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Coping, Emotion Regulation, and Self-Blame as Mediators of Sexual Abuse and Psychological Symptoms in Adult Sexual Assault

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Abstract

This study examined whether coping, emotion regulation, and self-blame mediate relationships of trauma histories with PTSD and depression in adult sexual assault (ASA) victims (N = 1863). A path analysis showed that theorized mediators partially mediated associations between trauma history variables and psychological symptoms. Specifically, child sexual abuse (CSA) severity was related to greater PTSD and depression indirectly through maladaptive coping and decreased emotion regulation, but not self-blame. Other traumas had direct relationships with symptoms and partially mediated effects through maladaptive coping and emotion regulation. CSA was unrelated to self-blame, but other traumas were related to greater self-blame. Results differed according to whether women had counseling post-assault. Implications are drawn for future research and clinical treatment of ASA victims.

Keywords

child sexual abuse; psychological symptoms; mediators; counseling; structural equation modeling

Coping, Emotion Regulation, and Self-Blame as Mediators of Sexual Abuse and Psychological Symptoms in Adult Sexual Assault

Sexual assault victims commonly experience psychological sequelae of PTSD and depressive symptoms (Campbell, Dworkin, & Cabral, 2009). Research suggests that various cognitive and emotional factors mediate the effects of trauma history on psychological symptoms. Several factors including maladaptive coping, emotion dysregulation, and attributions of self-blame may be particularly important for understanding recovery of adult sexual assault (ASA) survivors (Ullman, 2010). Histories of child sexual assault (CSA) and other traumas (i.e., violence, threatening environment, abusive family members) can make ASA survivors particularly vulnerable to developing PTSD and other problems (e.g., problem drinking, Ullman, Filipas, Townsend, & Starzynski, 2005). Although research shows the effects of trauma histories on psychological sequelae in ASA victims (Ullman, Townsend, Filipas, & Starzynski, 2007), little is known about the pathways through which trauma histories influence post-assault psychological sequelae. Such “mediators” are mechanism through which the predictor affects the criterion (Baron & Kenny, 1986). Research on emotional and cognitive mediators is needed to inform treatment of ASA survivors, by identifying modifiable targets for intervention (i.e., maladaptive cognitions, emotional responses).

What are some of the mechanisms through which trauma histories influence women's recovery after ASA? It is possible that a history of traumatic events allows women to learn maladaptive coping strategies and self-blame responses that they later apply to their ASA resulting in more PTSD and depressive symptoms. Furthermore, childhood trauma in particular may lead to poorer emotion regulation skills development that then carryover into adult responses to new traumatic events, though this has not been tested to date in ASA victims.

Maladaptive Coping Strategies

Maladaptive coping strategies are cognitive and behavioral strategies that alleviate distress without actually addressing the source of distress itself. These strategies can include cognitive disengagement (e.g., blocking out thoughts), behavioral disengagement (e.g., social withdrawal), denial, and/or use of substances to cope. These strategies are commonly used to deal with distress related to childhood abuse and/or ASA and can contribute to psychological symptoms of PTSD (Littleton, Horseley, & Nelson, 2007). In a community sample of ASA victims, Najdowski and Ullman (2009) found that maladaptive coping mediated the positive effect of cumulative traumas on current PTSD symptoms, indicating that cumulative traumas may impact PTSD symptoms because of increased use of maladaptive coping. This study suggested one critical pathway, maladaptive coping, through which traumas may lead to greater PTSD in ASA victims. Furthermore, Najdowski and Ullman (2011) found that maladaptive coping was related to depression in ASA victims. In a study of recent ASA victims of known perpetrators, those who used the maladaptive coping strategy of social withdrawal had worse PTSD symptoms over a three-month period (Gutner, Rizvi, Monson, & Resick, 2006).

Emotion Regulation Difficulties

Emotion regulation difficulties are also common sequelae of child maltreatment and are associated with psychological problems (Ehring & Quack, 2010). Emotion regulation difficulties include maladaptive ways of responding to emotions such as: nonacceptance of one's emotions (e.g., difficulty/lack of acceptance of one's feelings), difficulties in controlling one's behavior during emotional distress, and deficits in using emotions as information (Gratz & Roemer, 2004). Poor emotion regulation skills may also mediate the effects of CSA and other traumas on post-ASA recovery. In a cross-sectional study of college women, CSA and child physical abuse (CPA) were both predictive of poorer emotion regulation, perhaps because abuse disrupts the development of emotion regulatory mechanisms (Messman-Moore, Walsh, & DiLillo, 2010). Researchers have found evidence that emotional regulation may mediate the effects of various form of maltreatment, including CSA, on internalizing symptoms in children (Kim & Cicchetti, 2010). However, the authors did not evaluate competing mediators. Research has yet to investigate poor emotion regulation as a risk factor for worse psychological outcomes in ASA victims, yet higher rates of depressive and PTSD symptoms observed in women with ASA histories (Campbell et al., 2009) may be in part due to emotion regulation deficits.

Self-Blame Appraisals

Cognitive appraisals such as beliefs about why the assault occurred are predictive of psychological symptoms after ASA (Frazier, 2003; Koss, Figueredo, & Prince, 2002). When appraising the assault, women can attribute its cause to their own behavior in the situation (e.g., not being careful enough) or to their character in general (e.g., being a reckless person). According to attribution theory, beliefs about one's character are stable (i.e., resistant to change), global, internal attributions (Weiner, 1985). Such characterological self-blame attributions are common responses to CSA and ASA (Breitenbecher, 2006; Whiffen & MacIntosh, 2005), and unfortunately are harder to change than other attributions. For

example, although victims engage in behavioral self-blame post-assault, blaming one's character has been shown to relate to worse PTSD symptoms CSA and ASA victims (Frazier, 2003; Koss et al., 2002; Ullman & Filipas, 2005). Women with more extensive histories of abuse starting at an earlier age, such as CSA, may develop feelings of low self-worth and shame, and thus blame themselves feeling that the abuse was their fault (Arata, 1999; Feiring, Taska, & Chen, 2002). This phenomenon may help to explain why women with more extensive histories of sexual and nonsexual traumas have greater psychological symptoms in adulthood (Briere, Kaltman, & Green, 2008).

Women with CSA histories engage in more maladaptive coping and self-blame, both of which are related to worse psychological outcomes. Filipas and Ullman's (2006) cross-sectional research showed that college women who experienced ASA were more likely to engage in maladaptive coping strategies (i.e., acting out sexually, drinking to cope, self-blaming, withdrawing from others) if they had also experienced CSA than if they had not. This finding suggests that early trauma, particularly CSA, may lead to greater maladaptive coping. Other traumatic events and cumulative trauma exposure may also contribute to greater psychological symptomatology. CSA and abuse severity may be especially important in discriminating which ASA victims have more difficulty recovering, especially since we know that more severe CSA is more strongly related to PTSD (Kendall-Tackett, Williams, & Finkelhor, 1993).

Present Study

In a previous study, multiple ASA's were related to women's increased use of both maladaptive and adaptive coping efforts over a 1-year period (Najdowski & Ullman, 2011). However, studies have yet to integrate multiple mediators to determine whether they all play roles in explaining links of CSA and other trauma history variables with PTSD and depressive symptoms in ASA victims. In the present study, we extend this past work to a diverse community sample of ASA victims to test whether multiple mediators (e.g., maladaptive coping, emotion regulation, characterological self-blame) help to explain the relationships of trauma history variables (CSA, other traumas) to current symptoms (e.g., PTSD and depression). We hypothesize that CSA severity and other traumatic events will each be related to greater depression and PTSD (Ullman, Townsend, Filipas, & Starzynski, 2007) and to greater maladaptive coping (Fortier et al., 2009; Ullman, Townsend et al., 2007). We expect that part of the link between CSA and other traumas with current PTSD and depressive symptoms will be explained by women's use of maladaptive coping, emotion self-regulation difficulties, and greater characterological self-blame. Such a model test is needed to better understand pathways through which trauma variables impact ASA victims' psychological symptoms. These findings can inform clinical treatment with victims, by identifying targets to modify (beliefs and ways of coping, managing one's emotions).

The rationale for these hypotheses is that trauma exposure increases women's tendency to engage in maladaptive forms of coping (e.g., avoidance coping, denial, use of substances to cope). Therefore, victims with trauma histories are likely to be more distressed than those without previous traumas and hence more vulnerable prior to ASA experiences. Given that child trauma is associated with emotion dysregulation problems, we expect that victims with histories of childhood and other traumas will also have more problems regulating their emotions than victims without additional traumas. Finally, given that CSA is related to greater characterological self-blame, we expect that self-blame for ASA will be greater for those with CSA and perhaps other traumas as well. Should these mediational effects be significant, even in part, it would suggest that emotional and cognitive responses to trauma exposure could explain varying levels of symptoms following ASA and that such modifiable mediators should be targeted in treating sexually assaulted women with additional trauma histories. To test our hypotheses, we developed a path model with two exogenous trauma

variables (number of traumatic events, CSA), three correlated mediators (maladaptive coping, emotion regulation, characterological self-blame), and two dependent variables (PTSD, depression).

Method

Sample

A volunteer sample of women ($N = 1863$) from the Chicagoland area, age ranging from 18 to 71 ($M = 31.1$, $SD = 12.2$), completed a mail survey. The sample was ethnically diverse (45% African-American, 35% White, 2% Asian, 8.1% other; 14% Hispanic, assessed separately). The sample was well-educated (34.6% college degree or higher, 43.5% some college, 21.9% high school or less). Just under half of women (46.8%) were currently employed, although income was relatively low (68% household incomes less than \$30,000). The response rate was 85% ($N = 1863$), which is the percent of women returning surveys of those sent surveys.

Procedure

Recruitment was accomplished via weekly advertisements in local newspapers, on Craigslist, and through university mass mail. In addition, we posted fliers in the community, at other Chicago colleges and universities, as well as at agencies that cater to community members in general and victims of violence against women specifically (e.g., community centers, cultural centers, substance abuse clinics, domestic violence and rape crisis centers). Interested women called the research office and were screened for eligibility by trained female graduate research assistants who used a telephone script describing the study. We used the following criteria to assess women's eligibility on the phone: a) had an unwanted sexual experience at the age of 14 or older, b) were 18 or older at the time of participation, and c) had previously told someone about their unwanted sexual experience. We sent eligible participants packets containing the survey, a cover letter explaining the study rationale, an informed consent sheet, a list of community resources for dealing with victimization, and a stamped return envelope for the completed survey. Follow-up calls were made after 4–6 weeks to ensure women received the survey and/or in case they had any questions. If participants said they had not received the survey or lost it, they were sent another survey packet. Participants were paid \$25 following completion and return of the survey as in past studies of this nature (Ullman, 2011). This is unlikely to have biased participation significantly, as the survey is on a sensitive topic and took approximately an hour to complete. Women in similar past studies said they completed surveys on this topic: a) to help other women, b) as part of their recovery process, and c) to do something about ASA (Ullman, 2010). The university's Institutional Review Board approved all study procedures and documents.

Measures

Sexual assault—Sexual victimization in adulthood (and childhood) (at age 14 or older) was assessed using Koss, Gidycz, & Wisniewski's (1987) Sexual Experiences Survey (SES), a valid and reliable measure of sexual assault, which was modified and used successfully by Testa, VanZile-Tamsen, Livingston, & Koss (2004) with a community sample of women. CSA (prior to age 14) was also assessed using Testa and colleagues' (2004) modified version of Koss and colleagues' (1987) Sexual Experiences Survey (SES). The age 14 criteria is part of the SES measure and was the legal age of consent when and where it was developed, including Testa et al.'s (2004) revised version used in this study. Although it is difficult to distinguish a cutoff between child and adult experiences, prior studies have found differential effects using this cutoff. The severity of male-perpetrated CSA was coded as a 5-level ordinal variable from fondling/kissing through completed rape

from SES questions prior to age 14 ($M = 1.88$, $SD = 1.72$, $\alpha = .89$). Testa's modified version assesses various forms of sexual assault including: unwanted sexual contact (e.g., Have you ever been fondled, kissed, or touched sexually when you didn't want to because you were overwhelmed by a man's continual arguments and pressure?), verbally coerced intercourse (e.g., Have you given in to sexual intercourse when you didn't want to because you were overwhelmed by a man's continual arguments and pressure?), attempted rape (e.g., Have you had a man attempt to insert his penis (but intercourse did not occur) when you didn't want to by threatening or using some degree of force (twisting your arm, holding you down, etc.)?), and rape resulting from force (e.g., Have you had sexual intercourse when you didn't want to because a man threatened or used some degree of physical force (twisting your arm, holding you down, etc.) to make you?) or incapacitation (e.g., from alcohol or drugs) (e.g., Have you been in a situation in which you were incapacitated due to alcohol or drugs (that is, passed out or unaware of what was happening) and were not able to prevent unwanted sexual intercourse from taking place?). Women answered 11 no/yes questions to indicate whether they experienced each SES item since age 14. Testa and colleagues (2004) reported the 11-item SES measure had adequate reliability ($\alpha = .73$); similar reliability was found in this sample ($\alpha = .78$). Female-perpetrated CSA (experienced by 10% of women) was assessed dichotomously, so severity could not be established to include in analyses.

Traumatic life events—Trauma history was assessed with a revised version of Goodman, Corcoran, Turner, Yuan, and Green's (1998) Stressful Life Events Screening Questionnaire, developed as a brief self-report measure of various traumatic events. Green, Chung, Daroowalla, Kaltman, and DeBeneet's (2006) revised version (SLESQ-Revised) includes child abuse and adult violence experiences and stalking ("Has anyone, male or female, ever frightened you on more than one occasion by following you, spying on you, communicating with you against your will, or engaging in other harassing acts?", Logan, 2007), to which we also added a question on neighborhood/community violence (Have you ever lived in a neighborhood or community where you felt threatened or your life was in danger?). Other items included: life-threatening illness, life-threatening accident, robbery/mugging, suicide/homicide of close other, child physical abuse, adult intimate partner violence, emotional abuse, witness another person seriously hurt/killed, combat, other experience involving threat with weapon, or some other horrifying experience. This measure is scored as the sum of 14 events experienced by each respondent (excluding CSA and ASA both assessed by the Revised SES; $M = 5.68$, $SD = 3.18$). The measure had good test-retest reliability (median Kappa = .73), and adequate convergent validity (with a lengthier interview) with a median Kappa of .64.

Maladaptive coping—Participants completed the Brief COPE, a 28-item self-report scale of coping strategies (Carver, 1997). The Brief COPE measures both problem- and emotion-focused coping strategies, as well as dysfunctional (i.e., avoidant coping) versus adaptive strategies. Carver (1997) selected items for each scale based on the strength of the factor loadings in the original data (Carver et al., 1989). The measure consists of 14 2-item subscales, and it is scored by summing ratings of how often women used each strategy to cope with a specific stressor (in this case ASA; if women had more than 1 assault, they responded with respect to their most serious assault). Strategies used in the past year to cope with the assault were assessed on a scale ranging from 1 (*I didn't do this at all*) to 4 (*I did this a lot*). In this study, based on a factor analysis of coping items, maladaptive coping was the average of responses to 8 items (behavioral disengagement, denial, self-blame, substance use; see Carver, 1997 for items). The measure was reliable ($\alpha = .81$; $M = 16.35$, $SD = 5.78$).

Emotion regulation—Difficulties with emotion regulation were measured with a modified 6-item version of the Difficulties in Emotion Regulation Scale (DERS, Gratz &

Roemer, 2004), obtained from Messman-Moore (2012). Using a 5-point scale, ranging from 0 (almost never) to 4 (almost always), participants rated how often they had felt as described by each item during the past 12 months in relation to their most serious ASA. Items assessed clarity, awareness, nonacceptance, ability to engage in goal-directed behavior despite negative emotions, ability to refrain from impulsive behavior when experiencing negative emotions and flexible use of emotion regulation strategies. A total score was computed with higher scores indicating greater emotion dysregulation. The DERS has high internal consistency, good test–retest reliability, and adequate construct and predictive validity (Gratz & Roemer, 2004). Construct validity is supported by findings that DERS scores are positively associated with experiential avoidance and negatively correlated with emotional expressivity (Gratz & Roemer, 2004). The DERS has good internal consistency and test–retest reliability (Gratz & Roemer, 2004) and had acceptable reliability in our study ($\alpha = .75$; $M = 2.76$, $SD = .94$).

Self-blame—Participants completed the Rape Attribution Questionnaire (Frazier, 2003); a valid and reliable self-report measure of ASA victims' attributions about why the assault occurred. A 5-item scale assessed characterological (e.g., "I am unlucky") self-blame. Participants rated how often they thought they were assaulted during the past year using a 5-point scales from 1 (strongly disagree) to 5 (strongly agree). Frazier reported subscale alpha coefficients (.77-.89) and test–retest reliability (.68-.80) in ASA victims. The measure had acceptable reliability in this sample ($\alpha = .76$; $M = 2.56$, $SD = .96$).

PTSD symptoms—Current PTSD symptoms were assessed with items corresponding to numbing/avoidance, physiological arousal, and intrusion/re-experiencing criteria symptoms from the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995). The PDS is a 17-item brief self-report instrument used to provide a diagnosis of PTSD based on DSM-IV criteria and has been validated with ASA victims (Foa, Cashman, Jaycox, & Perry, 1997). The PDS has good test-retest reliability ($k = .74$) for the PTSD diagnosis over an average of a two-week interval, 87.3% agreement between diagnoses for two administrations, and a correlation of .83 for symptom severity scores for two administrations. The scale has good internal consistency (alphas = .92) and validity, with a correspondence between the PDS and the Structured Clinical Interview for DSM-IV Axis I disorders PTSD module (PTSD diagnoses of $k = .59$) with 79.4% agreement between the two measures. Respondents rated how often each symptom had bothered them in the past year for the specific sexual assault experience on a four-point scale (0 = not at all to 3 = almost always). In this study, 17 Likert items were summed to assess extent of posttraumatic symptomatology ($M = 21.13$, $SD = 12.93$), and the scale was reliable ($\alpha = .93$).

Depression—Depression was measured by the CESD-10 (Andresen, Carter, Malmgren, & Patrick, 1994), a short version of the Center of Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977). The 7 items on the shortened version are rated on four-point scales to indicate respondents' symptoms. The total score is the sum of items after reversing the two positive mood items. Higher scores indicate greater depressive symptoms. Using a 4-point scale (0 = none of the time or rarely to 3 = most or all of the time), participants rated how often they had felt as described by each item during the past year. Cronbach's alpha of the CES-D was .85 in the general population and .90 for patients. Content, concurrent, and discriminant validity are supported (Radloff, 1977). The CESD-10 is correlated ($r = .97$) with the 20-item scale (Andresen et al., 1994). Responses were averaged into a reliable scale ($\alpha = .86$; $M = 2.01$, $SD = .75$).

Results

First, we examined correlations of all study variables (see Table 1). CSA and other traumatic events were related to all three mediators and both PTSD and depression. Specifically both trauma history variables were related to greater maladaptive coping, self blame, and emotion regulation difficulties. More traumatic events and greater CSA severity also each related to both greater PTSD and depressive symptoms. Maladaptive coping was correlated with poorer emotion regulation, which was related to greater PTSD. Greater maladaptive coping and emotion regulation difficulties were both related to more self-blame and depressive symptoms.

To test the effects of our three mediators (maladaptive coping, self-blame, and emotion regulation) simultaneously, we conducted an observed variables path analysis with a structural equation modeling (SEM) framework using maximum likelihood estimation. Based on significant correlations, all three mediators were allowed to covary with each other. The exogenous variables of CSA and other traumatic life events were also allowed to covary, and PTSD and depressive symptom outcome variables were allowed to covary. Paths representing these associations are not discussed, because they are not the theorized relationships of interest.

First, we ran an observed variables SEM with Amos 19 (Arbuckle, 2010) using maximum likelihood estimation. The mediation model included direct effects of exogenous trauma variables on psychological symptoms, with correlated exogenous variables and all mediators correlated. Most measures were univariate normal with skew less than 1.5 and kurtosis less than 2.0 (Kline, 1998). We used the untransformed variables, given that with larger samples effects of violations of normality assumptions regarding kurtosis are minimal (Tabachnick and Fidell, 2001). We first tested the initial full model, which included all possible direct and indirect effects. This model fit the data adequately and several pathways were nonsignificant, including two direct effects and the path of one independent variable to a mediator. The direct effects of the trauma history variables were expected to be nonsignificant on psychological symptom dependent variables, because theoretically we expected their effects would be mediated, at least partially through their effects on coping, emotion regulation deficits, and self-blame. Thus, this direct effect model was really tested simply as a precursor to the mediated effects model.

To come up with the most parsimonious model, we dropped the 3 nonsignificant (i.e., $p > .05$, standardized coefficient $< .10$) paths from (a) CSA severity to depression, (b) CSA to PTSD, and (c) CSA severity to characterological self-blame. These modifications resulted in a good model fit, $\chi^2(5, 1863) = 43.39$, $p < .001$, CFI = .99, RMSEA = .065. (See Figure 1 for the final model with standardized beta weights.). Although the χ^2 is significant, this could be due to our large sample, therefore model fit assessment is not based exclusively on this test. Fit indices exceed the recommendation of 0.95 and the RMSEA is close to 0.06, indicating good fit (Hu and Bentler, 1999). Both CSA severity and other traumatic events were related to all three mediators (maladaptive coping, emotion dysregulation, self-blame) and all three mediators predicted both psychological symptom (PTSD, depressive symptoms) dependent variables. All paths were significant as hypothesized, except that characterological self-blame was not related to dependent variables, after controlling for other mediators. Thus, part of why trauma variables lead to greater symptoms may be because they relate to increased maladaptive coping and emotion regulation problems. Traumatic events predicted greater PTSD and depressive symptoms directly. Thus, the model shows partial mediation of trauma variables on PTSD and depressive symptoms, which we confirmed with Preacher and Hayes (2008) bootstrapping test for mediation.

We were also interested in the potential differential effects of life traumas and psychological mediators on symptoms for women who had counseling after the assault versus women who did not. Thus, we separated our sample into two groups based on their responses to the following question: “Did you receive counseling or therapy for help with the feelings you had after this experience?” Response options included No; No but I plan to; Yes, immediately after; Yes, within a year; Yes, after more than one year; we used a dichotomized variable to distinguish between women who received (N = 658) and did not receive (N = 1188) therapy. We found some important differences between the two groups (Figure 2). First, the effect of CSA on both maladaptive coping and emotion regulation is larger for women who did not receive therapy than for women who received therapy, indicating that CSA survivors might benefit from therapy (i.e., therapy might reduce the negative effects of CSA on symptoms). Of interest, the effect of other life traumas did not vary across these two groups. Specifically, the relation between histories of trauma and the psychological mediators was similar for both counseling and no-counseling groups. This suggests that counseling for ASA survivors might uniquely address issues related to CSA but not other traumas. Finally, the effect of poorer emotion regulation on depression symptoms was larger for women who received counseling than those who did not. Possibly, women seeking counseling have more severe mental health symptoms and/or greater histories of other problems like substance abuse that could explain this stronger relationship.

Discussion

Research shows that CSA and other traumas affect ASA survivors’ recovery, but few studies have examined multiple mediators of trauma-symptom associations. Such research is needed to clarify the processes by which ASA victims develop psychological symptoms and to identify targets for clinical treatment. Testing these mediators in one model is a better reflection of reality, because these reactions to trauma and sexual assault co-occur and covary. By testing multiple mediators we found, as hypothesized, that child and adult trauma histories lead survivors to develop maladaptive coping strategies and greater emotion dysregulation, which in turn relate to greater psychological symptoms after ASA.

Although a model with direct and indirect effects from trauma variables to psychological symptom outcome variables had adequate fit, a reduced model removing nonsignificant links of CSA to the outcome variables fit better. Because CSA severity is one continuous measure scored from Koss’s SES measure, it might not be as powerful as the traumatic events measure - which was a summed measure of multiple types of lifetime traumatic events, including interpersonal and contextual traumas. Also, the effect of CSA may have been completely explained by the mediators, which may be why the direct path was rendered nonsignificant. In contrast, the effect of other traumas was only partially mediated, leaving enough variance for the direct effect of other traumas to remain significant.

Maladaptive Coping

Child sexual abuse was positively related to maladaptive coping, consistent with past research showing that child abuse victims engage in greater maladaptive coping (Littleton et al., 2007). Past research has also shown that maladaptive coping (including forms of avoidance coping and substance use coping) relates to more PTSD and depression in victims of CSA and ASA (Ullman et al., 2007), this study confirmed that finding yet again in another large community-residing sample of ASA survivors.

Self-Blame

It is unclear why CSA was not related to greater characterological self-blame as in some past research in ASA survivors (Filipas & Ullman, 2006). Our analysis was a more conservative

test of the role of characterological self-blame in the context of testing multiple mediators simultaneously, whereas these factors were examined separately in past studies. All mediators, except self-blame, predicted both PTSD and depressive symptoms, an important finding, given that past research shows characterological self-blame predicts worse PTSD and depressive symptoms (Arata, 1999; Najdowski & Ullman, 2009). Possibly, a model with multiple mediators leads self-blame to be less significant in comparison with other behavioral and emotional responses. In general, self-blame was medium ($M = 2.56$ on 5 point scale), indicating women on average disagreed that their character was to blame for the assault. Even if women blame themselves, how they cope and their ability to regulate their emotional states may be more important than this particular cognition. There are positive implications to these findings: Changing women's beliefs about why they were assaulted, especially if characterological in nature (e.g., I was assaulted because I am a bad person) might be unrealistic given the stable, global, internal nature of such attributions. Instead, treatment should focus on helping women identify and reduce their reliance on maladaptive coping strategies and enhance their ability to regulate their emotions with cognitive processing techniques (Resick & Schnicke, 1992).

Emotion Regulation

As expected, CSA was related to poorer emotion regulation, which confirms past work showing that child trauma relates to emotion dysregulation (Ehring & Quack, 2010; Messman-Moore et al., 2010) and extends the finding to women ASA survivors. Emotion dysregulation was the strongest predictor of PTSD, suggesting that female ASA survivors with CSA and other trauma histories may need help in learning how to better regulate their emotional states to improve their ability to manage victimization-related symptoms. Results support both expert beliefs and empirical findings showing that teaching emotion regulation skills may be an important initial component of the emotionally challenging work in therapies for complex PTSD (Cloitre, Courtois, Charuvastra, Carapezza, Stolbach, & Green, 2011; Cloitre et al., 2010). The findings also are similar to, yet extend, the research on complex posttraumatic stress disorder (CPTSD). Several researchers have hypothesized that cumulative exposure to traumas in childhood and adulthood may lead to CPTSD (Cloitre et al. 2011; Herman, 1992). Although complex PTSD is not an official diagnosis, researchers define CPTSD as a combination of PTSD symptomatology along with additional self-regulation difficulties including emotional regulation, changes in attention and consciousness, difficulty with relational capacity, somatization, and altered belief systems (Cloitre et al. 2011). Whether CPTSD should be a diagnosis or merely reflects co-occurring issues remains controversial (Resick et al., 2012). However, similar to past findings on CPTSD, childhood exposures to trauma appears predictive of having both emotional regulation difficulties and PTSD symptoms (Cloitre et al., 2009). The present study adds to the literature by examining both the relative contributions of different traumas, including CSA, along with a test of the structure of this symptom presentation.

Limitations

Although the sample size was suitably large for the demands of SEM, the generalizability of our findings is limited by the cross-sectional design and nonrepresentative sample. Also, female perpetrated assault was not studied due to use of the SES measure, which also uses age 14, an arbitrary cutoff for CSA versus ASA. Future studies would benefit from separate CSA measures and perhaps a higher age cutoff of age 16. On the positive side, our sample was diverse with respect to socioeconomic status (e.g., half of participants unemployed and/or low income), making our sample more inclusive of less-studied ASA survivors than those using college and/or representatively sampled victims.

Implications for Future Research

All three mediators were intercorrelated suggesting that future work should look at the sequencing of these cognitive, emotional, and behavioral responses to ASA to see how they interrelate over time in longitudinal samples. Perhaps if one of the factors in this interrelated chain of responses is addressed, the others will also improve and lead to better recovery in survivors. Given that coping and emotion regulation strategies are possible to teach and change over time, these may be good treatment targets. Attributions of self-blame are known to decrease over time in rape victims as well (Ullman & Najdowski, 2011), but it is not clear whether reducing characterological self-blame is feasible. Prior research showed that when controlling for other psychosocial factors such as social support, social reactions, and avoidance coping, self-blame became nonsignificant in relationship to PTSD (Ullman et al., 2007). This suggests that models should examine multiple mediators to identify which ones are most critical in predicting post-assault symptoms and targeting in treatment.

Given that PTSD is a heterogeneous diagnostic category and symptom profiles vary by trauma type (Green et al., 2006), future analyses should look at PTSD symptom clusters separately as different mediators may affect reexperiencing, numbing, avoidance, and arousal differently. One would expect that avoidance coping would be most linked to PTSD avoidance symptoms, whereas emotion dysregulation would impact reexperiencing and arousal symptoms. Perhaps substance use coping would relate to numbing symptoms of PTSD (Ullman et al., 2009). Future work should examine whether such models vary for different types of CSA, as this study examined CSA severity, but did not account age of onset, chronicity, and relationship to the perpetrator, which may moderate its impact (Ullman & Filipas, 2005).

Practice Implications

Our study showed that CSA affected mediators for women without post-assault counseling more than for women with counseling, suggesting that counseling may be ameliorating the impact of CSA on survivors' responses. Specifically, counseling may reduce the effects of CSA on maladaptive coping and emotion regulation, but not the effects of other trauma history. These results suggest that trauma-focused counseling should assess and treat effects of both CSA and other traumas. Given that results of our model differ for women who had sought counseling after the assault from those who had not, we need to study the role of counseling in impacting mediators and symptom outcomes of ASA survivors. Our findings suggest that targeting CSA histories of women ASA survivors may help to ameliorate their psychological symptoms. Thus, the deleterious effects of CSA on coping and emotion regulation were larger for women who did not benefit from counseling, whereas the effects of other traumas were almost identical. Perhaps other traumas are not commonly discussed during therapy, because women and therapists are less aware of their negative effects on mental health. These findings suggest that therapists should attempt to discuss other traumatic events with ASA victims, as they probably do with CSA, and address their potential effects on learned coping strategies, emotion regulation, and attributions. Given the strong relationship of emotion dysregulation with maladaptive coping and PTSD symptoms, we need to target abuse-related negative affect (e.g., Skills Training in Affect and Interpersonal Regulation; Cloitre, 1998) and coping (e.g., Cognitive Processing Therapy, Chard, 2005; Resick & Schnicke, 1992).

Conclusion

To summarize, our findings suggest that various cognitive and emotional mediators may help to explain the links of CSA and other traumas to psychological symptoms in female victims of ASA. Both research and clinical treatment practitioners should assess these

factors and attend to how the relationships of histories of abuse and trauma may affect symptoms differently depending on whether survivors have had counseling after the assault.

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Figure 1.

Final model of relations among trauma, mediators, and PTSD and depressive symptoms. The numbers represent standardized coefficients.

Figure 2.

Final model of relations among trauma, mediators, and PTSD and depressive symptoms for women who received/ did not receive counseling related to the sexual assault. The model includes standardized coefficients for women who did not receive counseling. Coefficients for women who did receive counseling are noted parenthetically.

Table 1

Bivariate correlations between trauma variables, coping, and symptoms (ns = 1521–1854)

	1.	2.	3.	4.	5.	6.	7.
1. Child sexual abuse severity	—	.37*	.32*	.27*	.16*	.24*	.34*
2. Trauma history		—	.30*	.23*	.18*	.32*	.37*
3. Maladaptive coping			—	.62*	.44*	.46*	.55*
4. Emotion Regulation				—	.42*	.47*	.59*
5. Self-blame					—	.31*	.38*
6. Depression						—	.58*
7. PTSD							—

Note. * $p < .01$.