

Acute Stress Symptoms in Seriously Injured Patients: Precipitating Versus Cumulative Trauma and the Contribution of Peritraumatic Distress

John Briere,¹ Colin P. Dias,¹ Randy J. Semple,¹ Catherine Scott,¹ Noémie Bigras,² and Natacha Godbout³

¹Department of Psychiatry and Behavioral Sciences, Keck School of Medicine, University of Southern California, Los Angeles, California, USA

²Département de Psychologie, Université du Québec à Montréal, Canada

³Département de Sexologie, Université du Québec à Montréal, Canada

The relationship between type of trauma exposure, cumulative trauma, peritraumatic distress, and subsequent acute stress disorder (ASD) symptoms was examined prospectively in 96 individuals presenting with acute medical injuries to a Level 1 emergency/trauma department. Common precipitating traumas included motor vehicle-related events, stabbings, shootings, and physical assaults. At 2 to 3 weeks follow-up, 22.9% of participants had developed ASD. Univariate analysis revealed no relationship between type of precipitating trauma and ASD symptoms, whereas robust path analysis indicated direct effects of gender, lifetime cumulative trauma exposure, and peritraumatic distress. Peritraumatic distress did not mediate the association between cumulative trauma and symptoms, but did mediate the association between gender and symptomatology. These results, which account for 23.1% of the variance in ASD symptoms, suggest that ASD may be more due to cumulative trauma exposure than the nature of the precipitating trauma, but that cumulative trauma does not exert its primary effect by increasing peritraumatic distress to the most recent trauma.

Several decades of research indicate that traumatic events such as war, disasters, assaults, and serious accidents can have a range of enduring psychological impacts (American Psychiatric Association [APA], 2013; Briere, 2004; Norris, 1992). The most studied outcome is posttraumatic stress disorder (PTSD), which is composed of a characteristic group of symptoms that have lasted more than a month since the traumatic event. Less

investigated has been acute stress disorder (ASD), even though it is relatively common among trauma-exposed persons presenting for emergency care, ranging between 7% and 59% in various studies (Brewin, Andrews, Rose, & Kirk, 1999; Bryant, 2011), and is beyond its association with PTSD (Bryant, 2011; Bryant et al., 2015), associated with its own significant psychological distress, disability, and risks of suicidality and substance abuse (APA, 2013; Bryant, 2011).

The diagnosis of ASD first appeared in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; APA, 1994), and consisted of dissociative, intrusive, avoidant, and hyperarousal-related psychological reactions experienced within a month of exposure to a Criterion A stressor. Although the *DSM-IV* required dissociative symptoms for the diagnosis, the *DSM-5* (APA, 2013) has no requirement that any given symptom cluster be present, including dissociation—it is only necessary that nine or more symptoms be reported. There are fewer studies of ASD or ASD-related symptoms than of PTSD, although several risk factors have been identified. These include reduced social support (Yaşan, Güzel, Tamam, & Ozkan, 2009), fear of death (Chiu, deRoon-Cassini, & Brasel, 2011; Kjær Fuglsang, Moergeli, Hepp-Beg, & Schnyder, 2002), and peritraumatic and/or persistent dissociation (Panasetis & Bryant, 2003; Yaşan et al., 2009).

One obvious variable in the prediction of ASD is the type of trauma associated with the disorder, although most risk studies

This research was supported by the Southern California Clinical and Translational Science Institute (SC CTSI; National Institutes of Health [NIH]/National Center for Research Resources/National Center for Advancing Translational Sciences (5 UL1 RR031986)). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH. Dr. Briere receives royalties from Psychological Assessment Resources for the Detailed Assessment of Posttraumatic Stress (DAPS) instrument used in this study. The authors wish to thank the following individuals who served as research assistants or associates in this study: Dimitri Bacos, Jennifer Ben-Ami, Sarah Dillon, Emily Frank, Brittany Garrett, Jordan Hoffman, Ashley Huggins, Chris James, Melinda Kuoch, Rebecca Lee, Laila A. Madni, Nina Polyne, Brittany Reid, Katherine Roahrig, Tristan Sguigna, Saemy Son, Mario A. Souza, Melissa Packer West, Patrick Wiita, Parthena Denise Zapata, and Manuel Zetino.

Correspondence concerning this article should be addressed to John Briere, Department of Psychiatry and Behavioral Sciences, University of Southern California, 2250 Alcazar Street, CSC, Suite 2200, Los Angeles, CA 90033. E-mail: jbriere@usc.edu

Copyright © 2017 International Society for Traumatic Stress Studies. View this article online at wileyonlinelibrary.com
DOI: 10.1002/jts.22200

examine PTSD. In this regard, the PTSD literature indicates that interpersonal events (e.g., assaults) are more likely to produce posttraumatic stress than noninterpersonal events (e.g., disasters; Breslau et al., 1998; Briere, Hodges, & Godbout, 2010), and that sexual victimization tends to be more traumatic than nonsexual assaults (Kilpatrick, Edmonds, & Seymour, 1992).

Other research, however, suggests that the level of emotional distress (e.g., anxiety, depression) immediately arising from a trauma may be at least as predictive of later stress responses as the nature of the trauma itself (Thomas, Saumier, & Brunet, 2012). For example, measures of peritraumatic distress have been associated with an increased likelihood of posttraumatic stress in a number of studies (Briere, Scott, & Weathers, 2005; McCaslin et al., 2008). In most studies, however, peritraumatic distress was evaluated retrospectively and cross-sectionally, typically months or years after the index traumatic event (Thomas et al., 2012); a scenario that may introduce memory distortion and other confounding variables. In those cases where peritraumatic distress was assessed soon after the trauma (e.g., Jehel, Paterniti, Brunet, Louville, & Guelfi, 2006), the evaluated outcome was almost always PTSD. In one exception, Vaiva et al. (2003) found that “peritraumatic fright,” broadly defined (e.g., “including stopped thoughts, emptiness, and total psychological inhibition,” p. 397) was associated with a subsequent ASD diagnosis.

Notably, physical pain associated with a trauma also has been linked to PTSD (e.g., Gros, Szafranski, Brady, & Back, 2015; Norman, Stein, Dimsdale, & Hoyt, 2008). However, this variable may differ from peritraumatic emotional distress in significant ways, and it is not clear to what extent this variable represents a characteristic of the trauma (e.g., its severity) as opposed to a subjective peritraumatic response to it.

An additional factor in the development of a stress disorder is the individual’s exposure to past traumas. Recent research indicates that those with multiple types of prior trauma exposure, referred to as cumulative trauma (CT; Briere, Godbout, & Dias, 2015; Briere, Kaltman, & Green, 2008; Follette, Polusny, Bechtle, & Naugle, 1996), are more likely to experience posttraumatic stress in response to a current stressor than those with a single trauma exposure. For example, a history of prior motor vehicle accidents appears to be associated with ASD in response to a current motor vehicle accident (Bryant & Harvey, 2000; Harvey & Bryant, 2000), and those with prior traumas have been found to have increased risk for PTSD following a new trauma (Briere, Agee, & Dietrich, 2016; Kilpatrick et al., 2013).

Such results suggest that the emergence of ASD or PTSD, in some cases, may reflect the cumulative effects of multiple traumas over time, with the most proximal event serving as a “tipping point” for the development of a stress disorder (Briere & Scott, 2015a). The mechanism of this effect is unknown, although it is possible that previous trauma exposures produce subclinical symptoms that accumulate over the long-term, eventually summing to meet criteria for a stress disorder.

Alternatively, or in addition, CT may neurobiologically and/or psychologically “sensitize” the trauma survivor over time (Smith, Katz, Charney, & Southwick, 2007), leading to heightened peritraumatic distress following a new trauma, thereby increasing the likelihood of a stress disorder.

In the current study we sought to explore the prospective relationship between precipitating trauma, CT, peritraumatic distress, and ASD symptoms in an urban emergency room setting. As part of a larger, in-progress study of genetic and psychological predictors of ASD and PTSD, participants were evaluated upon admission for demographics, type of precipitating trauma, and self-reported peritraumatic distress and peritraumatic pain, and then followed-up 2 to 3 weeks later for assessment of ASD symptoms. Based on the literature, we hypothesized that the type of precipitating trauma would not be related to ASD, whereas CT would be a significant predictor. We further predicted that peritraumatic distress would mediate the relationship between CT and ASD symptoms (the sensitization hypothesis), but also that CT, irrespective of peritraumatic distress, would directly lead to ASD symptoms (the tipping point hypothesis). We did not specifically hypothesize the role of peritraumatic pain, although it was possible that it too might mediate the CT–ASD symptom relationship.

Method

Participants

Of the 203 acutely injured individuals entered into this study at admission, 96 (47.3%) returned for follow-up evaluation 2 to 3 weeks after the initial interview. Univariate chi-squares and correlational analyses, as appropriate, revealed no relationship between those who did not follow-up versus those who did, in terms of age ($p = .512$), gender ($p = .315$), race ($p = .294$), type of precipitating trauma exposure ($p = .899$), peritraumatic distress ($p = .165$), or CT ($p = .697$).

In the follow-up group, 21 participants (21.9%) were female and 75 (78.1%) were male; the mean age was 32.85 years ($SD = 13.31$). Racial breakdown was 68.8% ($n = 66$) Hispanic/Latino, 10.4% ($n = 10$) non-Hispanic Caucasian, 9.4% ($n = 9$) non-Hispanic Black/African American, and 11.4% ($n = 11$) other or more than one race. There were 62.5% of participants ($n = 60$) employed part- or full time. Including the precipitating event, participants reported a mean of 3.10 different types of *DSM-IV* Criterion A-level trauma in their lives ($SD = 2.50$). Some head injury was reported by 27.1% of the sample, 39.1% reported loss of consciousness, and the mean level of pain reported at bedside interview was 6.2 on a scale of 1 to 10.

Precipitating traumas varied in this sample, as indicated in Table 1, with the most frequent involving some form of motor vehicle-related trauma, and assaults with knives or guns. When summed into larger categories, 71.9% were exposed to motor vehicle accidents, 22.9% were physically assaulted, and 5.2% experienced some other trauma.

Table 1
Type and Frequency of Presenting Traumas in Acutely Injured Emergency Room Patients (N = 96)

Trauma category	Type of trauma	Frequency	%
Motor vehicle-related	Motor vehicle accident	30	31.3
	Auto vs. pedestrian	10	10.4
	Motorcycle	8	8.3
	Fell out of moving vehicle	1	1.0
	Auto vs. bicycle	11	11.5
	Motorcycle vs. auto	9	9.4
	Total	69	71.9
Assaults	Shot with gun	8	8.3
	Stabbed	9	9.4
	Beating or other physical assault	5	5.2
	Total	22	22.9
Other	Fall	4	4.2
	Other	1	1.0
	Total	5	5.2

Procedure

This study was conducted at the Los Angeles County + University of Southern California (LAC+USC) Medical Center, Department of Emergency Medicine, using a protocol reviewed and approved by the Institutional Review Board of the University of Southern California, Los Angeles, CA. Candidates for this study were identified through the Department of Emergency Medicine's real-time computerized tracking and reporting program (Wellsoft EDIS v.11) as they presented for emergency stabilization and treatment of acute medical trauma. Eligible participants were adults (18 years of age and older); had sustained moderate to severe trauma but had full consciousness, as determined by a score of 6 to 8 on the Revised Trauma Score (Champion et al., 1989); were able to speak and read English; had postdischarge access to a telephone; and were able to provide informed consent. Because sexual trauma victims are taken or referred to a specialized center within the LAC+USC system, there were no participants in this sample with sexual assault as a precipitating trauma. Patients were excluded if they reported symptoms indicative of psychosis or mania; were pregnant, a prisoner, or legal detainee; or were intoxicated or suffering other cognitive impairment that precluded informed consent. All participants who were not excluded, met inclusion criteria, and consented to participate were sequentially added to the sample.

Following initial treatment, and once medically stable, potential participants were approached at bedside by a member of the research team. Per the institutional review board-approved protocol, and after receiving an explanation of the study procedures, they were verbally administered a brief mental

health screen and, if not excluded and willing to participate, signed a university and medical center-approved informed consent form for the study. The mean time from the onset of the precipitating trauma to the research interview in this study was 19.3 hours ($SD = 13.5$).

Upon entry into the study, participants provided basic demographic information, and Wellsoft EDIS data were consulted for information on the trauma that led to their current admission. These traumas are presented in Table 1. Also collected at this time were reports of peritraumatic distress and pain associated with the trauma, based on the Acute Trauma Interview (ATI; Briere, 2011). Participants were then scheduled for a 2- to 3-week posttrauma follow-up visit. The mean number of weeks to follow-up was 2.7 (approximately 19 days) for the 90 participants with complete data on this variable (Time 2 contact date information was missing for six participants due to a coding error). At this follow-up, participants completed, among other instruments, the Detailed Assessment of Posttraumatic Stress (DAPS; Briere, 2001), after which a trained interviewer administered the Acute Stress Disorder Interview (ASDI; Bryant, Harvey, Dang, & Sackville, 1998). Participants were compensated \$25 for their time, both at recruitment and follow-up.

Measures

Demographics and mental health screening. A demographic information sheet and a brief mental health screening instrument were developed for this study. Sample mental health screening questions included, "Have you ever had a time when you heard voices when no one was actually present, had visions, or saw things that other people could not see?" and "Have you ever had a time that lasted 3 days or more when you needed much less sleep than usual (or no sleep at all) without feeling tired, or even feeling more energetic than usual?"

Detailed Assessment of Posttraumatic Stress. The DAPS (Briere, 2001) is a 105-item standardized self-report inventory that is commonly employed as a measure of posttraumatic stress and comorbidities in research and clinical practice (Briere et al., 2005; Elhai, Gray, Kashdan, & Franklin, 2005). The DAPS includes a Relative Trauma Exposure (RTE) score, consisting of a count of the number of different types of Criterion A-level trauma exposures (e.g., disasters, sexual and physical assaults, accidents) the individual has experienced over his or her life span, ranging from 0 to 11.

The RTE score was used to represent CT in the present study, as it has in other research (e.g., Bigras, Daspe, Godbout, Briere, & Sabourin, 2016; Briere et al., 2016). This variable is a form of cumulative risk (CR; see Appleyard, Egeland, Dulmen, & Sroufe, 2005) that is widely used in epidemiological research. As noted by Evans, Li, and Whipple (2013), CR variables are "statistically sensitive even with small samples, and make no assumptions about the relative strengths of multiple risk factors

or their collinearity. CR also fits well with underlying theoretical models . . . concerning why multiple risk factor exposure is more harmful than singular risk exposure” (p. 1). Notably, a CT index does not require quantification of the various characteristics associated with each type of trauma exposure, and avoids measurement error associated with the analysis of highly correlated variables and masked between-characteristic and/or between-event interaction effects (Evans et al., 2013).

Acute Trauma Interview. The Acute Trauma Interview (ATI; Briere, 2011) was used to record the details of the precipitating trauma at study intake, and includes the number of hours since trauma, current psychological concerns, and current pain rating (measured after medical stabilization and acute medication), as well as participants’ reports of peritraumatic distress in response to the precipitating trauma. The 8-item ATI Peritraumatic Distress scale directly replicates the items of the DAPS Peritraumatic Distress scale, and evaluates reports of eight different peritraumatic experiences, including fear, helplessness, guilt, shame and humiliation, horror, on a 0 (*none*) to 5 (*very much*) scale. In the current sample, Cronbach’s α for the ATI Peritraumatic Distress scale was .77.

Acute Stress Disorder Interview. The Acute Stress Disorder Interview (ASDI; Bryant, Harvey, Dang, & Sackville, 1998) is a 19-item, dichotomously scored clinical interview that is criterion-keyed to *DSM-IV* ASD. It has been shown in a number of studies to be a reliable and valid measure of ASD (Briere, 2004). In the current sample, Cronbach’s α for the ASDI total score was .86.

Because of changes to the ASD diagnosis in *DSM-5*, and the lack of validated *DSM-5* ASD interview measures at the time of the study, we employed classic ASDI scoring to determine the presence or absence of *DSM-IV* ASD, but then utilized the total ASDI score as a better approximation of *DSM-5* ASD in the statistical analyses. As noted, *DSM-5* criteria require the presence of 9 or more of 14 symptoms from any of five categories: intrusion, negative mood, dissociation, avoidance, and arousal. Similarly, the total ASDI score does not specifically prize dissociation over other ASD symptoms, and treats ASD symptoms as equally weighted components of a continuously measured construct.

Data Analysis

Missing values. There were a small number of missing values among predictor variables in the follow-up group: two (2.1%) for the cumulative trauma variable, two (2.1%) for race, and four (4.2%) for the Peritraumatic Distress scale. In each case, the SPSS 23 linear interpolation algorithm was used to replace these values. Linear interpolation is considered to be an effective and unbiased method of missing data replacement, especially at the low missing data rate found in this study (Tabachnick & Fidell, 2007).

Table 2

Total Acute Stress Disorder Interview (ASDI) Means and Standard Deviations According to Gender, Race, and Trauma Category (N = 96)

Variable	<i>n</i>	<i>M</i>	<i>SD</i>
Gender			
Male	75	6.37	4.37
Female	21	10.05	5.26
Race			
Caucasian	10	7.80	5.63
Black/African American	9	8.00	6.20
Latino	66	7.27	4.61
Other/Mixed	11	5.36	4.06
Trauma category			
MVR	40	7.75	5.10
Assault	51	6.92	4.72
Other	5	5.20	2.28

Note. MVR = motor vehicle-related.

Path analysis. Path analysis is a statistical technique that allows testing both direct and indirect relationships among different variables that may be correlated (Kline, 2011). This procedure was conducted using *Mplus* v.7 (Muthén & Muthén, 1998–2015), employing maximum likelihood with robust standard errors (MLR) to address nonnormality. As recommended by McDonald and Ringo Ho (2002), overall model fit was tested by considering the comparative fit index (CFI), the Tucker-Lewis Index (TLI), the root mean square error of approximation (RMSEA), and the chi-square statistic. A nonstatistically significant chi-square value, a CFI value of .90 or higher, a TLI value of .95 or higher, and a RMSEA value below .06 are indicators that the observed data are a good fit to the hypotheses in question. Examination of the magnitude and significance of the indirect effects was performed using the bootstrap confidence intervals method (MacKinnon & Fairchild, 2009) with *Mplus* (Muthén & Muthén, 1998–2015). This bias-corrected method is based on a distribution for the product of coefficients and generates confidence limits for the true value of the coefficient for indirect effects. When zero is not in the confidence interval, the indirect effect is considered significant (Preacher & Hayes, 2004).

Results

Of the 96 participants, 22 (22.9%) reported symptoms on the ASDI that met criteria for *DSM-IV* ASD. Univariate analyses revealed that total ASDI scores were associated with gender, $t(94) = 3.26, p = .002$; CT, $r = .30, p = .003$; and peritraumatic distress, $r = .37, p < .001$; but were not related to age, $r = .02, nonsignificant (ns)$; race, $F(3, 92) = 1.37, ns$; or type of precipitating trauma, $F(2, 93) = 0.78, ns$. See Table 2 for relevant means and standard deviations. Other trauma or impact variables that might affect total ASDI scores were not correlated

PREDICTORS OF ACUTE STRESS DISORDER

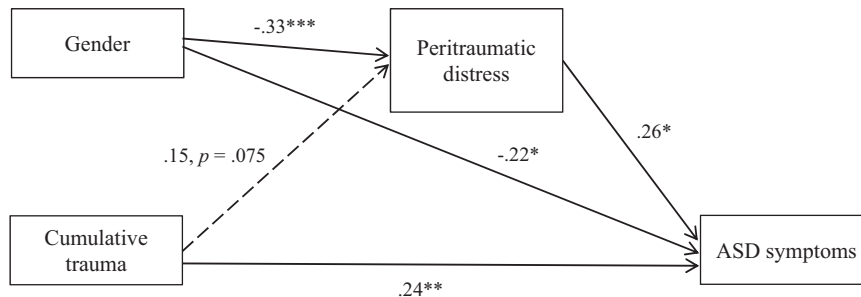


Figure 1. Path analyses of the effects of cumulative trauma, gender, and peritraumatic distress on acute stress disorder (ASD) symptoms. Values are standardized β coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

with the ASDI: head injury, $r = -.13$, *ns*; loss of consciousness, $r = .06$, *ns*; self-reported level of pain at the time of interview, $r = .19$, *ns*; and number of hours since the precipitating trauma, $r = -.12$, *ns*. As well, head injury and loss of consciousness were uncorrelated with peritraumatic distress scores, $r = -.04$, *ns* and $r = .03$, *ns*, respectively. Inspection of the predictor correlation matrix revealed no evidence of multicollinearity, with the highest correlation being between female gender and peritraumatic distress, $r = .32$.

Path analysis. The link from CT to ASD symptoms was tested with path analysis, and indicated a direct association, $\beta = .29$, $p = .003$. Following this significant relationship, the mediation model depicted in Figure 1 was examined, and found to be a good representation of the data, $\chi^2(1) = 0.23$, $p = .631$; RMSEA = .00, with 90% CI [0.00, 0.21]; CFI = 1.0, TLI = 1.11. After the inclusion of gender and peritraumatic distress, the direct association between CT and ASD symptoms decreased to $\beta = .24$, $p = .011$, suggesting partial mediation. There was no significant association between CT and peritraumatic distress in this model ($p = .075$), whereas gender, CT, and peritraumatic distress were each directly associated with ASD symptomatology. Results indicated a significant indirect effect of gender on ASD symptoms through peritraumatic distress, $\beta = -.09$, 95% CI [-0.20, -0.02], but no indirect effect of CT on ASD symptoms through peritraumatic distress, $\beta = .04$, 95% CI [-0.001, 0.11]. This integrative model explained 12.9% of the variance in peritraumatic distress and 23.1% of the variance in ASD symptoms.

In order to examine the specific effect of gender on CT, we amended this model by adding a correlation between these two variables. Although the subsequent model was just-identified, and thus precluded the determination of fit indices, there was no significant association between gender and CT, $\beta = -.05$, $p = .629$.

Because peritraumatic pain has been linked to subsequent PTSD, we tested an alternate path-analytic model in which pain at the time of interview was added as a second mediator, along with peritraumatic distress. However, there was no relationship

between peritraumatic pain and other variables in this model, including ASD symptoms. Fit indices remained similar to the first model, $\chi^2(1) = 0.23$, $p = .631$; RMSEA = .00, with 90% CI [0.00, 0.21]; CFI = 1.0, TLI = 1.20. Because peritraumatic pain was not a significant predictor, the original model was retained.

Discussion

This study suggests that the relationship between the precipitating trauma and subsequent ASD symptoms may be less a function of the nature of the trauma than gender, level of acutely reported peritraumatic distress, and cumulative trauma exposure. We did not find that CT exerts its primary effects on ASD symptomatology by increasing peritraumatic distress in response to the most recent trauma, as would be predicted by the sensitization hypothesis. Instead, CT and peritraumatic distress were independent risk factors for acute posttraumatic stress. Notably, although the effect of CT on acutely reported peritraumatic distress was nonsignificant in the present study ($p = .075$), it is possible that a larger sample would identify a significant contribution of CT-related peritraumatic distress to ASD symptoms, thereby providing potential support for the sensitization hypothesis.

These findings do not suggest a significant role of peritraumatic pain in the development of ASD. Further research is indicated, however, including the possibility that this variable is more predictive of PTSD than ASD symptomatology. The role of pain medication also should be examined, as participants in the present study were evaluated for peritraumatic pain after initial medical treatment, which may have blunted any pain-ASD relationship.

It should be noted that the current sample did not include participants with acute sexual trauma. Because sexual victimization is more likely to lead to PTSD (and thus, presumably, ASD) than most other traumas, the absence of these individuals in the present study may have reduced the relationship between trauma type and ASD. However, at least one investigation that included sexual trauma survivors still found cumulative trauma

to be a better predictor of traumatic stress than any specific precipitating event (Briere et al., 2016).

In the absence of a CT-peritraumatic distress effect, the mechanism whereby CT leads to acute stress remains unknown. One possibility is peritraumatic dissociation, which has been linked to subsequent posttraumatic stress (Lensvelt-Mulders et al., 2008). Given the complexity of controlling for a variable that is implicit in the variable being predicted, the current study did not prospectively evaluate the role of peritraumatic dissociation in ASD. Yet, it is possible that this variable mediates the CT–ASD symptom relationship in undetermined ways. In any event, the direct relationship between CT and ASD symptoms supports the cumulative trauma hypothesis that the effects of prior traumas may accumulate over time, at some point leading to clinical (“tipping point”) levels of acute stress.

Female gender was associated with ASD symptoms in the present study, both directly and through its relationship to peritraumatic distress. This is consistent with other findings that women report both more peritraumatic (Lilly, Pole, Best, Metzler, & Marmar, 2009) and posttraumatic stress (Bryant & Harvey, 2003) relative to men. The reason for this effect is unknown, but may include women’s sex role socialization to more easily report psychological distress (Lilly et al., 2009), as well as the greater likelihood of women experiencing traumas most associated with posttraumatic stress, especially sexual victimization (Breslau et al., 1998). Because there was an independent effect of gender on ASD, controlling for peritraumatic distress, however, these differences cannot be entirely explained by a tendency for women to report greater emotional distress than men in response to a given trauma.

The diagnosis of ASD in this study was based on a gold standard measure, the Acute Stress Disorder Interview, using *DSM-IV* criteria. Even though *DSM-IV* and *DSM-5* ASD diagnoses share virtually the same symptom set, the latter criteria include, but do not specifically require, dissociative symptoms. We cannot estimate the extent to which the current findings, which used the total ASDI score as a proxy for *DSM-5* ASD, are entirely relevant to the *DSM-5* version of ASD. Future research in this area is indicated, once validated *DSM-5* ASD measures become available.

Notably, the dropout rate from entry into the study to ASD assessment was 53%, even though all participants received phone calls reminding them of their follow-up session. As a result, these findings may be less generalizable to those who did not return in 2 to 3 weeks, even though we found no study variable that predicted dropout, and thus no source of systematic bias. Some of this attrition may reflect posttraumatic avoidance, given the stressful, often severe nature of the traumas involved, and the high stimulation level and potentially frightening context of the large urban emergency/trauma department in which the participants were initially treated.

Finally, we could not evaluate participants for trauma symptoms that they might have had at admission that were not due to the precipitating trauma. Thus, per the cumulative trauma hypothesis, it is possible that some participants were

additionally suffering from posttraumatic stress related to one or more prior traumas upon entry to the emergency service. Future researchers might have participants describe any posttraumatic symptoms they believe were present prior to the precipitating event, or might study at-risk individuals prospectively, before and after some proportion experience a traumatic event.

The current results support the notion that ASD can be a multitrauma outcome, reflecting the cumulative effects of various traumas, such that the additional stress associated with the precipitating trauma may at some point exceed a risk threshold (Karam et al., 2014) and lead to the development of a stress disorder. This is in contrast to the *DSM-IV* requirement that ASD be linked to a single traumatic event. It is, however, in agreement with *DSM-5* Criterion A, which newly allows more than one trauma to be implicated in the development of a stress disorder (APA, 2013, pp. 271–272; Briere & Scott, 2015b). The apparent importance of cumulative trauma over time in the genesis of ASD supports the validity of assessing the effects of more distal traumas when evaluating acute stress. Further, as suggested by some (e.g., Briere & Scott, 2015b; Courtois & Ford, 2013), interventions for acute trauma-related distress may be most helpful when they include processing memories of prior traumas as well as current ones. As well, if past traumas contribute persisting emotional distress to the effects of the precipitating trauma, interventions that increase affect regulation or emotional tolerance skills may help the survivor to downregulate these chronic trauma-related responses, thereby potentially reducing the likelihood or intensity of current ASD symptoms. Future researchers should examine the potential effectiveness of these additional treatment approaches, especially to the extent that they can augment current approaches to ASD and increase treatment efficacy.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Appleyard, K., Egeland, B., Dulmen, M. H. M., & Sroufe, A. L. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry*, *46*, 235–245. <https://doi.org/10.1111/j.1469-7610.2004.00351.x>
- Bigras, N., Daspe, M.-È., Godbout, N., Briere, J., & Sabourin, S. (2016). Cumulative childhood trauma and adult sexual satisfaction: Mediation by affect dysregulation and sexual anxiety in men and women. *Journal of Sex and Marital Therapy*, *43*, 377–396. <https://doi.org/10.1080/0092623X.2016.1176609>
- Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry*, *55*, 626–632. <https://doi.org/10.1001/archpsyc.55.7.626>
- Brewin, C. R., Andrews, B., Rose, S., & Kirk, M. (1999). Acute stress disorder and posttraumatic stress disorder in victims of violent crime. *American Journal of Psychiatry*, *156*, 360–366. <https://doi.org/10.1176/ajp.156.3.360>

- Briere, J. (2001). *Detailed Assessment of Posttraumatic Stress (DAPS)*. Odessa, FL: Psychological Assessment Resources.
- Briere, J. (2004). *Psychological assessment of adult posttraumatic states: Phenomenology, diagnosis, and measurement* (2nd ed.). Washington, DC: American Psychological Association.
- Briere, J. (2011). *Acute Trauma Interview*. Unpublished assessment interview, University of Southern California, Los Angeles, CA.
- Briere, J., Agee, E., & Dietrich, A. (2016). Cumulative trauma and current PTSD status in general population and inmate samples. *Psychological Trauma: Theory, Research, Practice and Policy*, 8, 439–446. <https://doi.org/10.1037/tra0000107>
- Briere, J., Godbout, N., & Dias, C. (2015). Cumulative trauma, hyperarousal, and suicidality in the general population: A path analysis. *Journal of Trauma and Dissociation*, 16, 1–17. <https://doi.org/10.1080/15299732.2014.970265>
- Briere, J., Hodges, M., & Godbout, N. (2010). Traumatic stress, affect dysregulation, and dysfunctional avoidance: A structural equation model. *Journal of Traumatic Stress*, 23, 767–774. <https://doi.org/10.1002/jts.20578>
- Briere, J., Kaltman, S., & Green, B. L. (2008). Accumulated childhood trauma and symptom complexity. *Journal of Traumatic Stress*, 21, 223–226. <https://doi.org/10.1002/jts.20317>
- Briere, J., & Scott, C. (2015a). Complex trauma in adolescents and adults: Effects and treatment. *Psychiatric Clinics of North America*, 38, 515–527. <https://doi.org/10.1016/j.psc.2015.05.004>
- Briere, J., & Scott, C. (2015b). *Principles of trauma therapy: A guide to symptoms, evaluation, and treatment* (2nd ed.; DSM-5 update). Thousand Oaks, CA: Sage.
- Briere, J., Scott, C., & Weathers, F. (2005). Peritraumatic and persistent dissociation in the presumed etiology of PTSD. *American Journal of Psychiatry*, 162, 2295–2301. <https://doi.org/10.1176/appi.ajp.162.12.2295>
- Bryant, R. A. (2011). Acute stress disorder as a predictor of posttraumatic stress disorder: A systematic review. *Journal of Clinical Psychiatry*, 72, 233–239. <https://doi.org/10.4088/JCP.09r05072blu>
- Bryant, R. A., Creamer, M., O'Donnell, M., Silove, D., McFarlane, A. C., & Forbes, D. (2015). A comparison of the capacity of DSM-IV and DSM-5 acute stress disorder definitions to predict posttraumatic stress disorder and related disorders. *Journal of Clinical Psychiatry*, 76, 391–397. <https://doi.org/10.4088/JCP.13m08731>
- Bryant, R. A., & Harvey, A. G. (2000). New DSM-IV diagnosis of acute stress disorder. *American Journal of Psychiatry*, 157, 1889–1890. <https://doi.org/10.1176/appi.ajp.157.11.1889>
- Bryant, R. A., & Harvey, A. G. (2003). Gender differences in the relationship between acute stress disorder and posttraumatic stress disorder following motor vehicle accidents. *Australian and New Zealand Journal of Psychiatry*, 37, 226–229. <https://doi.org/10.1046/j.1440-1614.2003.01130.x>
- Bryant, R. A., Harvey, A. G., Dang, S. T., & Sackville, T. (1998). Assessing acute stress disorder: Psychometric properties of a structured clinical interview. *Psychological Assessment*, 10, 215–220. <https://doi.org/10.1037/1040-3590.10.3.215>
- Champion, H. R., Sacco, W. J., Copes, W. S., Gann, D. S., Gennarelli, T. A., & Flanagan, M. E. (1989). A Revision of the Trauma Score. *Journal of Trauma: Injury, Infection, and Critical Care*, 29, 623–629. <https://doi.org/10.1097/00005373-198905000-00017>
- Chiu, K. B., deRoon-Cassini, T. A., & Brasel, K. J. (2011). Factors identifying risk for psychological distress in the civilian trauma population. *Academic Emergency Medicine*, 18, 1156–1160. <https://doi.org/10.1111/j.1553-2712.2011.01206.x>
- Courtois, C. A., & Ford, J. D. (2013). *Treatment of complex trauma: A sequenced, relationship-based approach*. New York, NY: Guilford Press.
- Elhai, J. D., Gray, M. J., Kashdan, T. B., & Franklin, C. L. (2005). Which instruments are most commonly used to assess traumatic event exposure and posttraumatic effects?: A survey of traumatic stress professionals. *Journal of Traumatic Stress*, 18, 541–545. <https://doi.org/10.1002/jts.20062>
- Evans, G. W., Li, D., & Whipple, S. S. (2013). Cumulative risk and child development. *Psychological Bulletin*, 139, 1342–1396. <https://doi.org/10.1037/a0031808>
- Follette, V. M., Polusny, M. A., Bechtle, A. E., & Naugle, A. E. (1996). Cumulative trauma: The impact of child sexual abuse, adult sexual assault, and spouse abuse. *Journal of Traumatic Stress*, 9, 25–35. <https://doi.org/10.1007/BF02116831>
- Gros, D. F., Szafranski, D. D., Brady, K. T., & Back, S. E. (2015). Relations between pain, PTSD symptoms, and substance use in veterans. *Psychiatry*, 78, 277–287. <https://doi.org/10.1080/00332747.2015.1069659>
- Harvey, A. G., & Bryant, R. A. (2000). Two-year prospective evaluation of the relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *American Journal of Psychiatry*, 157, 626–628. <https://doi.org/10.1176/appi.ajp.157.4.626>
- Jehel, L., Paterniti, S., Brunet, A., Louville, P., & Guelfi, J. D. (2006). Peritraumatic distress prospectively predicts PTSD symptoms in assault victims. *L'Encéphale*, 32, 953–956. [https://doi.org/10.1016/S0013-7006\(06\)76272-8](https://doi.org/10.1016/S0013-7006(06)76272-8)
- Karam, E. G., Friedman, M. J., Hill, E. D., Kessler, R. C., McLaughlin, K. A., Petukhova, M., . . . Koenen, K. C. (2014). Cumulative traumas and risk thresholds: 12-month PTSD in the World Mental Health (WMH) surveys. *Depression and Anxiety*, 31, 130–142. <https://doi.org/10.1002/da.22169>
- Kilpatrick, D. G., Edmonds, C. N., & Seymour, A. K. (1992). *Rape in America: A report to the nation*. Arlington, VA: National Victims Center.
- Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. *Journal of Traumatic Stress*, 26, 537–547. <https://doi.org/10.1002/jts.21848>
- Kjær Fuglsang, A., Moergeli, H., Hepp-Beg, S., & Schnyder, U. (2002). Who develops acute stress disorder after accidental injuries? *Psychotherapy and Psychosomatics*, 71, 214–222. <https://doi.org/10.1159/000063647>
- Kline, R. B. (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: Guilford Press.
- Lensvelt-Mulders, G., van der Hart, O., van Ochten, J. M., van Son, M. J. M., Steele, K., & Breeman, L. (2008). Relations among peritraumatic dissociation and posttraumatic stress: A meta-analysis. *Clinical Psychology Review*, 28, 1138–1151. <https://doi.org/10.1016/j.cpr.2008.03.006>
- Lilly, M. M., Pole, N., Best, S. R., Metzler, T., & Marmar, C. R. (2009). Gender and PTSD: What can we learn from female police officers? *Journal of Anxiety Disorders*, 23, 767–774. <https://doi.org/10.1016/j.janxdis.2009.02.015>
- MacKinnon, D. P., & Fairchild, A. J. (2009). Current directions in mediation analysis. *Current Directions in Psychological Science*, 18, 16–20. <https://doi.org/10.1111/j.1467-8721.2009.01598.x>
- McCaslin, S. E., Inslicht, S. S., Metzler, T. J., Henn-Haase, C., Maguen, S., Neylan, T. C., . . . Marmar, C. R. (2008). Trait dissociation predicts posttraumatic stress disorder symptoms in a prospective study of urban police officers. *Journal of Nervous and Mental Disease*, 196, 912–918. <https://doi.org/10.1097/NMD.0b013e31818ec95d>
- McDonald, R. P., & Ringo Ho, M.-H. (2002). Principles and practice in reporting structural equation analyses. *Psychological Methods*, 7, 64–82. <https://doi.org/10.1037/1082-989X.7.1.64>

- Muthén, L. K., & Muthén, B. O. (1998–2015). *Mplus user's guide* (7th ed.). Los Angeles, CA: Author.
- Norman, S. B., Stein, M. B., Dimsdale, J. E., & Hoyt, D. B. (2008). Pain in the aftermath of trauma is a risk factor for post-traumatic stress disorder. *Acute Pain, 10*, 103–103. <https://doi.org/10.1016/j.acpain.2008.05.030>
- Norris, F. H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology, 60*, 409–418. <https://doi.org/10.1037/0022-006X.60.3.409>
- Panasetis, P., & Bryant, R. A. (2003). Peritraumatic versus persistent dissociation in acute stress disorder. *Journal of Traumatic Stress, 16*, 563–566. <https://doi.org/10.1023/B:JOTS.0000004079.74606.ba>
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments and Computers, 36*, 717–731. <https://doi.org/10.3758/bf03206553>
- Smith, R. P., Katz, C. L., Charney, D. S., & Southwick, S. M. (2007). Neurobiology of disaster exposure: Fear, anxiety, trauma, and resilience. In R. J. Ursano, C. S. Fullerton, & L. Weisaeth (Eds.), *Textbook of disaster psychiatry* (pp. 97–120). Cambridge, UK: Cambridge University Press.
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston, MA: Pearson/Allyn & Bacon.
- Thomas, É., Saumier, D., & Brunet, A. (2012). Peritraumatic distress and the course of posttraumatic stress disorder symptoms: A meta-analysis. *Canadian Journal of Psychiatry. Revue Canadienne de Psychiatrie, 57*, 122–129. <https://doi.org/10.1177/070674371205700209>
- Vaiva, G., Brunet, A., Lebigot, F., Boss, V., Ducrocq, F., Devos, P., ... Goudemand, M. (2003). Fright (effroi) and other peritraumatic responses after a serious motor vehicle accident: Prospective influence on acute PTSD development. *Canadian Journal of Psychiatry. Revue Canadienne de Psychiatrie, 48*, 395–401. <https://doi.org/10.1177/0886260512475314>
- Yaşan, A., Güzel, A., Tamam, Y., & Ozkan, M. (2009). Predictive factors for acute stress disorder and posttraumatic stress disorder after motor vehicle accidents. *Psychopathology, 42*, 236–241. <https://doi.org/10.1159/000218521>