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Risk for Revictimization Following Interpersonal and Noninterpersonal Trauma: Clarifying the Role of Posttraumatic Stress Symptoms and Trauma-Related Cognitions

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Abstract

One victimization experience can increase risk for subsequent victimization, known as revictimization. The aims of this study were to build on sexual revictimization research by (a) broadening understanding of revictimization to interpersonal (and potentially noninterpersonal) trauma generally and (b) gaining specificity in the mechanisms that underlie revictimization. Using a prospective multisite design, an ethnically and racially diverse sample of 453 young women from the community (age range: 18–25 years, 60.7% European American) completed an initial survey and at least one follow-up survey within the following year. Participants completed self-report measures of trauma history, posttraumatic stress symptoms, and maladaptive posttraumatic cognitions. Structural equation models revealed that interpersonal revictimization was observed when controlling for past noninterpersonal trauma, odds ratio (*OR*) = 2.27, 95% CI [1.23, 4.18], and supported the role of posttraumatic stress symptoms as a mechanism underlying such revictimization, 95% CI of indirect effect (IE) [0.08, 0.51]. Additionally, a history of noninterpersonal trauma (controlling for past interpersonal trauma) increased risk of subsequent interpersonal victimization via posttraumatic stress symptoms, 95% CI of IE [0.01, 0.38]. Notably, however, when maladaptive cognitions were included as mediators in addition to posttraumatic stress symptoms, the only unique indirect effect was for the association between interpersonal trauma and risk of revictimization specifically through perceived threat of harm, 95% CI of IE [0.05, 0.20]. These findings suggest that efforts to reduce interpersonal revictimization should target maladaptive posttraumatic cognitions, particularly perceptions of threat in the environment.

Traumatic events are common occurrences. Nearly 90% of adults in the United States report a history of trauma, and most have had multiple traumatic experiences, with the modal number being three (Kilpatrick et al., 2013). Trauma exposure can heighten an individual's risk for a variety of physical and mental health problems, including posttraumatic stress

disorder (PTSD), depression, and substance use disorders (Breslau, 2009; Schnurr & Green, 2004). Furthermore, repeated trauma exposure can have a cumulative effect on physical and mental health outcomes (Nilsson, Dahlstöm, Priebe, & Svedin, 2015; Ogle, Rubin, & Siegler, 2014). Thus, understanding the occurrence of and risk factors for repeat trauma exposure is key to informing prevention efforts.

Perhaps the most consistent predictor of future trauma exposure is a history of prior trauma exposure (e.g., Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999). This association has been particularly well established in the area of sexual assault, in which a single sexual victimization increases the risk for a subsequent sexual victimization, both across developmental periods (i.e., from childhood to adulthood; Messman-Moore & Long, 2003) and within adulthood (Daigle, Fisher, & Cullen, 2008). The concept of revictimization also extends to other forms of interpersonal victimization. For example, sexual abuse in childhood can increase an individual's risk for physical and psychological victimization by an intimate partner in adulthood (e.g., Daigneault, Hébert, & McDuff, 2009; Messman-Moore & Long, 2000). Similarly, in a national sample of children and adolescents, Finkelhor, Ormrod, and Turner (2007) found that having experienced one form of victimization (e.g., sexual assault, physical assault, maltreatment, crime) heightened an individual's risk for experiencing that same form of victimization again in the subsequent year. Building on this work, Cougle, Resnick, and Kilpatrick (2009) found that in a national sample of women, any past interpersonal trauma—defined as physical assault, rape, molestation, or attempted sexual assault—was a significant predictor of future interpersonal trauma.

As these studies have illustrated, revictimization research has most often focused on traumatic events of an interpersonal nature, such as early sexual abuse and adult sexual assault. However, it is also possible that noninterpersonal traumatic events, such as motor vehicle accidents, fires, and disasters, could increase the risk for experiencing subsequent interpersonal traumas. For example, Lilly (2011) reported positive correlations between the number of noninterpersonal and interpersonal traumatic events. Similarly, Orcutt, Erickson, and Wolfe (2002) found an association between degree of combat exposure (classified in the current study as noninterpersonal trauma) and number of traumas experienced in the following 2 years among a sample of Gulf War veterans (including a physical or sexual assault, serious accident/illness, disaster, and witnessing a violent death or injury). In addition, Cougle et al. (2009) found that prior noninterpersonal trauma increased the risk for subsequent interpersonal traumas, albeit not as much as prior interpersonal trauma.

Evidence from cross-sectional and longitudinal studies suggests that PTSD symptoms that follow interpersonal trauma may increase the risk for later interpersonal trauma (Arata, 2000; Fortier et al., 2009; Risser, Hetzel-Riggin, Thomsen, & McCanne, 2006; Ullman & Peter-Hagene, 2016). For example, PTSD symptoms that stem from intimate partner violence have been linked to later revictimization in the form of physical and psychological partner violence (Iverson et al., 2013; Kuijpers, van der Knaap, & Winkel, 2012). After an interpersonal trauma, individuals with PTSD may display increased irritability and anger around other people, which can potentially exacerbate interpersonal conflicts and increase risk for physical and psychological victimization by a partner (Kuijpers et al., 2012). The

emotional numbing associated with PTSD may also heighten risk for revictimization by dulling awareness of one's surroundings, thereby interfering with risk detection (Chu, 1992). Indeed, women who have experienced multiple sexual assaults have shown delayed recognition of risk in sexual scenarios (e.g., Marx, Calhoun, Wilson, & Meyerson, 2001; Wilson, Calhoun, & Bernat, 1999; for an exception, see Chu, DePrince, & Mauss, 2014). Finally, more extreme numbing in the form of dissociation, a common consequence of interpersonal trauma (Nilsson, Gustafsson, & Svedin, 2010), may not only blunt awareness of environmental cues signaling danger (Cloitre, Scarvalone, & Difede, 1997; Wilson et al., 1999) but also lead individuals to appear disoriented, increasing their potential to be targeted by perpetrators (Cloitre et al., 1997).

In light of these findings that PTSD following interpersonal trauma increases risk for revictimization, a logical next question is whether PTSD that arises from noninterpersonal trauma also confers risk for later interpersonal trauma. For example, it is unclear whether disaster-related PTSD might increase risk for violent interpersonal victimization. Although noninterpersonal traumas tend to be associated with less severe PTSD symptoms than interpersonal traumas (e.g., Breslau, 2009; Cogle, Resnick, & Kilpatrick, 2013), some survivors of noninterpersonal trauma experience PTSD (Kilpatrick et al., 2013). Therefore, PTSD symptoms, including anger and disrupted relationships, that follow noninterpersonal trauma might also confer risk for subsequent victimization. Although deficits in risk recognition are most commonly studied in relation to interpersonal trauma, prior work has suggested that risk recognition is not differentially impaired following interpersonal versus noninterpersonal trauma (Reichert, Segal, & Flannery-Schroeder, 2015), which indicates that this mechanism underlying revictimization could extend to individuals who initially experience a noninterpersonal trauma. More research is needed to clarify whether PTSD increases the risk of interpersonal victimization among individuals whose initial trauma was noninterpersonal in nature.

Posttraumatic cognitions may also contribute to risk for subsequent interpersonal victimization. A large body of theoretical and empirical literature has shown that trauma can negatively affect one's core beliefs and potentially shatter assumptions that the self has value, people are trustworthy, and the world is just and benevolent (Epstein, 1991; Janoff-Bulman & Frieze, 1983). In this way, trauma can influence cognitions about the self (including perceptions of one's worth and capacity to make good decisions), other people (including beliefs about the dependability and trustworthiness of others), and the world (including perceptions of safety and the predictability of events; Herman, 1992; Janoff-Bulman, 1992; Janoff-Bulman & Frieze, 1983; Vogt, Shipherd, & Resick, 2012).

Posttraumatic cognitions regarding the self, others, and the world may be particularly influenced by interpersonal traumas. As Janoff-Bulman (1992) pointed out, human behavior is typically viewed as rational. Thus, experiencing violence at the hands of another person can lead individuals to question what it was about themselves that caused them to be targeted, and it can also engender doubts about their own autonomy and capacity for self-protection. Such thoughts can negatively influence perceptions of self-worth and competence. Perceptions of others may also shift drastically after interpersonal traumas. Violence committed by a loved one can evoke feelings of betrayal whereas violence

committed by a stranger can lead to a sense that others are unpredictable. Therefore, regardless of the victim's relationship to the perpetrator, experiencing interpersonal violence may lead to a distrust of others. Interpersonal traumas can also elicit negative reactions and blame from others (Ullman, 1999), which may be interpreted as additional evidence that previously trusted others are not reliable sources of support. Additionally, interpersonal traumas can contribute to a belief that the world is unsafe. Recognizing the self as a target for violence and viewing other human beings as threatening can lead to a general sense of unpredictability about the world. Victims of interpersonal violence may also be particularly concerned that their risk for future danger is outside of their control and unlikely to be eliminated by self-initiated protective behaviors (i.e., a potential perpetrator could still seek them out as a target for violence), thereby heightening beliefs about the dangerousness of the world.

Although negative posttraumatic cognitions are most strongly associated with interpersonal traumas (Lilly, 2011; Müller et al., 2010), noninterpersonal traumas can also disrupt beliefs about the self, others, and the world. For example, after an earthquake, individuals may experience self-blame or regret about preearthquake behaviors (O'Connell, Abbott, & White, 2017), which could contribute to more general negative views of the self. Individuals may also lose faith in other people after noninterpersonal trauma (O'Connell et al., 2017); for example, if postdisaster aid is not received. Noninterpersonal traumas can also contribute to negative beliefs about the safety of the world. In support of this notion, among a sample of young adults who, as adolescents, survived a collision between a bus and a train in Israel, a higher level of exposure to the disaster was associated with more negative cognitions about the benevolence and justice of the world (Solomon, Iancu, & Tyano, 1997). In this way, negative posttraumatic cognitions regarding the self, others, and the world may have important implications following both interpersonal and noninterpersonal traumas.

Although posttraumatic cognitions are closely related to PTSD (e.g., Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; Su & Chen, 2008), the unique role of cognitions in revictimization remains unknown. Negative posttraumatic beliefs may transmit a risk for subsequent victimization by lowering an individual's activation threshold for negative thoughts, thereby increasing the likelihood he or she will interpret subsequent neutral events as negative and potentially threatening (Foa & Riggs, 1993). Following an interpersonal trauma, negative cognitions may be particularly likely to be activated in interpersonal settings that are similar to the original trauma. For example, if a woman concludes that she was sexually assaulted because she failed to control certain aspects of the situation, these negative schemas about her own loss of control and powerlessness may be activated in subsequent sexual interactions, potentially increasing the risk for revictimization (Miller, Markman, & Handley, 2007). Indeed, self-blame has been implicated as a mechanism for sexual revictimization in both cross-sectional (Arata, 2000) and prospective (Miller et al., 2007) studies. Still, it is possible that negative posttraumatic cognitions following either interpersonal or noninterpersonal trauma could contribute to risk for subsequent victimization. For example, deeply held negative beliefs, including low self-worth and a belief that others will not fulfill one's needs, can impact one's choice in partners and interpersonal relationships, which may have implications for victimization risk. Consistent with this notion, early maladaptive schemas related to mistrust, disconnection, and rejection

have been identified as mechanisms that underlie the association between childhood maltreatment and intimate partner violence victimization (Atmaca, & Gençöz, 2016; Crawford, & Wright, 2007). In addition, assumptions that the world is dangerous may lead trauma survivors to interpret neutral events as threatening, which could lead to desensitization to true threats (Messman-Moore & Long, 2003) and thereby heighten the risk for subsequent interpersonal victimization. Perceptions of the world as threatening could also lead someone to seek safety through relationships even if the available partner is less than ideal. Indeed, perceived vulnerability to threat has also been identified as a mechanism explaining the association between childhood maltreatment and intimate partner violence victimization (Atmaca, & Gençöz, 2016).

Therefore, negative posttraumatic cognitions that arise from any trauma may influence individuals' behaviors, relationships, and interactions in ways that may increase the risk for subsequent interpersonal trauma. If maladaptive cognitions do increase risk for revictimization, this would provide valuable information for clinical settings, both to inform treatment planning and educate patients. Thus, it is important to directly examine whether negative cognitions about the self, others, and the world contribute to risk for revictimization.

In sum, prior trauma increases the risk for later interpersonal victimization (e.g., Acierno et al., 1999), particularly when the past trauma is also of an interpersonal nature. The aim of the present study was to replicate and extend these findings in a prospective design. We first examined whether interpersonal and noninterpersonal trauma increase risk for subsequent interpersonal victimization among a large sample of community women. Next, we considered two mechanisms—PTSD and posttraumatic cognitions—that may account for revictimization among women with a prior history of trauma exposure. Because risk for revictimization may also be influenced by a range of contextual factors, including socioeconomic status and an ongoing relationship with a perpetrator (Grauerholz, 2000; Pittenger, Huit, & Hansen, 2016), we included changes in income and romantic relationships as covariates when we examined mediators of revictimization (see Hypotheses 2a–3b).

Risk for Subsequent Victimization

Hypothesis 1a.

Consistent with prior work on revictimization (e.g., Cougle et al., 2009), we expected that any (vs. no) history of interpersonal trauma would be uniquely associated with increased risk for subsequent interpersonal trauma within the following year (when controlling for past noninterpersonal trauma).

Hypothesis 1b.

Based on the limited research on this topic (Cougle et al., 2009; Lilly, 2011; Orcutt et al., 2002), we also expected that a history of noninterpersonal trauma would be uniquely associated with a moderate increase in risk for subsequent interpersonal trauma (when controlling for past interpersonal trauma).

Mediating Role of PTSD Symptoms

Hypothesis 2a.

Consistent with previous work pointing to PTSD as a mechanism underlying revictimization (e.g., Arata, 2000; Fortier et al., 2009), we expected that, when controlling for past noninterpersonal trauma, any (vs. no) past interpersonal trauma would be associated with more severe PTSD symptoms and, subsequently, higher risk for interpersonal trauma.

Hypothesis 2b.

Given that noninterpersonal trauma can also lead to PTSD symptoms (Kilpatrick et al., 2013), any (vs. no) past noninterpersonal trauma was also expected to increase risk for subsequent interpersonal trauma via PTSD symptoms (when controlling for past interpersonal trauma).

Mediating Role of Posttraumatic Cognitions

Hypothesis 3a.

We anticipated that, controlling for past noninterpersonal trauma, the effect of any (vs. no) past interpersonal trauma on subsequent interpersonal victimization would be indirect through maladaptive posttraumatic cognitions, separate from the influence of PTSD symptoms. Specifically, we expected that, compared to those with no prior interpersonal trauma, individuals with a history of any interpersonal trauma would experience more maladaptive cognitions about the self (i.e., Self-Worth and Judgment, which reflect confidence in one's value and abilities), others (i.e., Reliability and Trustworthiness of Others, which reflect the belief that other people are deserving of trust), and the world (i.e., Threat of Harm, which reflects perceptions of the world as dangerous and unsafe). In turn, we expected that individuals with more negative cognitions in each of these areas would have a greater likelihood of subsequent interpersonal trauma.

Hypothesis 3b.

Given that noninterpersonal traumas can also contribute to negative cognitions about the self, others, and the world (O'Connell et al., 2017; Solomon et al., 1997), posttraumatic cognitions were also expected to mediate the association between prior noninterpersonal trauma and subsequent interpersonal victimization (when controlling for past interpersonal trauma).

Method

Participants

Participants were drawn from a larger, multiwave, multisite study of emotion dysregulation and sexual revictimization (also see Jaffe et al., 2017; Watkins, Franz, DiLillo, Gratz, & Messman-Moore, 2015; Watkins et al., 2014). A total of 491 women between 18 and 25 years of age were recruited from the community at four sites in the United States (Lincoln and Omaha, Nebraska; Jackson, Mississippi; Oxford, Ohio) for a study of women's life experiences. Participants were recruited without regard to prior victimization experiences.

Participants completed self-report assessments once every 4 months for 1 year (i.e., Wave 1 at Month 0; Wave 2 at Month 4; Wave 3 at Month 8; Wave 4 at Month 12). Specifically, 426 (86.8%) women completed the first follow-up (Wave 2), 402 (81.9%) completed the second follow-up (Wave 3), and 376 (76.6%) completed the third follow-up (Wave 4). Only the 453 (92.3%) participants who completed at least one of these follow-up assessments (i.e., at Waves 2–4) were included in the current analyses. There were no significant differences in demographics or trauma history among the 453 women who completed a follow-up and the 38 who did not.

At enrollment, these 453 women ranged in age from 18 to 25 years ($M = 21.71$ years, $SD = 2.23$). With regard to ethnicity, 60.7% identified as European American, 35.8% as African American, 5.5% as Latina, 4.2% as Asian, 3.3% as American Indian, and 2.4% as other (more than one ethnicity could be endorsed). Although participants were recruited from the community, 62.4% were full-or part-time students. Of the participants who were not in school, 60.1% reported current outside employment, 29.1% reported being unemployed, and 10.8% described themselves as homemakers. Regarding participants' education attainment, 3.8% had completed some high school, 20.1% had graduated high school or received a high school equivalency diploma, 53.1% had completed some college, 14.6% had received a bachelor's degree, and 8.4% had completed some graduate or professional school or received an advanced degree. The majority of participants (56.4%) reported an annual household income of less than \$20,000 (USD). With regard to sexual orientation, 85.3% identified as heterosexual, 8.7% as bisexual, 4.0% as gay or lesbian, and 2.0% as questioning. In addition, 56.9% reported that they were currently in a romantic relationship.

Procedure

All procedures were approved by the Institutional Review Boards of all participating institutions (University of Nebraska-Lincoln; Creighton University; University of Mississippi Medical Center; Miami University). A convenience sample of community young adults was recruited for a study on "women's life experiences and adjustment." The only inclusion criteria were age (18–25 years) and gender (female). Community members were recruited using flyers, newspaper advertisements, and online advertisements. In addition, a large database of publicly available residential mailing addresses near each site was obtained from Survey Sampling International (Shelton, CT), and recruitment letters were mailed to a randomly selected subset of eligible individuals. Interested individuals were invited to the lab, where they were given an opportunity to ask questions about the study and then provided verbal and written informed consent. All surveys were administered via Qualtrics (Provo, UT), an online survey platform, although paper surveys were made available for those without Internet access. To facilitate collection of other laboratory-based assessments, Waves 1 and 3 were completed on site whereas Waves 2 and 4 were completed by emailing participants a link to a Qualtrics survey. Participants were compensated \$50 (USD) for each laboratory-based assessment and \$25 for each web-based assessment.

Measures

Traumatic events.—The Life Events Checklist (LEC; Gray, Litz, Hsu, & Lombardo, 2004) was used to assess the occurrence of potentially traumatic events. The LEC has

demonstrated adequate test–retest reliability and convergent validity (Gray et al., 2004). The measure assesses exposure to 16 types of traumatic events, and participants were given the option to report and specify another event (i.e., the “other” category). Based on participants’ text responses, events in the “other” category were recoded into one of the 16 categories when possible. At the first assessment (Wave 1), participants were asked to endorse any events they had directly experienced (“happened to me”) at any time in their lives. At each follow-up assessment (Waves 2–4), participants were asked to endorse events that had occurred within the last 4 months or since the last survey they completed. Consistent with what has been reported in prior work (e.g., Coughle et al., 2009), traumatic life events were categorized as interpersonal (i.e., physical assault, assault with a weapon, rape, or other unwanted sexual experience) or noninterpersonal (i.e., any other event).

PTSD symptoms.—The PTSD Checklist–Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993) contains 17 items that assess PTSD symptoms outlined in the fourth edition text revision of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*; American Psychiatric Association [APA], 2000). At Wave 1, participants were asked to indicate how much they had been bothered by each of these symptoms in the past month as related to a participant-identified index trauma, which was also selected at Wave 1. The severity of each symptom was rated on a scale from 0 (*not at all*) to 4 (*extremely*), and a mean PTSD symptom score was computed. The PCL-C has been shown to have acceptable convergent validity (Weathers et al., 1993) and good internal consistency (Cronbach’s $\alpha = .94$; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). In the current sample, Cronbach’s alpha was .95 for the total score.

Posttraumatic cognitions.—The Posttraumatic Maladaptive Beliefs Scale (PMBS; Vogt et al., 2012) was administered at Wave 1 to assess cognitions since the occurrence of the index trauma. This scale was designed to be applicable to a wide range of traumatic experiences and, as such, measures general beliefs about the self, others, and the world. The PMBS consists of three subscales with five items each. The Self-Worth and Judgment subscale reflects confidence in one’s value and capacity to navigate daily tasks (e.g., “I trust my own judgment”; this item is reverse-scored). The Reliability and Trustworthiness of Others subscale assesses perceptions of other individuals as deserving of trust and capable of dependable interpersonal relationships (e.g., “I feel as though I can depend on other people”; this item is reverse-scored). The Threat of Harm subscale measures perceptions that the world is dangerous (e.g., “I don’t feel safe anywhere anymore”). Each item is rated on a 7-point Likert-type scale, from 0 (*not at all true for you*) to 6 (*completely true for you*), with higher scores reflective of more maladaptive beliefs. Support for the convergent and discriminant validity, content validity, and internal consistency (subscale alpha values ranged from .71 to .76) of the scale was provided in Vogt et al.’s (2012) study. Cronbach’s alpha values for the subscales in the current study ranged from .75 to .83.

Relationship change.—At Wave 1 and each follow-up, participants were asked whether they were in a relationship with an intimate partner. At each follow-up assessment, participants also answered whether they were in a new relationship since the last time they participated. Participants were considered to have had a relationship change during the

follow-up year (relative to Wave 1) if they went from being in a relationship to not (or vice versa) or if they reported being in a new relationship at any follow-up assessment.

Income change.—At each assessment, participants were presented with 10 income ranges and asked to select their current household income. At Wave 1, household income was under \$10,000 for 41.2%, \$10,000–19,999 for 15.2%, \$20,000–29,999 for 11.9%, \$30,000–39,999 for 8.3%, \$40,000–49,999 for 4.9%, \$50,000–59,999 for 4.3%, \$60,000–74,999 for 3.8%, \$75,000–99,999 for 4.5%, \$100,000–150,000 for 3.8%, and over \$150,000 for 2.2% (all incomes reported as USD). Relative to income reported at Wave 1, participants who reported a higher income at any follow-up were considered to have an income increase and those who reported a lower income at any follow-up were considered to have an income decrease.

Data Analyses

Hypothesis 1: Risk for subsequent victimization.—We first hypothesized interpersonal and noninterpersonal trauma would increase risk for subsequent interpersonal trauma in the following year. A logistic regression was conducted in Mplus (Version 8; Muthén & Muthén, 2017) to estimate risk for any (vs. no) interpersonal victimization during the follow-up year. Predictors were two dummy-coded variables used to represent (a) any (vs. no) past interpersonal trauma and (b) any (vs. no) past noninterpersonal trauma as reported at Wave 1. This dummy coding allowed for examination of each trauma type (past interpersonal and noninterpersonal trauma) as unique predictors of subsequent interpersonal trauma while controlling for the other trauma type.

Hypothesis 2: Mediating role of PTSD symptoms.—We next hypothesized that PTSD symptoms would mediate the associations between past interpersonal or noninterpersonal trauma and risk for subsequent interpersonal trauma. Because PTSD symptoms are only applicable for individuals with a history of trauma, only the 413 participants who reported at least one lifetime traumatic event at Wave 1 were considered. The predictor variables (any past interpersonal trauma and any past noninterpersonal trauma) and outcome variable (any interpersonal trauma during the follow-up year) remained the same as in analyses for Hypothesis 1. To isolate mechanisms underlying revictimization, the following contextual factors were included as covariates: change in romantic relationships, increase in income, and decrease in income during the follow-up year. Because there was variability in the length of time between assessments, time (in months) between the initial and last assessment was also included as a covariate. The mediator and outcome variables were regressed on all covariates.

Path analyses were conducted in Mplus (Version 8; Muthén & Muthén, 2017) using maximum likelihood estimation. With regard to missing data, 20 (4.8%) participants who endorsed a traumatic event did not select an index event and therefore were not prompted to respond to linked questions on PTSD. An additional 12 (2.9%) participants had missing data for the mediator. Four (1.0%) participants also had missing data on one or more covariate but were retained in analyses by including correlations between predictor variables. Given the just-identified nature of this model, global fit indices would reflect perfect fit. Still, to evaluate fit, the estimated model was compared to a baseline model in which all paths were

constrained to 0, using a chi-square difference test. Indirect effects were examined using nonparametric bootstrapping (Shrout & Bolger, 2002) to maximize power and minimize Type I error. Specifically, 5,000 resamples were used to derive the 95% bias-corrected bootstrap confidence intervals (CIs). Confidence intervals that do not contain zero reflect the presence of an indirect effect. Unstandardized coefficients are reported given the presence of dichotomous predictors (Hayes, 2013).

Hypothesis 3: Mediating role of posttraumatic cognitions.—Building on the second hypothesis, the third hypothesis focused on the mediating role of posttraumatic cognitions (i.e., Self-Worth and Judgment, Reliability and Trustworthiness of Others, and Threat of Harm) in revictimization, above and beyond PTSD symptoms. Specifically, among the same 413 participants who reported a history of at least one traumatic event at the first assessment, we examined the indirect effects of both any past interpersonal trauma and any past noninterpersonal trauma on the presence of any interpersonal trauma during the follow-up year through PTSD symptoms and posttraumatic cognitions. As in Hypothesis 2, indicators of change in relevant contextual factors (i.e., romantic relationship change, income increase, income decrease) and the number of months between the initial and last assessment were included as covariates. The residuals of the mediators (PTSD symptoms and posttraumatic cognitions) were allowed to covary. Similar to the analyses for Hypothesis 2, maximum likelihood estimation was utilized. All participants who were missing values for PTSD were also missing values for posttraumatic cognitions; therefore, the pattern of missing data was the same as in Hypothesis 2. Global fit indices again reflected perfect fit given the just-identified nature of this model; fit was further evaluated using a chi-square difference test to compare the current model to a baseline model. Conditional indirect effects from the index trauma type to the presence of a subsequent interpersonal trauma via PTSD symptoms and posttraumatic cognitions were examined using 95% bias-corrected bootstrap confidence intervals.

Results

Descriptive Statistics

Of the 453 participants, 413 (91.2%) reported the experience of at least one potentially traumatic event, with 311 (68.7%) reporting an interpersonal trauma (i.e., physical assault, assault with a weapon, rape, or other unwanted sexual experience) and 381 (84.1%) reporting a noninterpersonal trauma at Wave 1. These figures include 279 (61.6%) participants who reported both a prior interpersonal and noninterpersonal trauma at Wave 1. Within the following year, 82 (18.1%) participants reported experiencing a new interpersonal trauma. See Table 1 for additional descriptive statistics, Table 2 for intercorrelations, and Table 3 for more details about traumatic events endorsed.

Hypothesis 1: Risk for Subsequent Victimization

A logistic regression model that included all 453 participants revealed that a history of interpersonal trauma as reported at Wave 1 was associated with a significant increase in risk for experiencing subsequent interpersonal trauma in the following year, $B = 0.82$, $SE = 0.31$, $p = .009$, $OR = 2.27$, 95% CI [1.23, 4.18], when controlling for history of noninterpersonal

trauma. Further, history of noninterpersonal trauma reported at Wave 1 was not uniquely associated with risk for subsequent interpersonal victimization in the following year, $B = 0.15$, $SE = 0.38$, $p = .684$, $OR = 1.17$, 95% CI [0.56, 2.43]. As indicated by R^2 , this model accounted for 4.5% of the variance in the occurrence of a subsequent interpersonal trauma.

Hypothesis 2: Mediating Role of PTSD Symptoms

Next, we examined the role of PTSD symptoms in revictimization among the 413 individuals who reported any prior trauma. The estimated model fit significantly better than a baseline model, $\chi^2(28, N = 413) = 66.33$, $p < .001$. Model results are displayed in Table 4, Supplementary Table 1, and Figure 1. There was a direct effect of past interpersonal trauma on a subsequent interpersonal trauma, 95% CI [0.02, 1.64], when we controlled for prior noninterpersonal trauma, PTSD symptoms, months between assessments, and relationship and income changes. In addition, there was an indirect effect from past interpersonal trauma to the occurrence of a subsequent interpersonal trauma through PTSD symptom severity, 95% CI [0.08, 0.51]. Specifically, participants who experienced a prior interpersonal trauma endorsed more severe PTSD symptoms, which, in turn, were associated with greater risk for experiencing another interpersonal trauma in the following year. Past noninterpersonal trauma did not directly increase risk for subsequent interpersonal trauma, 95% CI [-0.41, 2.27], but there was an indirect effect via PTSD symptoms, 95% CI [0.01, 0.38], when we controlled for all other study variables. As indicated by R^2 , this model accounted for 11.3% of the variance in PTSD symptoms and 14.9% of the variance in the occurrence of a subsequent interpersonal trauma.

Hypothesis 3: Mediating Role of Posttraumatic Cognitions

Finally, three types of posttraumatic cognitions (Self-Worth and Judgment, Reliability and Trustworthiness of Others, Threat of Harm) were added to the model as potential mediators. The estimated model fit significantly better than a baseline model, $\chi^2(55, N = 413) = 291.14$, $p < .001$. Results are displayed in Table 4, Supplementary Table 1, and Figure 2. There remained a conditional total indirect effect from prior interpersonal trauma to subsequent interpersonal trauma through all mediators, 95% CI [0.34, 1.95]. However, when we controlled for posttraumatic cognitions, there was no longer a unique indirect effect of past interpersonal trauma on subsequent interpersonal trauma specifically through PTSD symptoms, 95% CI [-0.11, 0.39]. Although there also were not specific indirect effects through Self-Worth and Judgment, 95% CI [-0.18, 0.20], or Reliability and Trustworthiness of Others, 95% CI [-0.23, 0.06], there was a unique indirect effect through Threat of Harm, 95% CI [0.05, 0.55]. Specifically, when controlling for past noninterpersonal trauma, PTSD symptoms, other maladaptive posttraumatic cognitions, and other covariates, perceived threat of harm emerged as a significant unique mediator of the association between past interpersonal trauma and subsequent interpersonal trauma. With regard to the influence of past noninterpersonal trauma on subsequent interpersonal trauma, there were no indirect effects present through PTSD symptoms or any posttraumatic cognitions. As indicated by R^2 , this model accounted for 11.2% of the variance in PTSD symptoms, 10.3% of the variance in Threat of Harm cognitions, 7.0% of the variance in Self-Worth and Judgment cognitions, 3.1% of the variance in cognitions related to Reliability and Trustworthiness of Others, and 17.9% of the variance in the occurrence of a subsequent interpersonal trauma.

Discussion

The aim of this study was to examine both the breadth of revictimization and the specific mechanisms that underlie revictimization. Consistent with past research that has provided evidence of interpersonal revictimization (e.g., Cougle et al., 2009; Finkelhor et al., 2007; Messman-Moore & Long, 2003), findings supported Hypothesis 1a, revealing that the experience of any past interpersonal trauma (i.e., physical or sexual assault) increased risk for a subsequent interpersonal trauma within the following year. Although a substantial portion of our sample (61.6%) reported a lifetime history of both interpersonal and noninterpersonal trauma, we statistically controlled for past noninterpersonal trauma. The fact that revictimization was observed even in the context of this relatively conservative test provides further evidence that interpersonal revictimization is a robust phenomenon.

In addition to examining interpersonal revictimization, we considered the influence of prior noninterpersonal trauma on the risk for subsequent interpersonal trauma. In contrast to the prediction made in Hypothesis 1b and some limited prior research, the results did not support a direct association between prior noninterpersonal trauma and subsequent interpersonal trauma. Previously, Cougle et al. (2009) found that prior noninterpersonal trauma increased risk for interpersonal violence ($OR = 1.48$) within the following 2 years in a large sample of 2,850 women, when controlling for the influence of depression, substance use, and demographics. Given that the effect size in the present study was somewhat similar ($OR = 1.17$), it is possible that the strength of the association between past noninterpersonal trauma and subsequent interpersonal victimization is weak enough that it only reaches a statistically significant level in larger samples or during longer follow-up periods (e.g. 2 years vs. 1 year in the current study). Consistent with this notion, in a large sample of nearly 3,000 veterans followed over 2 years, Orcutt et al. (2002) found a modest but significant association between combat exposure and subsequent trauma exposure, including interpersonal victimization.

Potential mechanisms that underlie interpersonal revictimization were also examined. When we controlled for the influence of noninterpersonal trauma and other covariates, a history of interpersonal trauma was associated with more severe PTSD symptoms, which, in turn, were associated with heightened risk for experiencing a subsequent interpersonal victimization in the following year. This finding is consistent with Hypothesis 2a and a large body of work that has identified PTSD as a mechanism underlying sexual assault revictimization (e.g., Arata, 2000; Fortier et al., 2009; Risser et al., 2006; Ullman & Peter-Hagene, 2016). Moreover, this finding extends prior work by suggesting that PTSD symptoms increase the risk for interpersonal revictimization broadly and may not be specific to sexual victimization. However, because sexual and physical victimization were not examined separately in this study, further research is needed to examine whether physical assault-related PTSD can increase the risk for subsequent experiences of sexual assault, and vice versa.

Consistent with our expectations and Hypothesis 2b, when we controlled for prior interpersonal trauma and all other covariates, a history of noninterpersonal trauma was indirectly associated with increased risk of subsequent interpersonal trauma through

heightened PTSD symptoms. Together with the aforementioned finding regarding the lack of a direct association between noninterpersonal trauma and subsequent interpersonal trauma, this suggests that any small effect of noninterpersonal trauma on risk for subsequent interpersonal trauma may be due to the presence of PTSD symptoms. This is consistent with Cougle and colleagues' (2009) finding that past noninterpersonal trauma did not remain a significant predictor of subsequent interpersonal trauma after controlling for PTSD symptoms. This finding indicates that PTSD-related impairment, such as anger or irritability, may affect interpersonal relationships and increase the risk for conflict or violence. After a noninterpersonal trauma (such as a car accident), PTSD-related avoidance behaviors (e.g., not driving oneself and therefore relying on others for rides; substance use) may also lead to changes in interpersonal relationships that affect risk for experiencing violence.

Nonetheless, PTSD symptoms were not the most important mechanism through which one trauma increased the risk for another in the current study. Instead, when both PTSD symptoms and maladaptive posttraumatic cognitions were considered simultaneously, maladaptive cognitions associated with threat of harm emerged as the only unique factor that accounted for the association between past and subsequent interpersonal trauma, which provides partial support for Hypothesis 3a. Specifically, following the experience of any (vs. no) interpersonal trauma, participants reported heightened concerns about threat and the dangerousness of the world, which may have increased hyperarousal and impaired risk recognition over time. This is consistent with research that has suggested impaired risk recognition may play a central role in the occurrence of revictimization (Gidycz, McNamara, & Edwards, 2006). Alternatively, perceived threat of harm may have been more longstanding for some individuals, which may reflect realistic concerns of danger among those in adverse environments where interpersonal violence may be more common. For example, risk of revictimization is elevated in communities of low socioeconomic status (Klest, 2012). Thus, heightened risk of revictimization via perceived threat of harm in the current study may reflect realistic risk assessment.

Contrary to expectations, however, not all maladaptive cognitions increased the risk of revictimization after controlling for the influence of PTSD symptoms. Specifically, although past interpersonal trauma increased maladaptive perceptions of one's self-worth and judgment as well as concerns about the reliability and trustworthiness of others, these negative cognitions did not uniquely influence the risk for subsequent interpersonal trauma. Thus, they were not supported as mechanisms underlying interpersonal revictimization. Although we expected these self-and other-related cognitions to have implications for revictimization (given their potential negative impact on interpersonal interactions), the posttraumatic cognitions examined here were highly correlated. Therefore, although only maladaptive cognitions related to the world were uniquely associated with risk for interpersonal revictimization in this study, cognitions related to the self and others may also impact revictimization if examined separately. Nonetheless, the findings that perceived threat of harm in the world emerged as the only unique mediator of interpersonal revictimization may suggest the particular relevance of more general (i.e., threat of harm in general) versus specific (i.e., self-or other-related) maladaptive cognitions to risk for interpersonal revictimization.

Alternatively, risk for revictimization may depend less on the presence of maladaptive cognitions about oneself or others and more on how one responds to these cognitions. Some individuals may respond by avoiding others, thereby reducing the likelihood of any interpersonal interactions, let alone interactions that end in violence. Conversely, other individuals may continue to pursue intimate relationships, but may doubt their self-worth and have difficulty determining whom to trust, which may increase their risk for violence in relationships (Davis & Petretic-Jackson, 2000). Thus, individuals might respond to negative cognitions about the self and others in ways that could increase or decrease their risk for revictimization and ultimately limit the predictive capacity of posttraumatic cognitions alone in interpersonal revictimization. Future research should consider moderators of the association between posttraumatic cognitions and subsequent revictimization to facilitate identification of distinct risk profiles.

Finally, contrary to Hypothesis 3b, posttraumatic cognitions did not explain the association between past noninterpersonal trauma and subsequent interpersonal trauma. This may be due to the relatively weak association between noninterpersonal trauma and posttraumatic cognitions we found in this study, which is consistent with prior work showing that posttraumatic cognitions are more strongly influenced by interpersonal trauma and relatively unrelated to noninterpersonal trauma (Lilly, 2011; Müller et al., 2010). Although this suggests that being exposed to a noninterpersonal trauma (including a minor car accident or minor flooding) may not have a strong impact on later maladaptive cognitions, it is possible that a particularly severe noninterpersonal trauma (e.g., a severe transportation accident or natural disaster with fatalities) could lead to negative cognitions, especially regarding the dangerousness of the world (Solomon et al., 1997). Future studies should consider this possibility in clinical samples.

Limitations of the current study suggest areas for future research. First, although this study focused on PTSD-related symptoms and cognitions as important mechanisms underlying the risk for revictimization, other putative mechanisms (e.g., depression, substance abuse, risky sexual behavior) also warrant examination (Messman-Moore & Long, 2003). Although we made efforts to control for potential environmental changes, including changes in romantic relationships and income during follow-up, future studies should consider a broader ecological model of revictimization (Grauerholz, 2000; Messman-Moore & Long, 2003; Pittenger et al., 2016). Direct assessment of changes in living situation, shifts in social networks, and broader macrosystem policy or attitude changes would provide researchers with greater specificity in understanding individual-level mechanisms of revictimization, including PTSD and cognitions. Second, although we considered past interpersonal and noninterpersonal trauma as unique predictors of subsequent interpersonal trauma when controlling for the other trauma type, these predictors may be correlated (Lilly, 2011). Future studies should consider revictimization risk in exclusive groups of individuals exposed to only interpersonal or noninterpersonal trauma. Third, data collection began prior to the release of the fifth edition of the *DSM* (*DSM-5*; APA, 2013) and, thus, occurred before trauma-related negative alterations in cognitions were integrated into the assessment and conceptualization of PTSD. Although this allowed us to isolate the unique role of posttraumatic maladaptive cognitions versus other PTSD symptoms in the risk for revictimization, future studies should examine the role of cognitions related to threat

perception in the context of the *DSM-5* conceptualization of PTSD. Fourth, the current study involved only young adult women. Additional research is needed to determine whether findings generalize to men and older adults, and whether patterns of revictimization extend across the lifespan. Finally, although we suggest that threat-related cognitions may increase risk for revictimization due to impairments in risk recognition, additional work is needed to examine this possibility. For example, research is needed to examine whether modifying maladaptive cognitions through therapy can restore trauma survivors' natural capacity to recognize risks and, in turn, reduce risk of revictimization.

Through this study, we suggest an important distinction between patterns of revictimization (i.e., a phenomenon that reflects risk relations among specifically interpersonal traumatic experiences) and retraumatization (i.e., a phenomenon that reflects risk relations among any type of traumatic experiences). In this prospective study of young adult women, robust support was found for revictimization but not retraumatization. Results suggest that whereas PTSD symptoms played a role in both revictimization and retraumatization, perceived threat of harm was a mechanism specific to revictimization. Therefore, changes in posttraumatic cognitions may be an important target for intervention, both to reduce PTSD symptoms (e.g., Zalta et al., 2014) and to reduce risk for revictimization. Preliminary work by Iverson and colleagues (2011) indicated that reductions in PTSD following cognitive processing therapy (CPT; Resick & Schnicke, 1992; also see Resick, Monson, & Chard, 2017) were associated with reduced risk for revictimization in the form of physical partner violence. Additional research should explore whether treatments that target maladaptive posttraumatic cognitions could reduce the risk of subsequent interpersonal trauma exposure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Model examining the mediating role of posttraumatic stress disorder (PTSD) in revictimization. Dashed lines indicate nonsignificant paths. Unstandardized estimates (and standard errors) are shown. Control variables and covariances are shown by gray arrows. Full model results are in Supplementary Table 1. * indicates 95% confidence intervals do not contain zero and therefore signify an effect that is present.

Figure 2.

Model examining the mediating role of posttraumatic cognitions in revictimization, above and beyond the influence of posttraumatic stress disorder (PTSD) symptoms. Dashed lines indicate nonsignificant paths. Control variables and covariances are shown by gray arrows. All path coefficients can be seen in Supplementary Table 1.

Table 1

Descriptive Statistics

	Range	<i>n</i>	%	Total N
1. Past IPT	0, 1	311	68.7%	453
2. Past non-IPT	0, 1	381	84.1%	453
3. FU IPT	0, 1	82	18.1%	453
4. FU non-IPT	0, 1	198	43.7%	453
5. Relationship change	0, 1	199	44.0%	452
6. Income decrease	0, 1	131	29.3%	447
7. Income increase	0, 1	164	36.7%	447
		<u>Mean</u>	<u>SD</u>	
8. PTSD symptoms	0.00 – 4.00	0.91	0.91	381
9. SWJ	0.00 – 5.80	1.36	1.14	381
10. RTO	0.00 – 6.00	1.66	1.28	381
11. TH	0.00 – 6.00	1.77	1.17	381
12. Months between Wave 1 and last FU	3.42 – 15.08	11.29	2.27	453

Note. SD = standard deviation; IPT = interpersonal trauma, non-IPT = noninterpersonal trauma, FU = follow-up, PTSD = posttraumatic stress disorder, SWJ = Self-Worth and Judgment, RTO = Reliability and Trustworthiness of Others, TH = Threat of Harm.

Table 2

Intercorrelations Among Study Variables

	Correlations										
	2	3	4	5	6	7	8	9	10	11	12
1. Past IPT	.41 **	.27 **	.14	.18 *	-.10	-.07	.28 **	.23 **	.15 **	.25 **	-.02
2. Past non-IPT	—	.11	.25 **	.06	.01	-.06	.06	.03	.03	.06	-.09
3. FU IPT		—	.45 **	.17 *	-.23 *	.05	.19 **	.12 *	.06	.21 **	.04
4. FU non-IPT			—	.05	.07	.01	.06	.08	.11 *	-.01	.05
5. Relationship change				—	.11	-.10	.02	.02	.03	.00	.12 *
6. Income decrease					—	-.41 **	-.03	-.09	.01	-.09	.08
7. Income increase						—	.00	.04	.00	-.06	.08
8. PTSD symptoms							—	.46 **	.37 **	.60 **	-.13 **
9. SWJ								—	.51 **	.45 **	-.04
10. RTO									—	.40 **	-.06
11. TH										—	-.12 *
12. Months between Wave 1 and last FU											—

Note. IPT = interpersonal trauma, non-IPT = noninterpersonal trauma, FU = follow-up, PTSD = posttraumatic stress disorder, SWJ = Self-Worth and Judgment, RTO = Reliability and Trustworthiness of Others, TH = Threat of Harm. Correlations shown between dichotomous variables are tetrachoric correlations.

**
 $p < .01$.

*
 $p < .05$.

Table 3

Potentially Traumatic Events Reported by All Participants

Trauma Type	Wave 1		Follow-Up	
	<i>n</i>	%	<i>n</i>	%
Any interpersonal trauma	311	68.7	82	18.1
Physical assault	140	30.9	38	8.4
Assault with a weapon	37	8.2	20	4.4
Rape	107	23.6	22	4.9
Other unwanted sexual experience	248	54.7	59	13.0
Any noninterpersonal trauma	381	84.1	198	43.7
Natural disaster	113	24.9	26	5.7
Fire or explosion	41	9.1	24	5.3
Transportation accident	254	56.1	74	16.3
Serious accident	46	10.2	27	6.0
Exposure to toxic substances	12	2.6	18	4.0
Combat	4	0.9	15	3.3
Captivity	6	1.3	15	3.3
Illness or injury	28	6.2	26	5.7
Severe human suffering	9	2.0	18	4.0
Sudden, violent death	44	9.7	25	5.5
Sudden, unexpected death of someone close to you	182	40.2	72	15.9
Serious injury, harm or death you caused to someone else	16	3.5	19	4.2
Other event	79	17.4	83	18.3

Note. *N* = 453.

Table 4Indirect Effects for Hypotheses 2 and 3^{a,b}

Indirect Effect	Hypothesis 2: Mediating Role of PTSD Symptoms			Hypothesis 3: Mediating Role of Posttraumatic Cognitions		
	<i>B</i>	<i>SE</i>	95% CI	<i>B</i>	<i>SE</i>	95% CI
Total indirect effects						
Past IPT → FU IPT	0.28	0.11	[0.08, 0.51] ^c	0.36	0.13	[0.11, 0.64] ^c
Past non-IPT → FU IPT	0.14	0.09	[0.01, 0.38] ^c	0.20	0.13	[-0.02, 0.49]
Specific indirect effects						
Past IPT → PTSD → FU IPT	0.28	0.11	[0.08, 0.51] ^c	0.14	0.13	[-0.11, 0.39]
Past IPT → SWJ → FU IPT				0.01	0.10	[-0.18, 0.20]
Past IPT → RTO → FU IPT				-0.06	0.07	[-0.23, 0.06]
Past IPT → TH → FU IPT				0.27	0.13	[0.05, 0.55] ^c
Past non-IPT → PTSD → FU IPT	0.14	0.09	[0.01, 0.38] ^c	0.07	0.08	[-0.03, 0.30]
Past non-IPT → SWJ → FU IPT				0.01	0.05	[-0.08, 0.14]
Past non-IPT → RTO → FU IPT				-0.03	0.06	[-0.26, 0.04]
Past non-IPT → TH → FU IPT				0.15	0.11	[-0.01, 0.46]

Note. IPT = interpersonal trauma; non-IPT = noninterpersonal trauma; FU = follow-up; PTSD = posttraumatic stress disorder; SWJ = Self-Worth and Judgment; RTO = Reliability and Trustworthiness of Others; TH = Threat of Harm.

^aHypothesis 2 posed that interpersonal and noninterpersonal trauma would increase risk for subsequent interpersonal trauma via PTSD symptoms. Hypothesis 3 posed that interpersonal and noninterpersonal trauma would increase risk for subsequent interpersonal trauma via maladaptive posttraumatic cognitions, above and beyond the influence of PTSD symptoms.

^bDirect effects, covariances, intercepts, thresholds, and residual variances can be seen in Supplementary Table 1.

^c95% confidence intervals do not contain zero and therefore signify an effect that is present.