Journal of Anxiety Disorders 25 (2011) 978–987

Contents lists available at ScienceDirect

Journal of Anxiety Disorders

PTSD symptoms, potentially traumatic event exposure, and binge drinking: A prospective study with a national sample of adolescents

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ARTICLE INFO

Article history:
Received 1 October 2010
Received in revised form 13 June 2011
Accepted 16 June 2011

Keywords:
PTSD
Trauma
Assault
Binge drinking
Adolescents

ABSTRACT

Research demonstrates substantial comorbidity between PTSD and alcohol use disorders. Evidence for functional relationships between PTSD and problematic alcohol use has not always been consistent, and there have been few investigations with adolescent samples. Further, research has not consistently controlled for cumulative potentially traumatic event (PTE) exposure when examining prospective relationships between PTSD and problematic alcohol use (i.e., binge drinking). This study examines the prospective relationships between PTSD symptoms, problematic alcohol use, and cumulative PTE exposure measured at three time points over approximately three years among a nationally representative sample of adolescents exposed to at least one PTE ($n = 2399$ and age range = 12–17 at Wave 1). Results from parallel process latent growth curve models demonstrated that increases in cumulative PTE exposure over time positively predicted increases in both PTSD symptoms and binge drinking, whereas increases in PTSD symptoms and increases in binge drinking were not related when controlling for the effect of cumulative PTE exposure. Further analyses suggested that these relationships are specific to assaultive PTEs and are not found with non-assaultive PTEs. Theoretical implications are discussed.

Exposure to potentially traumatic events (PTE) is common, with a prevalence of approximately 65% in the general population (Kessler, 2000; Kilpatrick, Ruggiero, Acierno, Saunders, Resnick, & Best, 2003; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). Some individuals develop posttraumatic stress disorder (PTSD) following PTE exposure, which is characterized by intrusive re-experiencing of a traumatic event, avoidance of trauma-related cues, and physiological hyperarousal symptoms (American Psychiatric Association [APA], 2000). PTSD is associated with significant psychosocial impairment (Amaya-Jackson, Davidson, Hughes, Swartz, Reynolds, & George, 1999; Breslau, 2001; Kessler, 2000) and lower quality of life relative to other anxiety disorders (Olatunji, Cisler, & Tolin, 2007). In contrast to overall PTE prevalence, PTSD has an estimated prevalence of 8% (APA, 2000; Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Chiu, Demler, Merikanga, & Walters, 2005; Kilpatrick et al., 2003; Resnick et al., 1993). Given the prevalence and negative impact of PTSD, researchers have become increasingly interested in delineating mechanisms contributing to the development and maintenance of PTSD.

Systematically examining comorbid conditions is one approach that may shed light on these mechanisms of PTSD. Such research has shown that PTSD is often comorbid with substance use disorders (Chilcoat & Breslau, 1998; Breslau, Davis, & Schultz, 2003; McFarlane, 1998; Stewart, 1996; Stewart, Pihl, Conrad, & Dongier, 1998), particularly alcohol use disorders (AUDs; Mills, Teesson, Ross, & Peters, 2006; Stewart, 1996). For example, 52% of males and 30% of females diagnosed with PTSD are also diagnosed with an AUD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). This relationship is also found among young adults and adolescents (Kilpatrick et al., 2003; Reed, Anthony, & Breslau, 2007). For example, Kilpatrick et al. (2003) found that 30% of adolescent males and 24% of adolescent females diagnosed with PTSD were also diagnosed with a substance use disorder. The high prevalence of AUD and other substance use disorders among individuals with PTSD can be compared to the lower prevalence rates of 5% and 1% for AUDs and other substance use disorders, respectively, in the general population (Compton, Thomas, Stinson, & Grant, 2007; Hasin, Stinson, Ogburn, & Grant, 2007).

Given the robustness of these comorbidity patterns, there has been much speculation as to why PTSD is so frequently comorbid with AUDs and other substance use disorders. The explanation that

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doi:10.1016/j.janxdis.2011.06.006

has received the most empirical attention is the self-medication hypothesis (Khantzian, 1985). This hypothesis has two main predictions: (1) alcohol intoxication should at least temporarily reduce anxiety, which provides a mechanism for negative reinforcement via escape or avoidance of PTSD symptoms, and (2) PTSD development should precede the development of AUD. Research over the last two decades has yielded mixed results in support of the self-medication hypothesis. First, research has not consistently demonstrated that alcohol intoxication reliably causes straightforward reductions in anxiety during a stressor (Abrams & Wilson, 1979; Carrigan & Randall, 2003; Himle, Abelson, Haghightgou, Hill, Nesse, & Curtis, 1999; Kushner, Mackenzie, Fiszdon, Valentiner, Foa, & Anderson, 1996; Kushner, Abrams, & Borchardt, 2000; Morris, Stewart, & Ham, 2005). Instead, whether alcohol intoxication leads to reductions in anxiety depends on the type of stressor (e.g., carbon dioxide challenge versus public speech challenge; Himle et al., 1999; Kushner et al., 1996), whether drinking occurs before or after learning about the upcoming stressor (e.g., appraisal disruption hypothesis; Sayette, 1993), whether there is a family history of alcoholism or anxiety disorders (Sinha, Robinson, & O'Malley, 1998), and also on one's alcohol expectancies (Abrams, Kushner, Medina, & Voight, 2004). This research suggests that alcohol only offers escape or avoidance of anxiety symptoms under certain conditions, which calls into question the power of the self-medication hypothesis' first prediction. Second, the prediction that PTSD precedes the development of AUD also has inconsistent support. Several studies have found that PTSD prospectively predicts AUD onset (Breslau, Davis, Peterson, & Schultz, 1997; Chilcoat & Breslau, 1998; McFarlane, 1998; Reed, Anthony, & Breslau, 2007), while other prospective studies either suggest no relationship between PTSD and AUD (Breslau, Davis, & Shultz, 2003; Shipherd, Stafford, & Tanner, 2005) or show the opposite temporal pattern (Cottler, Compton, Mager, Spitznagel, & Janca, 1992). This research further questions the accuracy of the self-medication hypothesis.

A second explanation for the robust relationship between AUDs and PTSD is that AUDs increase vulnerability towards the development of PTSD (Kushner et al., 2000; Stewart et al., 1998). One central mechanism in this explanation is that AUDs are associated with anxiogenic withdrawal symptoms, and these anxiogenic withdrawal symptoms increase PTSD symptoms following PTE exposure (Kushner et al., 2000). Another mechanism posited in this explanation is that behavior during alcohol intoxication may increase the likelihood of PTE exposure, thus also increasing likelihood of PTSD development (Jacobsen, Southwick, & Kosten, 2001; Stewart et al., 1998). The hypothesis that AUDs may increase vulnerability to PTSD, regardless of the mediating mechanism, predicts that AUD onset should temporally precede the development of PTSD. However, this prediction has also not been consistently supported. Whereas some studies support this temporal relationship (Cottler et al., 1992), as noted above, several studies find the opposite temporal pattern (Breslau et al., 1997; Chilcoat & Breslau, 1998; McFarlane, 1998; Reed, Anthony, & Breslau, 2007).

A third hypothesis explaining the robust relationship between PTSD and AUDs posits that the relationship may be due to a shared third variable (Kushner et al., 2000; Stewart et al., 1998). While focus commonly has been on shared genetic influences as the third variable explaining the relationship between PTSD and AUD (cf., Kushner et al., 2000), another possibility that has not received thorough empirical testing is that cumulative PTE exposure may account for vulnerability towards both outcomes (cf., Brady & Sinha, 2005). Previous research has yielded mixed results regarding whether PTE exposure alone (i.e., independent of PTSD) increases risk for AUDs, with some studies yielding a null effect (e.g., Breslau et al., 2003; Chilcoat & Breslau, 1998; Reed et al., 2007) and others yielding a positive effect (e.g., Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997; Kilpatrick, Acierno, Saunders, Resnick, Best, & Schnurr, 2000; Stewart, 1996). One possible explanation for the inconsistent results is that studies often collapse the count of PTE exposure into a dichotomous variable of 'any exposure' versus 'no exposure.' PTE exposure may have a cumulative allostatic effect (Kollassa et al., 2010; McEwen, 2004; Neuner, Schauer, Karunakara, Klaschik, Robert, & Elbert, 2004; Steel, Chey, Silove, Marnane, Bryant, & van Ommeren, 2009; but also see Breslau, Peterson, & Schultz, 2008), and dichotomization may fail to capture this effect. Additionally, different PTEs may have different effects on mental health outcomes. For example, assaultive (e.g., rape, physical assault) PTEs are associated with higher rates of PTSD compared to non-assaultive PTEs (Kessler et al., 1995; Resnick et al., 1993). Studies that only examine 'any PTE exposure' versus 'no PTE exposure' necessarily collapse across types of PTEs, which could further obscure any clear independent effects of PTE exposure. No previous epidemiological study has examined the relationship between PTSD symptoms and drinking behavior when accounting for this effect of cumulative PTE.

A major limitation of the available studies examining relationships between PTEs, PTSD, and alcohol use is that they are almost exclusively limited to adult samples. There is a general lack of prospective studies examining these relationships among adolescents, and cross-sectional studies provide conflicting findings regarding functional relations (Danielson et al., in press; DeMilio, 1989; Lopez, Turner, & Saavedra, 2005). Examining these relationships among adolescents specifically is important for two reasons. First, children and adolescents may be more sensitive to the effects of PTE exposure. Evidence for this hypothesis comes from studies demonstrating that age of PTSD onset among children and adolescents positively correlates with brain volume (De Bellis et al., 1999) and that stress has a stronger negative biological impact during adolescence compared to adulthood (McCormick, Mathews, Thomas, & Waters, 2010; McCormick & Mathews, 2007). If adolescence is a developmental period marked by sensitivity to stress, then different relations might therefore be expected between cumulative PTE exposure, PTSD symptoms, and problematic drinking behavior. Second, drinking behavior during adolescence, particularly binge drinking, is strongly predictive of future AUD development (Bonomo, Bowes, Coffey, Carlin, & Patton, 2004; Hingson, Heeren, & Winter, 2006; Viner & Taylor, 2007). Accordingly, understanding the factors influencing adolescent drinking behavior may facilitate understanding of both adolescent and future drinking behavior.

The present study provides a unique opportunity to empirically examine the three competing explanations of the relation between PTSD and problematic alcohol use among a nationally representative sample of adolescents, and also to test whether these relationships differ as a function of type of traumatic event exposure (i.e., assaultive versus non-assaultive; cf., Resnick et al., 1993). Problematic alcohol use was indexed with binge drinking frequency. Binge drinking frequency during adolescence is strongly predictive of subsequent AUD (Bonomo et al., 2004; Viner & Taylor, 2007); thus, binge drink frequency is a useful measure of problematic alcohol use, even when there is no indication of impairment.

1. Method

1.1. Participants

The National Survey of Adolescents-Replication (NSA-R) is a longitudinal epidemiological study of youth aged 12–17 ($n = 3614$ at Wave 1) residing in the U.S, aimed at determining the prevalence and correlates of adolescents' exposure to PTEs. The sampling and