Journal of Traumatic Stress, Vol. 00, No. 0, 2011, pp. 1–11 (© 2011)

PTSD and Alcohol Use After the World Trade Center Attacks: A Longitudinal Study

Joseph A. Boscarino, H. Lester Kirchner, Stuart N. Hoffman, and Jennifer Sartorius
Geisinger Clinic

Richard E. Adams
Kent State University

Research suggests that posttraumatic stress disorder (PTSD) is associated with increased alcohol use, but the findings have not been consistent. We assessed alcohol use, binge drinking, and psychotropic medication use longitudinally in 1,681 New York City adults, representative of the 2000 census, 2 years after the World Trade Center attacks. We found that, with the exception of a modified CAGE Questionnaire index for alcohol, alcohol use showed a modest increase over time and was related to PTSD symptoms, with an increase of about 1 more drink per month for those with PTSD, even though overall levels appeared to be within the National Institute on Alcohol Abuse and Alcoholism's safe range. Psychotropic medication use followed a similar trend; those with PTSD used psychotropics about 20 more days over the past year than those without. Because the study analyses adjusted for key psychosocial variables and confounders, it is not clear if the increased alcohol use following trauma exposure is associated with self-medication of PTSD symptoms, whether increased alcohol use prior to exposure is a risk for delayed-onset PTSD, or whether a third unmeasured variable is involved. Further research is warranted.

Research suggests increases in substance use may be associated with symptoms of posttraumatic stress disorder (PTSD) following exposure to traumatic events (Boscarino, Adams, & Galea, 2006; Grieger, Fullerton, & Ursano, 2003; Pfefferbaum & Doughty, 2001; Stewart, Mitchell, Wright, & Loba, 2004). It has also been suggested that traumatic exposures may be a risk factor for future substance misuse (Chilcoat & Menard, 2003; Ouimette & Brown, 2003). Some evidence suggests that this association is due to the use of alcohol and other substances to ameliorate PTSD symptoms (Boscarino, Adams, & Galea, 2006; Chilcoat & Menard, 2003). Although studies of the association between substance use and exposure to psychological distress have a considerable history (Boscarino, 1981; Gottheil, Druley, Pashko, & Weinstein, 1987; Khantzian, 1990; Linsky, Colby, & Straus, 1991), these findings have not been consistent.

For example, although experimental studies have linked alcohol use to psychological distress, findings from community-based studies have been less consistent (Castaneda, Lifshutz, Galanter, & Franco, 1994; Gottheil et al., 1987). There are studies that suggest that this relationship may include mediating factors (Cerda, Tracy, & Galea, 2011; Cerda, Vlahov, Tracy, & Galea, 2008; North, Ringwalt, Downs, Derzon, & Galvin, 2011), as well as some that suggest a direct association between PTSD and substance misuse (e.g., Kilpatrick et al., 2000).

A recent large-scale national study of 34,635 adults, Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions (Leeies, Pagura, Sareen, & Bolton, 2010), found that approximately 20% of individuals with PTSD may use alcohol and drugs to relieve their symptoms. Men were significantly more likely than women to engage in self-medication. In addition, another recent study suggested that fluctuation in PTSD symptoms are synchronized with alcohol-dependence symptoms and misuse of other substances (Ouimette, Read, Wade, & Tirone, 2010). Because alcohol is readily available in the US and it can be readily misused (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2000), an investigation of the degree to which alcohol problems are related to PTSD symptoms consequent to widespread traumatic exposure from terrorism is timely and potentially valuable.

The present study examines the relationship between alcohol use before and following the World Trade Center (WTC) attacks in New York City and their relationship to PTSD and PTSD

Joseph A. Boscarino, Center for Health Research, Geisinger Clinic; H. Lester Kirchner, Center for Health Research, Geisinger Clinic; Stuart N. Hoffman, Department of Neurology, Geisinger Clinic; Jennifer Sartorius, Center for Health Research, Geisinger Clinic; Richard E. Adams, Department of Sociology, Kent State University.

This research was supported by grants from the National Institute of Mental Health (Grants R01 MH66403 and R21-MH-086317) and the Pennsylvania Department of Health (Contract 4100042573), to Dr. Boscarino.

Correspondence concerning this article should be addressed to Joseph A. Boscarino, Center for Health Research, Geisinger Clinic, 100 N. Academy Avenue, Danville, PA 17822-4400. E-mail: jaboscarino@geisinger.edu.

 DOI: 10.1002/jts.20673

symptoms. Early postdisaster WTC studies documented an increased use of psychoactive substances, including alcohol, cigarettes, and marijuana and linked this use to PTSD (Vlahov et al., 2002, 2004). Previously we reported that greater exposure to the WTC attacks was associated with higher alcohol consumption at 1 year and 2 years after this event (Boscarino, Adams, & Galea, 2006) and we also reported that exposure to the attacks was associated with binge drinking at 1 year, but was not associated with binge drinking measured 2 years after the attacks. Alcohol misuse, as measured using a CAGE Questionnaire for alcohol, also was associated with greater exposure. After adjusting for exposure and other covariates, however, PTSD at Year 1 was not associated with alcohol use. This earlier report suggested that exposure to psychological trauma was associated with increases in problem drinking after exposure; absent was the longitudinal examination from predisaster to postdiaster of those with delayed onset PTSD. This a key focus of the present report.

We expected that delayed-onset PTSD would be associated with both higher alcohol consumption and alcohol misuse up to 2 years after the attacks, independent of other factors such as demographics, history of traumatic exposures, recent stressful life events, and available psychosocial resources. To guide our analytical approach, we used a stress-process model (Adams & Boscarino, 2005). This model suggests that individuals subjected to stressors often respond physiologically through alterations in neuroendocrine functions (Boscarino, 1996), psychologically through alterations in cognitive status (Keane, Zimering, & Caddell, 1985), and behaviorally through, among other things, changes in use of psychoactive substances (Adams, Boscarino, & Galea, 2006a), as well as through help-seeking behaviors (Boscarino, Adams, & Figley, 2011). It is suggested that these stressor events could put individuals at risk for alcohol misuse because some persons may use psychoactive substances to help manage PTSD symptoms (e.g., hyperarousal or sleep disturbance), a phenomenon that has been conceptualized as a self-medication model of substance use (Bolton, Cox, Clara, & Sareen, 2006; Khantzian, 1990). Given recent reports (Cerda et al., 2008; Jacobson et al., 2008), and our previous work cited above describing the level of stress response (Adams, Boscarino, & Galea, 2006a), we did not expect the misuse of alcohol to rise to the level of symptoms of alcohol dependence (e.g., tolerance, withdrawal, etc.). Rather, we looked for the impact in greater frequency of binge drinking and increased alcohol consumption.

METHOD

Participants and Procedures

The data for the present study come from a longitudinal survey of adults aged 18 and older living in New York City (NYC) on the day of the WTC attacks; that is, all respondents lived in NYC and were in the city (or the metropolitan area) during the attack, so they all experienced this exposure. For the baseline survey, using random-digit dialing, we conducted a telephone survey in NYC from October to December 2002. Interviews were conducted in English and Spanish. The follow-up interviews occurred 12 months later, from October 2003 to February 2004. For both surveys, trained interviewers using a computer-assisted telephone interviewing system conducted the interviews.

Overall, 2,368 individuals completed the baseline survey. As part of the study design, we oversampled residents at baseline who reported receiving mental health treatment in the year after the attacks. The baseline survey cooperation rate was 63% (Boscarino, Adams, & Figley, 2004). During follow-up, we reinterviewed 71% of these baseline respondents ($N = 1{,}681$). Sampling weights were developed for each wave to correct for potential selection bias related to the number of telephone numbers and persons per household, and for the oversampling of treatment-seeking respondents. In addition, weights also were used to adjust for slight differences in response rates by different groups (Groves et al., 2004; Kessler, Little, & Groves, 1995). Because younger persons, men, Blacks, and Latinos tended to have somewhat lower follow-up rates, survey weights were developed to correct for this selection bias. After this adjustment, the demographic profile of our study at baseline and follow-up matched the year 2000 census data for NYC (Adams, Boscarino, & Galea, 2006b). The Institutional Review Board (IRB) of Geisinger Clinic (Danville, PA) approved the study and subjects gave informed consent. More detailed information on this survey can be found elsewhere (Adams et al., 2006b; Boscarino et al., 2004; Boscarino, Adams, Stuber, & Galea, 2005; Boscarino & Adams, 2008; Boscarino & Adams, 2009; Boscarino et al., 2011).

Study Measures

During the baseline and follow-up surveys, questions were asked related to mental health symptoms and substance use during the past year. Respondents were also asked to retrospectively report these for the 12 months prior to the attacks, as is often done in the National Comorbidity Study and other mental health studies (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995).

The survey asked about binge drinking and alcohol consumption over various periods. Following a standard for binge drinking (Allen & Columbus, 1995; Naimi et al., 2003; Wechsler & Kuo, 2000), we asked how many times in the past year the respondent had six or more drinks on one occasion and was coded 0 = *never* to 4 = *daily/almost daily*. We used six-plus drinks in our study because we wanted to assess a higher risk use, rather than the lower five-drink threshold typically used (Allen & Columbus, 1995). For alcohol consumption, we asked respondents to report the number of drinks per day they drank in the past month on the days they drank. We also collected data on the number of drinks per month

consumed, which was developed from two survey questions: how many days in the past month the respondents drank and how many drinks they had on the days they drank. These were multiplied to calculate the number of drinks per month. Because this variable was positively skewed, we transformed this measure by taking the log of the original value. We also inquired about alcohol misuse based on the CAGE Questionnaire alcohol scale (Ewing, 1984), a widely used and validated measure (Ewing, 1984; King, 1986; O'Brien, 2008). We modified the standard measure and counted the CAGE Questionnaire symptoms that occurred (e.g., being criticized for drinking, drinking in the morning), which resulted in a continuous modified CAGE index ranging from 0 to 4. This was largely dictated by the fact that relatively few persons in our study endorsed two or more of the four items on the dichotomous CAGE Questionnaire scale (Boscarino, Adams, & Galea, 2006). Cronbach's α ranged from .73 to .70. For all the alcohol measures, nonusers were coded as 0.

We also included questions about psychotropic medication use (Kessler et al., 1999) drawn from the National Comorbidity Survey. Specifically, respondents were asked, "Have you ever taken medications prescribed by a doctor, such as anti-depressants, tranquilizers, sleeping pills or other medicines for problems with your emotions or nerves, problems with concentrating, to help you sleep or cope with stress?" After this, respondents were asked if they took these medications in the past 12 months. If they did, they were asked on how many days they took these medications in the past 12 months, which was recorded as the number of days taking these medications. Nonmedication users were coded as 0. Because these values were positively skewed, as with drinks per month, these values were also log-transformed to aid in multivariate data analyses. As with alcohol use, medication use was collected for three time points: 12 months before, 12 months after, and 24 months after the WTC attacks.

Our analyses included three stressor variables that could have placed individuals at higher risk for alcohol misuse and one psychosocial resource variable that could have lowered this risk. In the baseline survey, we inquired about 14 stressor events that the respondent could have experienced during the WTC attacks. These included being on the WTC campus or in one of the WTC buildings during the attack, seeing people jump from buildings, seeing persons injured/killed, seeing the attack in person or in real time, being forced to evacuate, knowing someone killed or injured in the attack, or losing one's possessions. Because there was no a priori method of assigning the individual severity of these events, we summed these into an exposure scale as follows: 0 or 1 event = *low exposure*; 2–3 = *moderate exposure*; 4–5 = *high exposure*; and 6+ = *very high exposure* for cross-tabular analyses. For multivariate analyses, this variable was used as a continuous variable, trimmed at a value of 6 or higher. A negative life event scale was also used that was the sum of eight experiences that the respondent could have experienced (e.g., divorce, death of spouse, etc.) in the past 12 months at follow-up (Freedy, Kilpatrick, & Resnick, 1993). For cross-tabular analyses, we coded respondents into three groups: no life events, one life event, and two or more life events. For multivariate analyses, we used this measure as a continuous variable, coded from 0 to 5 or more negative life events. The third stressor measure included lifetime traumatic events (Freedy et al., 1993), other than the WTC attacks, which the respondent could have experienced (e.g., forced sexual contact, being attacked with a weapon, etc.). For cross-tabular analyses, these items were collapsed into three categories: 0–1, 2–3, and 4+ traumas. For multivariate analyses, due to variable skewness, this measure was coded into quartiles and used as an ordinal-level continuous variable.

We included a measure of social support (Sherbourne & Stewart, 1991). This measure was the sum of four questions about emotional, informational, and instrumental support (e.g., someone was available to help if you were confined to bed, etc.). Cronbach's α for this scale was .83 based on the data from Boscarino et al. (2004). Based on an examination of the scale results, we coded respondents into approximately three equal subgroups for cross-tabular analyses, representing low, moderate, and high social support. For multivariate analyses, because of skewness, this measure was coded into quintiles and used as a continuous ordinal-level variable.

Our PTSD measure was based on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994). This scale was initially developed for telephone administration (Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) and used in previous national and regional surveys (e.g., Kilpatrick et al., 2003). To be coded as having current PTSD, subjects had to meet *DSM-IV* criteria A through F, but the time frame was based on the past 12-month period, both at baseline and at follow-up. Specifically, for each of the 17 PTSD symptoms, respondents were asked if there had ever been a period of 2 weeks or more during which they were troubled by that symptom. If the response was yes, they were asked if the symptom was related to the WTC or something else. They then indicated the last time they had experienced this symptom, using the past 30 days, 6 months, 12 months, or more than 12 months ago as response categories. Respondents were specifically asked these questions for the WTC disaseter, as well as for up to two other traumatic events identified. As reported elsewhere, Cronbach's α for these PTSD items was .90 (Boscarino et al., 2004). In the current study, we used any PTSD in the past 12 months as our PTSD measure (not just WTC-related PTSD), but this was mostly related to the WTC disaster, given the study design and the impact of this event (Boscarino & Adams, 2009). To have PTSD in the current study, the respondents had to meet the full *DSM-IV* criteria, A through F. For delayed-onset PTSD, respondents had to meet the full PTSD criteria (A through F) at follow-up, while having been PTSD negative for the full criteria at baseline.

In the present study, our primary PTSD assessment included current PTSD at follow-up, which was 24-months postattack. In

addition, we also included a measure of delayed-onset PTSD at follow-up as discussed above.

Because PTSD is often associated with depression (Kessler, Sonnega, et al., 1995; O'Donnell, Creamer, & Pattison, 2004), we included a measure of current depression at follow-up to examine if taking account of depressive symptoms altered our results. As described, this depression measure also was based on the *DSM-IV* diagnostic criteria, designed for telephone surveys, and previously validated (Boscarino et al., 2004; Galea et al., 2002; Kilpatrick et al., 2003).

Demographic variables included age, education, gender, marital status, race, ethnicity, and income. For cross-tabular analyses, age was coded into four categories: 18–29, 30–44, 45–64, and 65+. Education, gender, and marital status were coded as binary variables: noncollege graduate versus college graduate, male versus female, and not married versus married. Race and ethnicity were coded as White versus non-White, African American versus non-African American, and Latino versus non-Latino. Income was coded into four categories: less than $30,000, $30,000–$99,999, $100,000 or more, and "not reported" for cross-tabular analyses. For multivariate analyses, income was coded as a continuous variable (coded 1–7), with the mean value replacing missing data, which was less than 5%.

Statistical Analyses

Descriptive statistics for our main variables pooled across all three time points as well as by PTSD at 24 months and binge drinking status were tabulated. Tests of statistical significance were conducted using χ^2. To assess substance use over time by PTSD status at follow-up, we first used generalized estimating equation (GEE) analyses to predict the alcohol and medication variables, in a regression model that included only PTSD status and a PTSD × time interaction effect. For these analyses, 12 months prior to WTC = 0, 12 months after WTC = 1, and 24 months after WTC = 2. These GEE models assumed a Gaussian distribution and an unstructured correlation matrix (Hardin & Hilbe, 2003; Twisk, 2003).

Based on the descriptive analyses and previous research (Boscarino, Adams, & Galea, 2006), we selected variables for inclusion in multivariate GEE analyses, that controlled for relevant covariates and confounding variables. The outcomes of interest included drinks per drinking day and drinks per month over the past 30 days, as well as binge-drinking scores, CAGE index scores, and psychotropic medication use over the past year. In the multivariate GEE models, we assessed the impact of both PTSD onset and delayed-onset PTSD at 24 months by time, which was also expressed as a PTSD × Time covariate. For all analyses, we used the survey weights described previously. Because drinks per month and psychotropic medication days per year were positively skewed, these were log-transformed. To prevent model overfitting, variables not significant in any of the models were eliminated from the regression analyses. Statistical analyses were performed using Stata version 11.2 (Stata Corp., 2011).

RESULTS

The descriptive data for all observations, pooled across all time points, were these: (a) binge drinking ($M = 0.54$, $SD = 1.09$), (b) number of drinks per drinking day past month ($M = 1.10$, $SD = 2.05$), (c) number of drinks per month ($M = 7.87$, $SD = 16.86$), (d) modified CAGE Questionnaire ($M = 0.07$, $SD = 0.33$), (e) number of medication days past 12 months ($M = 19.45$, $SD = 57.50$), (f) WTC attack events ($M = 2.02$, $SD = 1.61$), (g) negative life events ($M = 0.53$, $SD = 0.96$), (h) lifetime trauma exposure ($M = 1.26$, $SD = 1.39$), and (i) social support ($M = 2.19$, $SD = 1.84$). For PTSD at 24 months, the point estimate was 5.4% and a 95% CI [4.3, 6.7]; for delayed-onset PTSD at 24 months, the point estimate was 4.4% and a 95%CI [3.4, 5.7].

Table 1 presents study predictor variables for PTSD at follow-up and binge drinking at any time. PTSD at follow-up was associated with demographics such as age, income, and race, exposure to the three trauma variables, and social support level. For binge drinking at any of the three time points there were fewer predictors: being male, age, being Latino, and lifetime trauma and WTC attack exposure.

The means and standard deviations of alcohol and medication use by PTSD at 24 months are presented in Table 2. The GEE analyses to assess these trends over time, by PTSD status at follow-up are presented in Table 3. These analyses revealed increased alcohol use over time for those with PTSD at follow-up for binge drinking and for drinks per drinking day. Psychotropic medication use also showed an effect of time, but drinks per month and the modified CAGE index did not.

The adjusted multivariate GEE results predicting alcohol and medication use longitudinally are presented in Tables 4–6. For brevity, we only discuss the PTSD results; the other predictors in the models were principally used as control variables. The PTSD × Time effect was significant for drinks per drinking day for both PTSD at follow-up and delayed-onset PTSD. The association for drinks per month was not statistically significant for PTSD, but was significant for delayed-onset PTSD (see Table 4). The PTSD × Time effects for both PTSD at follow-up and delayed-onset PTSD were significant for binge drinking. The b coefficients for drinks per drinking day, drinks per month, and binge drinking were all positive, indicating that each respective alcohol outcome assessed in these GEE models increased over time for those with later or delayed-onset PTSD, compared to those without. In addition, the GEE results for psychotropic medication use were positive and significant for PTSD at 24 months and delayed-onset PTSD, suggesting that psychotropic medication use also increased

Table 1. PTSD at 24 Months, Binge Drinking, and Total Sample Characteristics

	PTSD at 24 months		Any binge drinking		Total sample	
Variable	*n*	Weighted %[a]	*n*	Weighted %[a]	*n*	Weighted%[a]
Gender						
Male	49	5.0	327	48.2***	693	46.2
Female	85	5.7	260	26.8	988	53.8
Age						
18–29	12	1.9***	142	51.3***	284	22.7
30–44	56	8.0	257	42.5	596	32.9
45–64	61	6.7	152	26.6	586	32.5
65+	5	1.0	36	20.5	215	11.9
Income						
< $30,000	73	8.2**	201	35.0	599	33.8
$30,000–$99,999	45	3.4	287	37.9	782	46.5
$100,000+	15	6.0	81	36.8	247	16.1
Not reported	1	1.0	18	36.1	53	3.6
Married						
No	90	6.1	341	37.0	972	49.7
Yes	44	4.6	246	36.4	709	50.3
Education						
Noncollege graduate	87	5.9	330	36.9	906	58.3
College graduate	47	4.6	257	36.4	775	41.7
White						
No	94	6.7**	343	39.1	899	57.1
Yes	40	3.6	244	33.5	782	43.0
Black						
No	103	5.7	439	37.4	1,259	74.0
Yes	31	4.4	148	34.8	422	26.0
Latino						
No	80	3.9***	432	34.3**	1,314	75.9
Yes	54	9.9	155	44.3	367	24.1
Events during/after WTC attacks						
0–1	14	2.7***	100	29.0**	362	26.7
2–3	47	4.3	255	37.5	719	43.9
4–5	40	7.2	156	42.5	416	21.8
6+	33	15.6	76	42.7	184	7.6
Negative life events						
0	42	2.9***	332	35.0	991	63.3
1	30	4.2	151	38.4	429	24.7
2+	62	20.5	104	42.2	261	12.0
Lifetime traumatic events						
0–1	47	3.4***	274	32.9**	866	57.0
2–3	37	5.8	170	39.1	484	26.8
4+	50	11.4	143	46.2	331	16.2
Social support						
Low	70	7.5*	210	37.8	601	36.0
Moderate	43	4.5	236	36.8	655	37.9
High	21	3.8	141	35.2	425	26.2

(Continued)

Table 1. Continued

	PTSD at 24 months		Any binge drinking		Total sample	
Variable	*n*	Weighted %[a]	*n*	Weighted %[a]	*n*	Weighted%[a]
PTSD at follow-up						
No	–	–	529	36.4	1,547	94.6
Yes	–	–	58	42.7	134	5.4
PTSD: delayed onset						
No	36	1.0***	544	36.4	1,583	95.6
Yes	98	100.0	43	43.9	98	4.4

Note. $N = 1{,}681$. PTSD = posttraumatic stress disorder; WTC = World Trade Center. Any binge drinking tabulates binge drinking reported during the 12 months before the World Trade Center attacks, or during the 12 months after the attack, or during the 13-24 months after.
[a]Weighted percentages shown are based on row percentage for positive status of PTSD or binge drinking; percentages for those with negative status are not given; *n* values are unweighted.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2. Alcohol and Psychotropic Medication Use at 3 Time Points Relative to WTC Attacks by PTSD Status at 24 Months

	No PTSD at 24 months ($n = 1{,}547$)		PTSD at 24 months ($n = 134$)	
Variable	*M*	*SD*	*M*	*SD*
Binge drinking during past year				
12 months before	0.53	0.06	0.70	0.23
12 months after	0.55	0.06	0.80	0.24
24 months after	0.51	0.06	0.87	0.26
Drinks per drinking day past month				
12 months before	0.94	0.10	0.95	0.44
12 months after	1.04	0.11	1.49	0.56
24-months after	1.28	0.15	1.85	0.70
Drinks per month during past month				
12 months before	7.11	0.96	7.07	3.19
12 months after	7.54	0.96	9.68	3.62
24 months after	8.78	1.02	10.01	3.10
Modified CAGE Questionnaire past year				
12 months before	0.04	0.39	0.10	1.79
12 months after	0.08	0.50	0.23	2.19
24 months after	0.07	0.48	0.22	2.30
Psychotropic use days past year				
12 months before	14.58	35.03	50.42	248.46
12 months after	13.73	33.52	77.54	309.96
24 months after	20.35	47.64	101.60	371.17

Note. WTC = World Trade Center; PTSD = posttraumatic stress disorder.

over time for those with either later or delayed-onset PTSD (see Table 6).

When PTSD at 1 year after the WTC attacks was used as a covariate in the model for all dependent variables; it was significant only in the medication use model. In addition, including overall psychotropic medication use in the alcohol models, and conversely, overall alcohol use in the medication models did not affect the final results (results not presented). We also examined the correlations between our alcohol and the medication use measures, but these tended to be only weakly correlated (r ranged from .01 to .076). For the 79 subjects whose PTSD had remitted at follow-up, there was a slight but significant decrease in drinks per month compared

Table 3. Means, Standard Errors, and Unadjusted GEE Coefficients of Alcohol and Psychotropic Medication Use at 3 Time Points for PTSD at 24 Months

	12 Months before WTC		12 Months after WTC		24 Months after WTC		PTSD × Time		
Variables	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*	b	*SE*	*p*
Binge drinking									
No PTSD	0.53	0.03	0.55	0.03	0.51	0.03	–	–	–
PTSD	0.70	0.12	0.80	0.12	0.87	0.13	.09	.04	.018
Drinks/drinking day									
No PTSD	0.94	0.05	1.04	0.06	1.28	0.07	–	–	–
PTSD	0.95	0.22	1.49	0.28	1.85	0.36	.29	.13	.023
Drinks/month[a]									
No PTSD	7.11	0.49	7.54	0.49	8.78	0.52	–	–	–
PTSD	7.07	1.63	9.68	1.85	10.01	1.58	.13	.07	.066
Modified CAGE Questionnaire									
No PTSD	0.04	0.01	0.08	0.01	0.07	0.01	–	–	–
PTSD	0.11	0.04	0.23	0.05	0.22	0.06	.04	.03	.195
Medication days[a]									
No PTSD	14.58	0.85	13.73	0.82	20.35	1.16	–	–	–
PTSD	50.42	6.06	77.54	7.56	101.60	9.07	.39	.12	.001

Note. $N = 1{,}681$. GEE = Generalized estimating equation; PTSD = posttraumatic stress disorder; WTC = World Trade Center.
[a]Variables were log-transformed for GEE analyses.

Table 4. GEE Coefficients for Drinks per Drinking Day and Drinks per Month for PTSD and Delayed PTSD Onset at 24 Months

	Drinks per drinking day				Drinks per month[a]			
	PTSD		Delayed PTSD		PTSD		Delayed PTSD	
Variables	b	*SE*	b	*SE*	b	*SE*	b	*SE*
Age (years)	−.01	.003***	−.01	.003***	−.005	.002*	−.004	.002*
Female	−.75	.10***	−.75	.10***	−.53	.07***	−.52	.07***
Income	.07	.03**	.07	.03**	.10	.02***	.10	.02***
Married	−.22	.10*	−.22	.10*	−.20	.07**	−.20	.07**
White	.48	.10***	.48	.10***	.52	.08***	.52	.08***
Latino	.43	.14**	.43	.14**	.10	.08	.10	.08
WTC attack events	.06	.04	.06	.04	.06	.03*	.06	.03*
Lifetime trauma	.12	.05**	.12	.04**	.08	.03*	.08	.03*
Negative life events	−.07	.05	−.07	.05	−.04	.04	−.04	.04
PTSD	.04	.26	.04	.28	.05	.15	.10	.17
Time period	.16	.04***	.17	.03***	.13	.02***	.13	.02***
PTSD × Time period	.29	.13*	.33	.15*	.13	.07	.16	.08*
Constant	1.19	0.22***	1.18	0.22***	0.69	0.15***	0.69	0.15***

Note. $N = 1{,}681$. GEE = Generalized estimating equation; PTSD = posttraumatic stress disorder; WTC = World Trade Center.
[a]Log transformed for GEE analyses.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 5. GEE Coefficients for Binge Drinking and Modified CAGE Questionnaire for PTSD and Delayed-Onset PTSD at 24 Months

	Binge drinking				Modified CAGE Questionnaire			
	PTSD		Delayed PTSD		PTSD		Delayed PTSD	
Variables	b	*SE*	b	*SE*	b	*SE*	b	*SE*
Age (years)	−.01	.002***	−.01	.002***	−.001	.001*	−.001	.001*
Female	−.43	.05***	−.43	.05***	−.06	.01***	−.06	.01***
Income	−.02	.01	−.02	.01	−.005	.004	−.005	.004
Married	−.05	.05	−.05	.05	−.03	.02	−.03	.02
White	.03	.06	.03	.06	−.01	.02	−.01	.02
Latino	.15	.07	.14	.07	.01	.02	.01	.02
WTC attack events	.04	.02	.04	.02	.02	.01*	.02	.01*
Lifetime trauma	.04	.02	.04	.02	.02	.01*	.02	.01*
Negative life events	.04	.03	.04	.03	.01	.01	.02	.01
PTSD	.05	.13	.11	.14	.02	.05	.02	.05
Time period	−.01	.01	−.01	−.01	.01	−.01	.01	.01*
PTSD × Time period	.09	.04*	.09	.04*	.04	.03	.05	.04
Constant	1.03	0.12***	1.03	0.12***	0.09	0.04**	0.09	0.04*

Note. $N = 1{,}681$. GEE = Generalized estimating equation; PTSD = posttraumatic stress disorder; WTC = World Trade Center.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 6. GEE Coefficients for Psychotropic Medication Days for PTSD and Delayed PTSD Onset at 24 Months

	PTSD		Delayed PTSD	
Variables	b	*SE*	b	*SE*
Age (years)	.01	.001***	.01	.001***
Female	.13	.05*	.14	.05*
Income	−.04	.02**	−.04	.02**
Married	−.22	.05***	−.22	.05***
White	.31	.06***	.31	.06***
Latino	.17	.07*	.18	.07*
WTC attack events	.01	.02	.02	.02
Lifetime trauma	.10	.02***	.10	.02***
Negative life events	.14	.04**	.16	.04***
PTSD	.61	.21**	.53	.24*
Time period	.05	.02**	.06	.02**
PTSD × Time period	.39	.12**	.34	.13**
Constant	−0.20	0.10*	−0.23	0.10*

Note. $N = 1{,}681$. GEE = Generalized estimating equation; PTSD = posttraumatic stress disorder; WTC = World Trade Center.
* $p < .05$. ** $p < .01$. *** $p < .001$.

to those in the nonremitted group, $t(1{,}679) = 1.71$, $p = .046$ (one-tailed test). In addition, including depression status in all the multivariate GEE models did not affect the findings (results not shown).

DISCUSSION

We examined whether PTSD and potentially delayed-onset PTSD would be related to alcohol use up to 2 years after the WTC

attacks. We anticipated detecting a population increase in alcohol use over time, consistent with a self-medication model of PTSD symptom management (Bolton et al., 2006; Stewart, 1996). As seen, our results are consistent with such a formulation. We found that alcohol consumption and binge drinking were associated with PTSD onset at follow-up. This finding is consistent with previous reports suggesting that PTSD victims may use alcohol to cope with their posttrauma mental health status (Jacobson et al., 2008), which would be consistent with Khantzian's model of substance misuse (Khantzian, 1990).

Our results showed that alcohol use tended to modestly increase over time from before the WTC until study follow-up at 24 months, but only among those with later or delayed-onset PTSD. Posttraumatic stress disorder at baseline was not associated with alcohol use over time, as previously reported (Boscarino, Adams, & Galea, 2006). Because this study adjusted for confounders and other key factors, including race/ethnicity, socioeconomic factors, trauma exposures, and negative life events, our results are consistent with Khantzian's hypothesis that those with increased alcohol use may be using this substance to regulate symptoms. As reported, PTSD onset was also associated with a statistically significant population-level increase in psychotropic medication use. Furthermore, although this subgroup is small ($n = 79$), those with remitted PTSD at follow-up also tended to have lower alcohol use over time, also consistent with the self-medication hypothesis. The addition of a measure of depression in our GEE models as a final step did not change our results, suggesting that it is PTSD symptoms that are implicated. The absolute level of alcohol used in our sample, however, appears to be within the range that the NIAAA (2011) deems to be safe—up to two drinks per day for men, and one for women and older people.

This study has a number of limitations. First, by omitting individuals without telephones and those who did not speak either English or Spanish, we may have missed vulnerable individuals, but because our sample matched the 2000 Census, this exclusion did not appear to introduce significant demographic bias (Adams et al., 2006b). Nevertheless, we are limited in generalizations about the association between alcohol use and PTSD with respect to groups not in our sample. Second, our conclusions are limited by the retrospective nature of our predisaster data, though such self-reported data are often used in public health surveys and have been shown to be accurate (Edwards et al., 1994). Moreover, given the salience of the attacks, there is a better chance of participants being aware of changes, even though we corrected for response bias using survey weights (Adams et al., 2006b), our GEE analyses may still be biased. Third, the measures of alcohol and medication use in our study were limited. In particular, the use of the modified CAGE Questionnaire as a continuous measure makes comparison with other studies problematic. Fourth, not all of the cases of PTSD were in response to the WTC attacks, so there may be some overestimate of the impact of the WTC. Fifth, our data do not allow us to study changes in alcohol use over a longer period. Sixth, our focus on only alcohol consumption and psychotropic medication excludes the role of illicit drug use. Finally, in those categorized as having delayed-onset PTSD, we were unable to distinguish between those who might have been relatively symptom free from those who might have had substantial but subthreshold symptoms.

The sizes of the effects we found are relatively small. For example, the multivariate results for drinks per drinking day indicated that, compared to before the attacks, at follow-up those with PTSD tended to consume about half a drink more per drinking day than those without PTSD. Finally, although our findings are consistent with self-medication as a method to manage symptoms, we cannot rule out other explanations for the relationships because we did not directly query this. One possible explanation for the increase could be the ongoing threat of terrorist attacks at the time of follow-up or ongoing financial difficulties from the economic disruption following the attacks.

These limitations, however, should not overshadow the strengths of the study. These include the use of a large representative sample of adults, the assessment of alcohol use and PTSD using standard measurements, and the study of a well-defined traumatic event. Our study suggests a link between population-level increases in problematic drinking and the onset of PTSD up to 2 years after exposure to a traumatic event. Increased alcohol use in the context of PTSD onset is typically considered harmful and clinically advised against (Benedek & Wynn, 2011). Noteworthy is that recent research has suggested that those who received emergency crisis counseling after the WTC attacks, not only had better PTSD outcomes, but also better outcomes in a number of clinical areas, including alcohol misuse (Boscarino et al., 2011). This finding tends to reinforce the findings reported here.

Additional research is warranted to further illuminate the link between PTSD onset and alcohol misuse reported in the trauma literature. If the self-medication finding is further validated in other studies, then substance misuse screening, surveillance, and treatment might need to become the standard of care among those with a history of traumatic stress exposures.

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