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Posttraumatic Stress Hyperarousal Symptoms Mediate the Relationship Between Childhood Exposure to Violence and Subsequent Alcohol Misuse in Mi'kmaq Youth

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*This study was part of a school-based collaborative research project with a Canadian Mi'kmaq community that examined the potential role of posttraumatic stress (PTS) symptom clusters in mediating the relationship between childhood exposure to violence (EV) and alcohol misuse in a sample of Mi'kmaq adolescents (*N = *166). The study employed a cross-sectional design and used several well-validated self-report questionnaires. Path analytic results showed that when each PTS symptom cluster was independently investigated for mediating effects while controlling for depressive symptoms, age, and gender, only the PTS hyperarousal symptom cluster fully mediated the EV–alcohol misuse relationship. Results are discussed within the context of previous theory and research on the topic of PTS as a mediator between EV and alcohol misuse.*

Interpersonal violence, whether experienced directly or indirectly, especially during childhood, can either precipitate the onset of posttraumatic stress disorder (PTSD) or act as a risk factor that later increases the odds of developing PTSD after subsequent traumas (Brewin, Andrews, & Valentine, 2000). Unfortunately, youth exposure to violence (EV) is not rare. Two American studies (Finkelhor, Ormrod, Turnery, & Hamby, 2005; Hanson et al., 2008) using nationally representative samples of youth suggest that one fifth to one half of all children and adolescents have experienced a physical assault, a little over one third have witnessed violence perpetrated toward another, and almost one tenth have been sexually victimized. These figures are particularly alarming because EV increases the risk of developing PTSD over-and-above other types of traumatic events (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Kilpatrick et al., 2000). Furthermore, not only is EV more likely to result in PTSD or become a risk factor for the later onset of PTSD after a subsequent trauma, but EV is also more likely to predict the development of PTSD with comorbid substance misuse (Wekerle & Wall, 2002). This study refers to both posttraumatic stress disorder (PTSD) and posttraumatic stress (PTS) symptoms of PTSD. The term PTSD is used when referring to the literature that employed this diagnostic label, but as we did not measure diagnostic status in this study, we employ the term PTS when referring to the construct we have measured.

There are well-documented relationships between childhood maltreatment and both PTSD and alcohol abuse/dependence (Langeland, Draijer, Nel, & van den Brink, 2004). Moreover, much of the research indicates that PTSD symptoms and alcohol misuse are commonly "comorbid" (Stewart, 1996)—that is, they occur together in the same individuals far more commonly than can be explained by chance. Studies show that the lifetime

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prevalence rate of having an alcohol-use disorder (abuse or dependence) for those with PTSD ranges from 21.6 to 51.9% but only from 8.1 to 34.4% for those without PTSD (Breslau, Davis, Peterson, & Schultz, 1997; Kessler et al., 1995).

Although there are a number of possible explanations for the comorbidity of PTSD and alcohol abuse/dependence, many researchers suggest that those with PTSD misuse alcohol (or other substances) to self-medicate their PTSD symptoms (De Bellis, 2002; Chilcoat & Breslau, 1998; Stewart, Mitchell, Wright, & Loba, 2004). The self-medication theory argues that central nervous system depressants like alcohol may help attenuate certain fear/startle responses as well as the intrusive memories that are characteristic of PTSD (Jacobsen, Southwick, & Kosten, 2001). The relationship between PTSD symptoms and alcohol misuse can become further complicated by a process known as *mutual maintenance* (Stewart, Pihl, Conrod, & Dongier, 1998). Although an individual receives initial PTSD symptom relief immediately following the consumption of alcohol, once its effects have worn off the PTSD symptoms return. Some of those symptoms, particularly hyperarousal, can return with even greater severity, largely due to the physiological arousal relating to withdrawal from central nervous system depressants like alcohol (Jacobsen et al., 2001). This maintenance or intensification of PTSD symptoms then resets the stage for continued alcohol misuse, potentially causing further dysregulation of biological stress response systems (De Bellis, 2002), and interfering with the body's natural habituation to traumatic memories. In this way, alcohol misuse can actually serve to maintain PTSD symptoms in the longer term creating a vicious cycle between PTSD symptoms and alcohol misuse.

In essence, the self-medication/mutual maintenance theories suggest that PTSD acts as a mediating variable, that is, a variable that intervenes and helps explain the relationship between trauma exposure and subsequent alcohol misuse. Previous studies that attempted to show the role of PTSD as a mediator between a traumatic event and later alcohol misuse have shown weak to moderate support (Epstein, Saunders, Kilpatrick, & Resnick, 1998; White & Widom, 2008; Zlotnick et al., 2006) though a couple have shown no support (Prigerson, Maciejewski, & Rosenheck, 2002; Ullman, Filipas, Townsend, & Starzynski; 2005). However, none of these studies examined the potential mediating role of the individual PTSD symptom clusters.

In light of the relationships between the PTSD symptom clusters of hyperarousal (McFall, MacKay, & Donovan, 1992; Stewart, Conrod, Pihl, & Dongier, 1999; Stewart et al., 2004), reexperiencing (McFall et al., 1992; Read, Brown, & Kahler, 2004; Simons, Gaher, Jacobs, Meyer, & Johnson-Jimenez, 2005) and avoidance and numbing (Sullivan & Holt, 2008; Taft et al., 2007) with alcohol misuse, it is important to examine the potential mediating role of each PTSD symptom cluster because the self-medication model would predict that individuals exposed to violence might be drinking to cope with any of these symptoms (Stewart, 1996). An important variable to control for in the trauma–alcohol misuse relationship is depressive symptoms, however, because of the strong relationships found between depressive symptoms and EV, depressive symptoms and alcohol misuse, and depressive symptoms and PTSD (Kessler, Davis, & Kendler, 1997; Kilpatrick et al., 2003).

The present study is also novel in that this is the first attempt we know of to address this issue in both an adolescent sample and in partnership with an Aboriginal community. Although the literature is scant, there is evidence to attest to the fact that some North American Aboriginal communities are facing severe issues that involve high rates of EV, PTSD, depressive symptoms, and alcohol misuse. For example, the Canadian Aboriginal People's Survey reported that 73% of Aboriginal respondents reported that alcohol was a problem in their community (Statistics Canada, 1991). From the same survey, 44% of respondents indicated that family violence was a problem in their community, while just over half indicated that both physical and sexual abuse were problems as well (Statistics Canada, 1991). Another study showed that approximately 40% of a sample of 234 American Indians reported exposure to severe child maltreatment, which was most strongly associated with PTSD over other psychological disorders (Duran et al., 2004). Boyd-Ball, Manson, Noonan, and Beals (2006) examined the relationship between trauma exposure and alcohol-use disorders in a sample of American Indian adolescents and young adults. Compared to nontraumatized youth, youth that reported three or more severe traumas were 3.6 times more likely to have a lifetime diagnosis of an alcohol-use disorder, and that interpersonal violence compared to other traumas was more predictive of having a lifetime diagnosis of an alcohol-use disorder (Boyd-Ball et al., 2006); however, they did not examine the potential influence of PTSD. Other studies with American Indian populations have linked both childhood physical and sexual assault to both depressive symptoms and subsequent alcohol misuse (Libby et al., 2004, 2005). With respect to alcohol consumption, a recent literature review concluded that rates of alcohol use among American Indians and Alaskan Natives are higher than in the general population for both adults and adolescents, and that Native adolescents experience more negative consequences of their drinking than other adolescents (Szlemko, Wood, & Jumper-Thurman, 2006). Thus, by working in partnership with a Mi'kmaq community (see Zahradnik et al., 2007), we attempted to address the question of PTS mediation with a community that is not alone in struggling with the issues highlighted.

We hypothesized that adolescents with higher amounts of EV would be more likely to develop PTS symptoms, and these PTS symptoms would in turn more likely lead to alcohol misuse, even after controlling for the influence of depressive symptoms. Consistent with current studies on how best to organize PTSD symptom clusters (Asmundson et al., 2000; King, Leskin, King, & Weathers, 1998; Stewart et al., 1999) we specified four separate path models—hyperarousal, reexperiencing, numbing, and avoidance—in which it was predicted that in each model EV

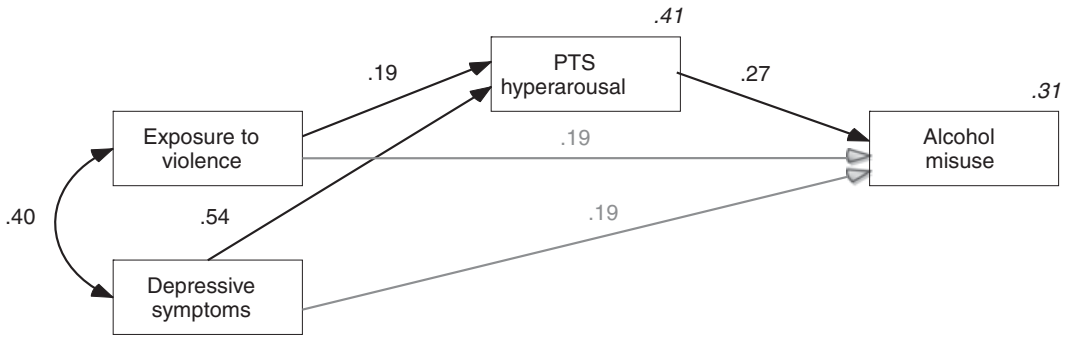

Figure 1. The structural model involving posttraumatic stress (PTS) hyperarousal. Black arrows represent significant paths (i.e., $p < .05$). Grey arrows represent nonsignificant paths. Rectangles represent manifest variables. Path coefficients are standardized. Italicized numbers (e.g., *. 31*) positioned in the upper right hand of endogenous variables (e.g., alcohol misuse) represent the proportion of variance explained by associated exogenous variables. In the interest of clarity, error terms and demographic variables are not displayed.

would be indirectly related to alcohol misuse through one of our four PTS symptom clusters. See Figure 1 for an example of the hypothesized mediational model for PTS hyperarousal.

METHOD

Participants and Procedure

There are approximately 25,000 Mi'kmaq living in Nova Scotia. The Mi'kmaq makeup about 2.6% of the provincial population and there are 35 reserves administered by 13 self-governing communities. The community of focus in the current study is one of the larger communities; but given this community's request for anonymity, identifying information cannot be provided. A more general description of the community and the steps involved in acquiring community consent and participation in the research process is available (Zahradnik et al., 2007; Zahradnik et al., 2010).

The sample was drawn from the community's school-attending youth, and data were collected in both the spring and the fall of 2006. Across the two schools, 166 students participated in the study, which is just over half of those that were eligible to participate. With respect to gender, there were 91 female students and 75 male students. Ages ranged from 14 to 18 years ($M = 16.69$, $SD = 1.39$), with most students (77.7%) being 16 years of age or older, the age at which according to Nova Scotian law (Children and Family Services Act, 1990) a child can choose whether she or he wishes to report a case of abuse where she or he alone was the victim. The self-reported education level ranged from Grades 8–12 ($M = 9.91$, $SD = 1.05$).

Measures

Physical, sexual, and emotional abuse/exposure to domestic violence was measured with the Childhood Experience of Violence Questionnaire (CEVQ; Walsh, MacMillan, Trocmé, Jamieson, & Boyle, 2008), an 18-item self-report measure of childhood EV for use with children/youth 12–18 years. The CEVQ collects information about whether abuse has been experienced and if so, about the severity (measured continuously), onset, and duration of abuse experienced. The reliability coefficient for this measure in our sample was high ($\alpha = .92$).

Posttraumatic stress symptoms were measured with the Child PTSD Symptom scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001). The CPSS is a 17-item self-report measure designed to tap each of the three symptom clusters of PTSD according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994)—reexperiencing, avoidance/numbing, and hyperarousal—in children/youth from ages 8–18 years. In this sample, two of the CPSS subscales (Numbing and Reexperiencing) had unacceptably low Cronbach's α (below .60) because of three problematic items: two items from the Numbing subscale (Item 8: traumatic episode-related memory problems, and Item 12: having a sense of a foreshortened future), and one item from the Reexperiencing subscale (Item 4: emotional reactivity to triggers). We did not include these items in our subscale totals. Research on PTSD has identified these two numbing items as problematic (King et al., 1998). Thus, it is not unusual for some researchers to also drop these items from their studies (Palyo, Clapp, Beck, Grant, & Marques, 2008). Furthermore, we felt it was justified to drop the reexperiencing item because its low internal consistency can be interpreted within the context of other findings that suggest that First Nations people are more likely to experience their anxiety somatically than emotionally (Barker-Collo, 1999). Thus, our four subscales were as follows: Reexperiencing (four items; e.g., nightmares; $\alpha = .72$), Hyperarousal (five items; e.g., exaggerated startle response; $\alpha = .63$), Avoidance (two items; e.g., avoiding thinking about

the trauma; α = .71), and Emotional Numbing (three items; e.g., feeling emotionally numb; α = .73).

The 20-item Centre for Epidemiological Studies Depression Scale (CESD; Radloff, 1977) was used to measure depressive symptoms. The CESD has been used previously with adolescents (Radloff, 1991). Summing all items yields one overall score ranging from 20 to 80 that reflects depressive symptom severity (present sample α = .84). This scale has been well-validated in different samples of Aboriginal adolescents (Manson, Ackerson, Dick, Barón, & Fleming, 1990; Thrane, Witbeck, Hoyt, & Shelley, 2004).

Evidence suggests measuring the problems that arise as direct consequences of alcohol consumption is a good indicator of alcohol-use disorders in youth (White & Labouvie, 1989); therefore, the Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) was used to measure alcohol misuse. The RAPI is a well-validated 23-item self-report measure that assesses adolescent problem drinking symptoms. It has been validated in community, clinical, and First Nations samples (Noel et al., 2010; White & Labouvie, 1989; Winters, 1999). Responses were summed across the 23 items (α = .97), as recommended by the authors of the RAPI, to yield a composite score that takes problem frequency into account (cf. Winters, 1999). Forty percent of the participants reported not drinking and were given a score of zero for this measure.

Analysis

Path analysis was conducted with AMOS 7.0 (Arbuckle, 2006). Goodness-of-fit of structural models was evaluated via multiple indices. Adequate fit is indicated by a chi-square-to-degrees of freedom ratio (χ^2/df) around 2, an incremental fit index (IFI) and a comparative fit index (CFI) around .95, and a root-mean-square error of approximation (RMSEA) around .08 (Kline, 2005). The RMSEA values are reported with 95% confidence intervals (95% CIs). Given concerns about multivariate nonnormality in our data, we used bootstrapping to address the possible effect of multivariate nonnormality in our path models (Schumacker & Lomax, 2010). All paths in each of the four path models examining the potential mediating role of the PTS symptom clusters were reexamined using bootstrapping procedures. Paths generated using bootstrapping were virtually identical to the results shown in Figure 1 and summarized in text, suggesting multivariate nonnormality had little or no influence on the results. Bootstrapping estimates are not presented in the main text because such estimates are excessively strict if significant deviations from multivariate normality are not present (Nevitt & Hancock, 2001). In sum, bootstrapping procedures suggested the results were not unduly influenced by possible deviations from multivariate normality.

Mallinckrodt, Abraham, Wei, and Russell (2006) assert that a significant indirect effect suggests mediation has occurred. The significance levels of all indirect effects were tested using random sampling with replacement to generate 20,000 ($n = 166$) bootstrap samples. Bootstrapping allowed us to estimate bias-corrected standard errors for our indirect effects. Confidence intervals were also calculated, and indirect effects were considered significant ($p < .05$) if the 95% CI for these indirect effects did not contain zero.

RESULTS

With respect to EV, based on the CEVQ's more stringent definitions of physical and sexual abuse (Walsh et al., 2008), 47% of the sample reported physical abuse, 34.3% of the sample reported sexual abuse, and 57.8% of the sample reported either physical or sexual abuse. Exposure to violence was moderately correlated with all four PTS symptoms, depressive symptoms, and alcohol misuse (see Table 1 for correlations, means, and standard deviations). Furthermore, both depressive symptoms and all four PTS symptoms were correlated with alcohol misuse. Age correlated with EV, PTS hyperarousal, PTS avoidance and alcohol misuse, and gender correlated with PTS reexperiencing and

Table 1. Bivariate Correlations Among Variables, Means, and Standard Deviations

Variables	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. EV	14.38	13.15	–	.41***	.47***	.44***	.45***	.42***	.18*	−.11	.41***
2. PTS Hyperarousal	5.27	3.35		–	.52***	.44***	.59***	.62***	.16*	−.12	.49***
3. PTS Reexperiencing	2.67	2.49			–	.63***	.59***	.56***	.10	−.21**	.31***
4. PTS Avoidance	1.75	1.79				–	.57***	.47***	.17*	−.11	.25***
5. PTS Numbing	2.42	2.42					–	.70***	.07	−.11	.33***
6. Depressive Symptoms	19.07	19.89						–	.06	−.22**	.45***
7. Age	16.69	1.39							–	.13	.26***
8. Gender										–	−.03
9. Alcohol Misuse	9.65	11.06									–

Note. $N = 166$ with 91 males and 75 females. EV = Exposure to violence; PTS = posttraumatic stress.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2. Model Fit Indices for Structural Models Testing the Indirect Effects of Exposure to Violence on Alcohol Misuse Through PTS Symptoms

Structural models	χ^2	χ^2/df	IFI	CFI	RMSEA	95% CI
Hyperarousal	15.25	2.18	.96	.96	.09	[0.00, 0.15]
Reexperiencing	14.43	2.06	.96	.96	.08	[0.00, 0.15]
Avoidance	14.85	2.12	.95	.95	.08	[0.00, 0.15]
Numbing	13.80	1.97	.97	.97	.08	[0.00, 0.15]

Note. PTS = Posttraumatic stress; IFI = incremental fit index; CFI = comparative fit index; RMSEA = root-mean-square error of approximation; CI = confidence interval.

depressive symptoms. To account for their potential influence, age, gender, and depressive symptoms were included as covariates in all structural models involving PTS symptom clusters as potential mediators.

Fit indices for path models appear in Table 2. All four models fit the data reasonably well. However, when controlling for the effects of depressive symptoms in each of the four models, only one of the models, the PTS hyperarousal model, resulted in a significant relationship between the potential mediator and the outcome variable ($\beta = .27, p = .06$; see Figure 1). For each of the other three models there were no significant relationships between the PTS symptom cluster and alcohol misuse once depressive symptoms were controlled for: reexperiencing ($\beta = -.02, p = .85$), avoidance ($\beta = -.06, p = .17$), and numbing ($\beta = -.04, p = .681$). In contrast, the pathways from depressive symptoms to alcohol misuse were significant in these models (βs = .35, .36, and .37, respectively). Consequently, only the indirect effect of EV on alcohol misuse through PTS hyperarousal was significant (see Table 3.

In summary, all four of the PTS symptom cluster models adequately fit the data. As hypothesized, PTS hyperarousal symptoms involved a significant indirect effect; however, indirect effects were not observed for the three other PTS symptom cluster models.

DISCUSSION

Although prior studies have examined the role of PTSD as a mediating variable between some form of EV and alcohol misuse (Epstein et al., 1998; White & Widom, 2008; Zlotnick et al., 2006), this is the first study of which we are aware to examine the contribution of specific PTS symptom dimensions while controlling for depressive symptoms, which are known to be related to all three aforementioned variables (Kilpatrick et al., 2003). It is also the first study we know of to provide support for the PTS-specific self-medication hypothesis by demonstrating mediation in a sample of Mi'kmaq (First Nation) adolescents. Our findings supported an indirect relationship of EV to alcohol misuse through PTS hyperarousal symptoms, but not any of the other PTS symptoms.

That hyperarousal symptoms fully mediated the relation between EV and alcohol misuse is consistent with research linking PTSD hyperarousal symptoms to alcohol misuse (McFall et al., 1992, Simons et al., 2005, Stewart et al., 1999). With respect to the other PTS symptoms, although at the univariate level of analysis these symptom clusters were related to alcohol misuse, contrary to our hypothesis, they did not explain the relationship between EV and alcohol misuse after controlling for the influence of depressive symptoms. Although these null results were contrary

Table 3. Bootstrap Analyses for Indirect Effects of PTS Symptoms Between Exposure to Violence and Alcohol Misuse

			Bootstrap estimates	
Hypothesized indirect effect	Unstandardized indirect effect	Standardized indirect effect	*SE* for standardized indirect effect	95% CI for standardized indirect effect (lower, upper)
PTS hyperarousal	.042	.051	.028	[0.010, 0.126]*
PTS reexperiencing	−.004	−.004	.031	[−0.075, 0.051]
PTS avoidance	−.014	−.017	.027	[−0.075, 0.034]
PTS numbing	−.007	−.008	.021	[−0.057, 0.030]

Note. PTS = Posttraumatic stress; CI = confidence interval; *SE* = bias-corrected standard error.
*Confidence intervals excluding zero are significant (i.e., $p < .05$).

to our hypothesis, we note not all studies examining the relationships between the PTSD symptoms and alcohol misuse report a significant univariate relationship (Stewart et al., 1999), let alone a multivariate one. Alternatively, other PTS symptoms, like reexperiencing symptoms, might better explain why EV can lead to misuse of drugs other than alcohol (McFall et al., 1992). Thus, future research might also use illicit drug use and/or misuse of prescription medications as outcomes, but researchers are cautioned not to group arousal-enhancing and arousal-dampening drugs together as mediation findings may be specific to arousal-dampening drugs (Stewart & Conrod, 2008).

The results of our study are consistent with other research in this area. For example, our sample had comparable but higher rates of abuse to a large community-based study of high risk adolescents that examined the impact of abuse before age 10 on subsequent drinking, and found that abuse increased the risk of ever using alcohol, preteen-first-age alcoholic drink, and binge drinking (Hamburger, Leeb, & Swahn, 2008). This study also makes several novel contributions. Our study is the first we know to examine the mediating effects of specific PTS symptom clusters, as opposed to examining the PTS construct as a whole (Prigerson et al., 2002; Ullman et al., 2005; White & Widom, 2008; Zlotnick et al., 2006). Furthermore, we show that even when controlling for the role of depressive symptoms—a construct with strong associations to alcohol misuse—PTS hyperarousal symptoms better explain the link between EV and alcohol misuse. In other words, those youth with a history of EV were more likely to misuse alcohol in relation to their hyperarousal symptoms (i.e., perhaps to dampen these symptoms) regardless of whether or not they would meet all the necessary criteria for a diagnosis of PTSD. Our results are also consistent with the theory of mutual maintenance in which bouts of alcohol consumption, used to dampen hyperarousal symptoms, can actually, by way of alcohol withdrawal, increase the severity of these very symptoms (Jacobsen et al., 2001); however, our design cannot make causal assertions.

Our study found support for mediation effects of PTS symptoms in a sample of adolescents, and it is rare to examine this question in a First Nations community. This is important because it is common for First Nations' communities, and North American Aboriginal communities more generally, given their history of colonization and cultural discontinuity (Kirmayer, Brass, & Tait, 2000), to be struggling with a variety of social issues (e.g., suicide, fetal alcohol syndrome, community violence, and child maltreatment) that are related to problematic alcohol use.

The results of this study might be of interest to Aboriginal communities looking to better understand a specific pathway that can lead to problematic alcohol use in their youth. The results of this study, however, must be understood and interpreted within the greater socioeconomic determinants of problematic alcohol use in Aboriginal communities, determinants that include poverty, unemployment, poor health, low educational levels, and low or absent community economic development (Health Canada, 1998).

A limitation of our study was that it was a school-based study that used a nonrandom sample. Consequently, the sample is not representative of the whole community and may not generalize to those youth who are not enrolled in school, and who may be dealing with more severe sequelae of EV. Another important caveat is that this study relied on a cross-sectional design. Thus, inferences made about mediation and causation, given the lack of information about temporal sequencing, are not dispositive. This being said, most empirical evidence suggests trauma and PTS symptoms precede alcohol misuse in the majority of comorbid cases (Bremner, Southwick, Darnell, & Charney, 1996; Chilcoat & Menard, 2003). Also of note, stressful life events are common in many First Nations communities (e.g., youth suicides or motor vehicle accidents). Thus, the expression of PTS symptoms as they were captured in this study may not be solely attributable to EV. Although face-to-face interviews are an ideal way to disentangle the chronologies of multiple traumas as they relate to the development of PTS, our use of anonymous self-report measures made this impossible. Furthermore, our sample size precluded us from examining the models with pure cases of one form of abuse (e.g., sexual abuse alone); however, two separate studies have now confirmed that the relationship to alcohol use from both childhood sexual abuse (Epstein et al., 1998) and physical abuse (Zlotnick et al., 2006) is mediated by PTSD. Our sample size also limited our ability to examine all four PTS symptoms and covariates simultaneously within the same path model. Given the strong relationship between these four variables, such an analysis is recommended in the future.

Another limitation is that we measured depressive symptoms with the CES-D, which some argue is better conceived of as a measure of general distress, given research showing the CES-D measures anxious in addition to depressive symptoms (Fechner-Bates, Coyne, & Schwenk, 1994). Thus, future inquiries into this topic might use a purer measure of depressive symptoms. Also of note, White and Widom (2008) suggest that stressful life events, and not PTSD symptoms, fully mediate the relationship between early abuse/neglect and subsequent alcohol problems. Thus, future studies should examine the interplay between factors that contribute to stressful life events (e.g., chaotic interpersonal relationships at home) and PTS symptoms. We were also not able to control for parental alcohol use, a variable that is strongly related to problematic drinking in adolescents (Shin, Edwards, & Heeren, 2009). The literature on adolescent misuse of alcohol, however, also suggests childhood maltreatment is related to adolescent problem drinking even after controlling for parental alcoholism (Harter, 2000; Shin et al., 2009).

Finally, our study did not directly measure motivations to consume alcohol, and measuring explicit motivation is important because drinking to cope (Cooper, 1994) is strongly related to PTSD and alcohol misuse (Dixon, Leen-Feldner, Ham, Faldner, Lewis, & 2009; Kaysen et al., 2007). Measuring a construct like drinking motives could provide more direct evidence of intentional

self-medication, particularly because self-medication is a dynamic process and researchers are only beginning to analyze the fluctuating interplay between both sets of symptoms across time (Ouimette, Read, Wade, & Tirone, 2010). Ultimately then, future studies would be strengthened by using a prospective design with a direct measure of drinking motives, and with a sample large enough to evaluate all symptoms simultaneously, and to explore the model for both genders and specific types of abuse, while controlling for parental alcohol consumption and the potential mediating role of stressful life events.

The results nonetheless have important clinical implications. For individuals with both PTS hyperarousal symptoms and alcohol-use problems, clinicians should give consideration to whether or not a trauma-specific therapy that reduces hyperarousal symptoms should be used as an adjunct to treatment for the alcohol misuse. A treatment like imaginal exposure therapy might be useful because it has been demonstrated to decrease all three *DSM-IV* symptom domains (Robertson, Humphreys, & Ray, 2004). In keeping with best practice guidelines, individuals who meet the diagnostic criteria for both PTSD and an alcohol-use disorder should receive an integrated treatment approach (Health Canada, Centre for Addiction and Mental Health, 2002) that targets the functional relationship between both disorders (Zahradnik & Stewart, 2008). An integrated treatment approach to treating PTSD-alcohol use comorbidity is important given that unremitted PTSD is implicated in deleterious substance use disorder outcomes (Read et al., 2004). As such, treatments that only focus on the alcohol/substance use disorder and not the PTSD symptoms will likely be less effective in the long term. Therefore, individuals with both disorders will likely benefit from services in which mental health and addiction services are working together.

Overall, this study offers support for a more specific version of the trauma-PTS/PTSD-alcohol self-medication model by highlighting the importance of hyperarousal symptoms. After controlling for age, gender, and depressive symptoms, our results suggest that for these Mi'kmaq adolescents, it is hyperarousal symptoms, and hyperarousal symptoms alone, that explain the relationship between EV and alcohol misuse. Thus, future research in this area should consider the role of specific PTSD symptom clusters, and not just the construct of PTSD as a whole.

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