sexual abuse severity</td>
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<td>Frequency</td>
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<td>Duration</td>
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<tr>
<td>Type</td>
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<td>.04</td>
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<td>PTSD reexperiencing</td>
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<tr>
<td>Odd questions</td>
<td>.95**</td>
<td>.08</td>
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<tr>
<td>Even questions</td>
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<td>PTSD avoidance</td>
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<td>Odd questions</td>
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** \( p < .01 \).
clusters. In contrast, only PTSD hyperarousal significantly predicted increased ASA severity; the effects of reexperiencing and avoidance on ASA severity were not statistically significant, $t_s (1,448) = 0.29$ and 1.20, respectively.

Posttraumatic stress disorder arousal was a significant mediator of sexual revictimization effects. This is reflected by the fact that the magnitude of the path coefficient for the direct effect of CSA severity on ASA severity was smaller in the mediated model (.07) than in the unmediated model (.11). In addition, the indirect (mediated) effect was statistically significant according to the conservative Sobel test, $t(1,448) = 3.95$, $p < .01$ (see MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). This indicates that the CSA severity–ASA severity relationship is significantly mediated by PTSD, and specifically by PTSD hyperarousal. Child sexual abuse severity and PTSD together accounted for 11% of the total variance in ASA severity, and the indirect effect of CSA severity on ASA severity, mediated by PTSD hyperarousal, accounted for over one third (39%) of the total effects of CSA severity on ASA severity.

**DISCUSSION**

Our results show that PTSD is a significant mediator of the relationship between CSA and ASA. At the zero-order level, each of the three PTSD symptom clusters was significantly associated with all indicators of both CSA severity and ASA severity. However, the results of our SEM analyses indicated that only the hyperarousal cluster of PTSD significantly mediated the relationship between CSA and ASA severity; although both reexperiencing and avoidance were associated with CSA, they did not predict adult revictimization. The difference between the results of the SEM and the univariate analyses highlights the advantages of SEM, which controlled for the relatively high intercorrelations among the symptom clusters and eliminated the influence of measurement error, enabling us to isolate more precisely the role of individual symptom clusters.

Previous studies purporting to examine the mediating role of PTSD in sexual revictimization (Arata, 2000; Messman-Moore et al., 2005; Sandberg et al., 1999) used measures of PTSD that assess only the reexperiencing and avoidance components. Given our finding, that hyperarousal was the only symptom cluster to mediate the relationship significantly, this is a serious limitation. Nonetheless, both Arata (2000) and Messman-Moore et al. (2005) found that PTSD—along with other factors—mediated sexual revictimization effects. It is not surprising that a global measure of PTSD—even one that omits the hyperarousal cluster—would result in significant mediation, given the high intercorrelations among the clusters. Thus, the present study can be considered a replication and extension of Arata’s (2000) and Messman-Moore et al.’s (2005) studies.

The other previous study that examined PTSD as a mediator of sexual revictimization (Sandberg et al., 1999) did not find support for mediation. The reason for this discrepancy is not clear. However, as Sandberg et al. note, it is possible that the 10-week duration of their study was too short to reveal effects of symptoms on later victimization. (The other prospective study, conducted by Messman-Moore and colleagues (2005) had 8-month duration.) It should also be noted that the mediation effect in the Sandberg et al. study narrowly missed statistical significance. Although the correlation between PTSD and ASA was not statistically significant ($r = .11$), it was trivially different from the statistically significant correlation between prior victimization and PTSD ($r = .12$). The balance of the evidence therefore suggests that PTSD symptoms do constitute a mediator of sexual revictimization effects.

The present study was the first to examine the individual and joint effects of the reexperiencing, avoidance, and hyperarousal symptom clusters that comprise PTSD in explaining sexual revictimization. Our findings suggest that, of these three symptom clusters, hyperarousal plays the primary role in explaining the association between CSA severity and ASA severity. Given the high intercorrelations among symptom clusters, it is particularly important to replicate these findings with other measures and in other kinds of samples to establish their robustness. Nonetheless, if replicated, our findings may suggest that therapists working with women who display PTSD symptoms pay particular attention to hyperarousal in treating their
clients. Moreover, in addition to constituting a component of PTSD, arousal is a common element of many other disorders, such as anxiety disorders. Our findings raise the possibility that women with other disorders involving high levels of hyperarousal may also be at heightened risk of sexual revictimization. However, PTSD-arousal is likely to differ from the arousal present in other anxiety disorders, in that PTSD-arousal may be more likely to be strongly linked to situational cues. Future research could examine these issues by evaluating the anxiety levels and behavioral responses of women with PTSD and women with other anxiety disorders in different situational contexts.

At a theoretical level, assessing the role of each symptom cluster may help to shed light on the specific mechanisms by which PTSD influences sexual revictimization effects. This, in turn, may provide therapists with ideas about how to intervene to stop the cycle of sexual revictimization. The present results are consistent with explanations that highlight mediating processes likely to be exacerbated by high levels of hyperarousal. For example, our findings are consistent with the notion that PTSD leads to revictimization because the high levels of hyperarousal associated with PTSD may interfere with the individual’s ability to discriminate and respond appropriately to situation-specific danger cues. Orcutt et al. (2002) suggested that hyperarousal symptoms “may lead to risk perception that has high sensitivity but low specificity” (p. 264). That is, hyperarousal may result in increased awareness of danger cues, but reduced ability to differentiate more from less-serious cues. There are doubtless many routes through which hyperarousal might influence cognition, affect, and behavior in ways that increase the likelihood of sexual victimization. The present study was not designed to address the specific mechanisms by which PTSD symptoms led to revictimization; pending confirmation of our results, it would be important to elaborate the different arousal-based mechanisms that might lead to increase risk of ASA, and to design studies that directly test for the operation of these specific mechanisms. It would also be interesting to examine the generality of the present findings empirically to determine whether different PTSD symptom clusters or different specific mechanisms underlie retraumatization effects in different populations or for exposure to different types of traumas. For example, Orcutt et al. (2002) suggested that the reexperiencing cluster may be most important in mediating the association between combat exposure and exposure to subsequent traumatic events among veterans.

We tested a relatively simple model in which the association between CSA severity and ASA severity was partially explained by the intervening variable of PTSD symptomatology. Consistent with this model, in our sample the average age of onset of PTSD ($M = 15.1$, $SD = 3.80$) was 13.2 months prior to the average age at the first experience of ASA ($M = 16.2$, $SD = 1.60$). Thus, our results are consistent with those of previous research showing that PTSD is associated with increased risk of subsequent ASA (Acierno et al., 1999; Arata, 2000; Messman-Moore et al., 2005). However, previous research has also documented the reverse direction of causality: Just as PTSD can increase the risk or severity of ASA, ASA can also result in PTSD (e.g., Foa et al., 1995; Resnick et al., 1993). Although we focused on the potential role of PTSD in increasing the severity of subsequent ASA, the actual situation is likely to be much more complicated, involving bidirectional causal pathways between PTSD and ASA. Moreover, effects of trauma exposure may be additive or multiplicative, with each additional traumatic event increasing the likelihood of both PTSD (e.g., Arata, 2000; Boney-McCoy & Finkelhor, 1996; Classen et al., 2002) and subsequent exposure to trauma (Sorenson, Siegel, Golding, & Stein, 1991); these possibilities were not examined in the present research.

Several limitations of the present study should be noted. First, our measures were based on retrospective self-reports, which are subject to both deliberate distortions and problems of inaccurate recall. Second, the PTSD measure was not specific to CSA, but measured PTSD symptoms that could have arisen from, or been exacerbated by, other traumatic experiences. As discussed previously, victims of multiple traumas are at increased risk of PTSD; in the present study, we were unable to control for the co-occurrence of other types of traumas along with CSA. Thus, the present results do not speak to the issue of whether PTSD
symptoms resulting from other kinds of trauma may increase severity of ASA as much as PTSD symptoms resulting from CSA. Third, our sample consisted of female college students. Although this is an important population to study because female college students report high rates of ASA (e.g., Schaaf & McCanne, 1998), it is important to replicate our findings in community and clinical samples. An important limitation of the present research is our reliance on a cross-sectional design to address a causal hypothesis. It is important to bear in mind that although structural models imply a causal ordering of variables, they do not constitute evidence of causality. To examine the causal role of PTSD in sexual revictimization directly, and to examine patterns of reciprocal causality, it is necessary to conduct longitudinal research. In conducting such research, careful consideration should be given to the period studied, as the timing of both PTSD symptoms and sexual revictimization are likely to be highly variable across women.

In the present study, PTSD symptoms accounted for over a third of the variance in sexual revictimization, and the strength of the direct revictimization effect was reduced when PTSD symptoms were included in the model. This is evidence for a mediating role of PTSD—and particularly of PTSD hyperarousal—in sexual revictimization. Nonetheless, more than half of the variance in sexual revictimization remains to be explained. Ultimately, a full and accurate accounting of the relationships between CSA and ASA is likely to include a number of additional mediators (see Messman-Moore & Long, 2003), as well as reciprocal causal pathways, with trauma exposure increasing PTSD and PTSD increasing the likelihood of subsequent trauma exposure. The present study—like research investigating other specific mediators of the CSA–ASA relationship—is an important building block toward the ultimate development of such comprehensive explanatory models.

REFERENCES


PTSD as a Mediator of Sexual Revictimization


