

# Posttraumatic Stress Disorder and the Incidence of Nicotine, Alcohol, and Other Drug Disorders in Persons Who Have Experienced Trauma

Naomi Breslau, PhD; Glenn C. Davis, MD; Lonni R. Schultz, PhD

**Background:** We examine whether exposure to traumatic events increases the risk for nicotine dependence or alcohol or other drug use disorders, independent of posttraumatic stress disorder (PTSD).

**Methods:** Data come from a longitudinal epidemiologic study of young adults in southeast Michigan. Prospective data covering a 10-year period and retrospective lifetime data gathered at baseline were used to estimate the risk for onset of substance use disorders in persons with PTSD and in persons exposed to trauma without PTSD, compared with persons who have not been exposed to trauma. The National Institute of Mental Health Diagnostic Interview Schedule for *DSM-III-R* was used. Logistic regression was used to analyze the prospective data, and Cox proportional hazards survival analysis with time-dependent variables was applied to the lifetime data.

**Results:** The prospective and retrospective data show an increased risk for the onset of nicotine dependence

and drug abuse or dependence in persons with PTSD, but no increased risk or a significantly ( $P = .004$ ) lower risk (for nicotine dependence, in the prospective data) in persons exposed to trauma in the absence of PTSD, compared with unexposed persons. Exposure to trauma in either the presence or the absence of PTSD did not predict alcohol abuse or dependence.

**Conclusions:** The findings do not support the hypothesis that exposure to traumatic events per se increases the risk for substance use disorders. A modestly elevated risk for nicotine dependence might be an exception. Posttraumatic stress disorder might be a causal risk factor for nicotine and drug use disorders or, alternatively, the co-occurrence of PTSD and these disorders might be influenced by shared risk factors other than traumatic exposure.

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From the Departments of Psychiatry (Dr Breslau) and Biostatistics and Research Epidemiology (Drs Breslau and Schultz), Henry Ford Health System, Detroit, Mich; Department of Psychiatry, Case Western Reserve University School of Medicine, Cleveland, Ohio (Dr Breslau); Department of Psychiatry, University of Michigan School of Medicine, Ann Arbor (Dr Breslau); and Office of the Dean, Department of Psychiatry, Michigan State University College of Human Medicine, East Lansing (Dr Davis).

**A**SSOCIATIONS BETWEEN posttraumatic stress disorder (PTSD) and substance use disorders have been reported in studies<sup>1-11</sup> of Vietnam veterans and civilian samples. Little is known about the causal pathways that might explain these associations. Alternative explanations have been proposed.<sup>7</sup> First, substance use disorders increase the likelihood of PTSD either by their association with lifestyles that involve an elevated risk of exposure to traumatic events that induce PTSD or by increasing persons' susceptibility to the PTSD-inducing effects of trauma. Second, PTSD is a causal risk factor for substance use disorders, when substances are used to relieve distressing symptoms of PTSD. Third, the association of substance use disorders with PTSD might be noncausal, reflecting shared genetic or environmental factors. Genetic factors common to PTSD, alcohol consumption or disorder, and other drug disorders have been reported.<sup>12,13</sup> A suspected shared environ-

mental risk factor in the PTSD–substance use disorders comorbidity is the exposure to trauma.<sup>1,14</sup> It has been suggested that traumatic events that lead to PTSD also induce diatheses for other disorders.<sup>15</sup> According to this hypothesis, some persons who experience trauma develop PTSD, whereas others develop other disorders, depending on the persons' vulnerabilities; comorbidity of PTSD with other disorders would, thus, reflect the co-occurrence of distinct diatheses.<sup>15,16</sup>

This report uses data from a longitudinal study of young adults in southeast Michigan to examine the potential role of exposure to trauma as a shared risk factor in the PTSD–substance use disorder connection. The PTSD–drug abuse or dependence (A/D) relationship has previously been described, based on combined data from baseline and the 5-year follow-up.<sup>17</sup> This report extends the investigation beyond drug A/D and includes alcohol A/D and nicotine dependence, using prospective data that cover a

10-year follow-up period. The hypothesis that traumatic experiences increase the risk of substance use disorders independent of their PTSD-inducing effects would be supported by evidence of a higher incidence of substance use disorders in persons exposed to trauma who did not develop PTSD, compared with persons who were not exposed to trauma. Conversely, evidence of an increased risk of substance use disorders in exposed persons with PTSD but not in exposed persons without PTSD would not support that hypothesis. It would suggest, instead, that PTSD might cause substance use disorders or that shared antecedent factors other than the exposure to trauma account for the PTSD–substance use disorders sequence.

## METHODS

### SAMPLE

A sample of 1200 persons were randomly selected from all 21- to 30-year-old members of a large health maintenance organization in southeast Michigan. Personal interviews were conducted first in 1989 with 1007 persons (83.9%). Follow-up interviews were conducted in 1992, 1994, and 1999 to 2001. Complete data from all assessments are available for 899 persons, 89.3% of the initial sample and 90.1% of surviving respondents. (Information on the sample and the population was previously reported.<sup>6,17</sup>)

### ASSESSMENT

The National Institute of Mental Health Diagnostic Interview Schedule (DIS)<sup>18</sup> for *DSM-III-R* was used to diagnose PTSD and substance use disorders. The baseline interview inquired about lifetime history of disorders, and each follow-up assessment inquired about disorders occurring during the interval since the previous assessment. The diagnosis of PTSD in *DSM-III-R* requires exposure to a qualifying traumatic event and the presence of PTSD criterion symptoms that are linked to the traumatic event. The DIS opens with a question that lists typical PTSD-level events, using the examples in the *DSM-III-R*. A respondent's report of an event that does not fit the *DSM-III-R* stressor criterion (eg, divorce or loss of a job) is excluded from further inquiry, and respondents are asked whether they have experienced any other event of the sort described in the question. In persons who report 2 or more traumatic events, the DIS inquires first about PTSD criterion symptoms in relation to an event designated by the respondent as the worst event and continues up to 3 events.<sup>18</sup> Two studies<sup>8,19</sup> reported high concordance between the diagnosis of PTSD by lay interviewers using structured interviews based on the DIS and independent clinical reinterviews. The small inaccuracy in the first study<sup>8</sup> was the tendency of the structured interview to underdiagnose PTSD, whereas in the second,<sup>19</sup> it was the interview's tendency to overdiagnose PTSD compared with the clinical assessment.

The *DSM-III-R* applies a single definition across substance use disorders. Dependence is defined by the presence of 3 or more symptoms from a list of 9 covering cognitive, behavioral, and physiological problems that characterize compulsive use of a psychoactive substance. The DIS ascertains drug A/D for the following classes of drugs used on one's own to get high: stimulants, sedatives or tranquilizers, marijuana, cocaine, heroin or other opiates, and hallucinogens. Prescribed psychoactive drug A/D was based on symptoms of A/D due to the following: (1) extramedical use of 3 classes of drugs that often are prescribed stimulants (eg, amphetamines), sedatives, and tranquilizers (eg, barbiturates, sleeping pills, Seconal, Valium, Librium, Quaalude, and

Xanax) and opiates other than heroin (eg, codeine, Demerol, morphine, Percodan, methadone, Darvon, and Dilaudid); and (2) use as prescribed of a tranquilizer, sedative, pain pill, antidepressant, or headache medicine every day for 2 weeks or more. The second type of prescribed drug A/D defines cases of A/D despite adherence to medical instructions. Nicotine dependence in the National Institute of Mental Health DIS requires 3 symptoms from a list of 7. The 2 criterion symptoms excluded from the DIS diagnosis of nicotine dependence are (1) a great deal of time spent in activities necessary to get the substance and (2) frequent intoxication or withdrawal symptoms when expected to fulfill major role obligations. The exclusion of these symptoms from the assessment of nicotine dependence in the DIS is based on observations that they do not characterize tobacco dependence. Substance abuse is a residual category, defined by continued use despite knowledge of health, psychological, or social problems caused by the substance or recurrent use in situations in which use is hazardous. In the *DSM-III-R* (and the *DSM-IV*), there is no category of nicotine abuse.

### STATISTICAL ANALYSIS

Prospective data from follow-up interviews at 3, 5, and 10 years after baseline were combined to yield a 10-year cumulative incidence of substance use disorders. We compared the 10-year incidence of nicotine dependence and alcohol and other drug A/D in persons classified according to their baseline history of the following: (1) PTSD; (2) exposure to trauma only, without PTSD; and (3) no exposure to trauma. To identify incidence cases (ie, the first onset) of each substance use disorder, persons with a history of the specific disorder at baseline were excluded as not at risk for the incidence of that disorder. Logistic regression was used to calculate odds ratios for the onset of each disorder in persons with a baseline history of PTSD and in persons with a history of exposure to trauma only, using persons with no history of exposure to trauma as the reference. All estimates were adjusted for sex, race, and educational level.

We also examined the role of exposure to trauma and PTSD as risk factors for the onset of substance use disorders in the lifetime data gathered at baseline, using Cox proportional hazards models with time-dependent covariates.<sup>20-22</sup> While the prospective data provide information on the incidence of substance use disorders during a 10-year period in adulthood, the baseline data cover the lifetime history of the respondents up to the initial interview, when they were aged 21 to 30 years. For each substance use disorder, we used 2 time-dependent covariates, representing the 3 categories of the independent variable (ie, exposed to trauma–PTSD and exposed to trauma–no PTSD), with not exposed to trauma as the reference. The model yields estimates of the risk for the onset of the disorder in persons with prior PTSD and in persons exposed to trauma without PTSD, compared with persons who were not exposed to trauma. SAS statistical software for proportional hazards regression was used.<sup>23</sup> Survival models with time-dependent covariates allow consideration of independent variables whose value for any given person may change over time. For example, a person's status can change from no exposure to trauma to exposure to trauma only or to exposure to trauma plus PTSD at any time until the onset of the other disorder (eg, drug A/D) or age at interview (whichever comes first). All models included sex, race, and educational level as covariates.

## RESULTS

### CHARACTERISTICS OF THE SAMPLE

**Table 1** describes the composition of the sample with complete data (n=899) at baseline by sex, race, educational level,

and marital status. The sample comprised 63.0% female and 81.2% white subjects. These proportions, and the sample's distribution by educational level and marital status, reflect the characteristics of the total sample of 1007 respondents interviewed at baseline, as would be expected given the high follow-up completion rate.

Of the 899 respondents with complete data, 40.0% had been exposed to 1 or more *DSM-III-R* traumatic events up to the time of the baseline interview. Of those exposed, 23.6% met the *DSM-III-R* criteria for PTSD. The lifetime prevalence of nicotine dependence at baseline was 19.9%; alcohol A/D, 21.4%; and drug A/D, 11.8%.

#### CUMULATIVE INCIDENCE OF NICOTINE DEPENDENCE, ALCOHOL A/D, AND DRUG A/D DURING FOLLOW-UP

During the 10-year follow-up since baseline, the number of new cases of nicotine dependence was 108, 15.0% of respondents with no history of nicotine dependence at baseline. The number of new cases of alcohol A/D was 98, 13.9% of those with no history of alcohol A/D at baseline. The number of new cases of other drug A/D was 25, 3.2% of those with no history of other drug A/D at baseline.

The 10-year cumulative incidences, and adjusted odds ratios, of each substance use disorder in persons with a baseline history of PTSD, exposure to trauma only, and no exposure to trauma are presented in **Table 2**. The risk for nicotine dependence was increased significantly in persons with PTSD and in persons with exposure to trauma only, compared with persons with no history of exposure to trauma; the risk in persons with PTSD was significantly higher than in persons with exposure to trauma only ( $P = .04$ ). The risk for alcohol A/D was not significantly elevated in either persons with PTSD or persons exposed to trauma in the absence of PTSD, compared with persons who were not exposed to trauma. The risk for other drug A/D was increased significantly in persons with PTSD, but not in persons who were exposed to trauma but did not develop PTSD, compared with persons who were not exposed to trauma. The risk of other drug A/D in the group with PTSD was significantly higher than in the group exposed to trauma only ( $P = .004$ ). Sex-by-group interactions were tested in each analysis, but none was detected ( $\alpha = .15$ ).

The observed associations of substance use disorders with sex, race, and educational level, variables controlled in these analyses, were in accord with previous findings.<sup>24,25</sup> Men were at a higher risk than women for alcohol and other drug A/D, but not for nicotine dependence. White persons were at a higher risk than black persons for nicotine dependence and alcohol A/D; the estimated risk for other drug A/D was also higher in white vs black persons, but the odds ratio had a wide confidence interval (CI) that included the null value of 1. Persons with less than a college education had a higher risk for nicotine dependence and other drug A/D, but not for alcohol A/D, compared with persons who completed college.

These analyses were repeated, controlling for history of other substance use disorders at baseline. The results showed only small reductions in the estimates in Table 2. The odds ratio for nicotine dependence during

**Table 1. Sample Characteristics at Baseline**

Characteristic	% of the Sample (n = 899)*
Sex	
Male	37.0
Female	63.0
Race	
White	81.2
Black	18.8
Educational level	
<High school	3.6
High school	20.8
Some college	46.3
College	29.4
Marital status	
Married	45.9
Separated or divorced	5.5
Never married	48.5

\*Percentages may not total 100 because of rounding.

the 10-year follow-up, adjusted in addition for preexisting alcohol or other drug A/D, in persons with PTSD was 3.30 (95% CI, 1.69-6.42), and in persons exposed to trauma without PTSD, 1.81 (95% CI, 1.12-2.92), compared with persons with no exposure to trauma. The adjusted odds ratio for alcohol A/D in persons with PTSD was 1.19 (95% CI, 0.53-2.64), and in persons with exposure to trauma only, 1.06 (95% CI, 0.65-1.73). The adjusted odds ratio for other drug A/D in persons with PTSD was 3.36 (95% CI, 1.24-9.12), and in persons with a history of exposure to trauma only, 0.65 (95% CI, 0.22-1.89).

Other results of interest include the observation that history of substance use disorder at baseline increased the risk for the first onset of a new substance use disorder during the 10-year follow-up. Specifically, the odds ratio for the onset of nicotine dependence associated with history of alcohol or other drug A/D was 2.53 (95% CI, 1.55-4.11), the odds ratio for the onset of alcohol A/D associated with history of nicotine dependence or other drug A/D was 2.46 (95% CI, 1.52-4.00), and the odds ratio for the onset of other drug A/D associated with history of nicotine dependence or alcohol A/D was 3.40 (95% CI, 1.41-8.24).

#### RESULTS FROM THE RETROSPECTIVE LIFETIME DATA

By using lifetime data gathered at baseline, when the respondents were aged 21 to 30 years, and applying Cox proportional hazards models with time-dependent variables, we estimated the risk for the subsequent onset of a substance use disorder associated with PTSD and exposure to trauma without PTSD (**Table 3**). Posttraumatic stress disorder predicted the subsequent onset of nicotine dependence and other drug A/D, whereas exposure to trauma without PTSD did not predict the onset of these disorders. Neither PTSD nor exposure to trauma in the absence of PTSD predicted the subsequent onset of alcohol A/D. A significant ( $P = .02$ ) interaction was detected between sex and exposure to trauma without PTSD, with an increased risk for alcohol A/D in

**Table 2. Ten-Year Incidence of Substance Use Disorders and AORs in the 899 Persons in the 3 Groups Studied\***

Baseline Status	Nicotine Dependence		Alcohol A/D		Other Drug A/D	
	Incidence, %	AOR (95% CI)	Incidence, %	AOR (95% CI)	Incidence, %	AOR (95% CI)
PTSD	31.7	4.03 (2.10-7.72)	15.8	1.45 (0.67-3.17)	10.6	4.34 (1.63-11.53)
Exposed to trauma and no PTSD	19.9	1.95 (1.22-3.12)	15.6	1.14 (0.71-1.85)	2.2	0.72 (0.25-2.05)
Not exposed to trauma	10.5	...	12.8	...	2.6	...

Abbreviations: A/D, abuse or dependence; AOR, adjusted odds ratio; CI, confidence interval; ellipses, data not applicable (reference category); PTSD, posttraumatic stress disorder.

\*For each disorder, an analysis was conducted on a subset with no history of the specific disorder at baseline. The AORs were adjusted for sex, race, and educational level from logistic regression analyses.

**Table 3. The AHRs for Subsequent Substance Use Disorders Associated With Prior PTSD and Exposure to Trauma Only in Lifetime Data Gathered for 1007 Persons at Baseline\***

Variable	AHR (95% CI)		
	Nicotine Dependence	Alcohol A/D	Other Drug A/D
PTSD	1.83 (1.04-3.22)	1.72 (0.86-3.45)	3.53 (1.77-7.02)
Exposed to trauma and no PTSD	1.24 (0.84-1.83)	1.33 (0.90-1.96)	1.44 (0.87-2.39)

Abbreviations: A/D, abuse or dependence; AHR, adjusted hazards ratio; CI, confidence interval; PTSD, posttraumatic stress disorder.

\*The AHRs were adjusted for sex, race, and educational level from Cox proportional hazards models with time-dependent covariates.

exposed women, but not in exposed men, compared with same-sex persons with no history of exposure to trauma. Specifically, the hazards ratio for alcohol A/D in exposed women without PTSD was 2.21 (95% CI, 1.20-3.92), and in men, 1.00 (95% CI, 0.65-1.73). No other interactions with sex were detected. Controlling for other preexisting substance use disorders resulted in little change in the estimates displayed in Table 3.

#### COMMENT

Results from the 10-year prospective data show the following. (1) In the case of nicotine dependence, exposure to trauma predicted subsequent onset, but the risk was significantly higher in the presence of PTSD than in the absence of PTSD. (2) In the case of alcohol A/D, exposure to trauma did not predict subsequent onset in either the presence or the absence of PTSD. (3) In the case of other drug A/D, exposure to trauma predicted subsequent onset only in the presence of PTSD.

The results from the lifetime retrospective data are in accord with the results from the prospective data with 2 exceptions. One exception is the significantly increased risk for nicotine dependence associated with exposure to trauma in the absence of PTSD, observed in the prospective data but not in the retrospective data. The other exception is the significant sex-by-exposure only interaction in relation to alcohol A/D, with exposed women (but not men) displaying an increased risk for alcohol A/D in the retrospective, but not the prospective, data.

The results should be interpreted in light of the following methodologic considerations. First, the lifetime data on which some of the analyses are based come from retrospective reports and are, thus, subject to recall error. However, an important strength of the study is the

availability of prospective data. These prospective data have the added advantage that they are derived from several follow-up interviews, each covering a relatively short period, assuring better recall than if a single 10-year follow-up interview were conducted. The 2 analyses complement each other in that each covers a different period in the respondents' lives. The retrospective data cover the respondents' lifetime up to the baseline assessment, when they were aged 21 to 30 years. The prospective data cover the 10 years since baseline, with the last follow-up assessment conducted when the respondents were aged 31 to 40 years. Second, the use of structured interviews for diagnosing PTSD might raise concerns about the validity of the PTSD diagnosis. Evaluations of PTSD by structured interviews reported high concordance with clinical reinterviews.<sup>8,19</sup> The performance of structured interviews in ascertaining an accurate history of exposure to traumatic events has not been evaluated. Structured interviews offer at least 1 clear advantage in testing causal pathways between postulated risk factors and outcomes. Clinicians' diagnosis of PTSD and other disorders might be influenced by the information they elicit about subjects' histories, including history of traumatic experiences. Such contamination is far less likely to occur when structured interviews are used.

Our finding of no increased risk of subsequent alcohol A/D in persons exposed to trauma with or without PTSD is at variance with previous results from a study of women.<sup>26</sup> That study found an increased risk for alcohol A/D in women exposed to trauma with and without PTSD, compared with women who were not exposed to trauma. The possibility that exposure to trauma predicted an increased risk for alcohol A/D in women was suggested in the retrospective data from this study as well. In other reports, direct comparisons with the results from the prospective analysis or the survival analysis applied



to the lifetime data cannot be made because of methodologic differences. Some studies<sup>1,7,27-29</sup> of Vietnam combat veterans and samples of persons who have experienced a disaster, crime, or rape reported lifetime or concurrent associations between exposure to trauma and alcohol A/D. The associations were considerably higher in the presence than in the absence of PTSD.<sup>1,7</sup> Other studies<sup>30-32</sup> of Vietnam veterans and persons who have experienced a civilian disaster found a weak or no association between exposure to trauma and alcohol drinking or an alcohol-related disorder. Reports on the temporal order between the 2 disorders in comorbid cases have yielded mixed results. One study<sup>33</sup> found that PTSD generally preceded the onset of alcohol A/D, whereas another<sup>34</sup> found the opposite trend. A study<sup>35</sup> of psychiatric disorders in Australian Vietnam veterans currently not meeting PTSD criteria reported a significant association between level of combat and current alcohol A/D. The study did not compare the prevalence of alcohol A/D in the veterans with the prevalence in a matched sample of nonveterans. Furthermore, the association between level of combat and any disorder was rendered insignificant when veterans with a history of PTSD (in addition to veterans with current PTSD) were excluded. (The specific association with alcohol A/D in veterans with no history of PTSD was not reported.) With 1 exception,<sup>26</sup> these studies did not evaluate the relative risk for the subsequent onset of alcohol A/D associated with prior PTSD or exposure to trauma alone, relative to persons who were not exposed to trauma.

As in the case of alcohol A/D, our findings of an increased risk for other drug A/D in persons with PTSD, but not in persons exposed to trauma in the absence of PTSD, cannot be directly compared with previous reports. Previous studies reported cross-sectional associations or the tendency of one disorder to begin before the other disorder in comorbid cases, whereas this study evaluated the subsequent risk for a drug disorder given earlier exposure or PTSD, based on data from the entire sample. (A discussion of this methodologic issue appears elsewhere.<sup>17</sup>)

Few studies have reported on smoking in persons who have experienced trauma. One study found a high rate of current smoking in Vietnam combat veterans<sup>8</sup> and a significantly higher rate of current heavy smoking in combat veterans with PTSD than in combat veterans without PTSD.<sup>9</sup> An increased prevalence of smoking also was observed in women with PTSD<sup>10</sup> and in adolescents who experienced violence.<sup>11</sup> None of these studies considered the temporal order between exposure to trauma and the onset of smoking. To our knowledge, information on the relationship of PTSD or exposure to traumatic events with nicotine dependence, as distinct from smoking, has not been reported.

Results from the prospective and retrospective data, taken together, indicate that exposure to traumatic events in the absence of PTSD does not increase the risk for the subsequent onset of substance use disorders. Thus, for these disorders, the data do not support the hypothesis that PTSD-level traumatic events also induce other disorders. The results for nicotine dependence are less clear than the results for the other substance use disorders. The

prospective and retrospective analyses showed an increased risk for nicotine dependence in persons who experienced trauma and had PTSD; in both analyses, the magnitude of the risk in persons who experienced trauma and did not develop PTSD was significantly lower than in persons who experienced trauma and developed PTSD. However, an analysis of the prospective data showed that the risk in persons who experienced trauma and did not develop PTSD, although lower, was also significantly elevated; an analysis of the retrospective data failed to detect a significantly increased risk in persons exposed to trauma without PTSD, compared with the unexposed group. The differences in the findings might be because of the methodologic disparities between the 2 components. They also might be substantive, reflecting heterogeneity in the transition to nicotine dependence across age periods in the respondents' lives. The retrospective data cover the respondents' lifetime up to the ages of 21 to 30 years, whereas the prospective data cover a 10-year follow-up period until the respondents reached the ages of 31 to 40 years. We must, therefore, be equivocal in drawing inferences on this question and leave open the possibility that exposure per se, in the absence of PTSD, may confer a modestly elevated risk for developing nicotine dependence. A more definitive answer must await additional studies.

The evidence of a considerably increased risk for other drug A/D and nicotine dependence only in persons who experienced trauma and had PTSD suggests that PTSD might be the cause of nicotine dependence and other drug A/D or, alternatively, that PTSD and the subsequent onset of these disorders are caused by shared risk factors other than the exposure to trauma. Other shared risk factors might include a familial influence on PTSD and substance use disorders,<sup>12,13</sup> personality traits, early conduct problems, and family history of antisocial behavior, factors previously associated with PTSD and with substance use disorders.<sup>1,6,36-39</sup>

There is no support in these data for the notion that men who experience trauma, whose risk for PTSD is generally lower than that of women,<sup>5,6,31,40,41</sup> respond to these experiences by abusing alcohol and other drugs. Such a susceptibility would result in an increased risk of substance use disorders in the absence of PTSD, relative to unexposed men. We found that, for alcohol A/D, men who experienced trauma were not at an increased risk, and for other drug A/D, only men with PTSD, but not exposed men without PTSD, were at an increased risk.

Key findings from this study parallel previously reported findings<sup>42</sup> on the PTSD–major depression association, based on data from this epidemiologic study. An increased risk for major depression was observed in persons with PTSD, but not in persons who experienced trauma and did not develop PTSD. A previous study<sup>26</sup> of women yielded the same results. A similar pattern can be discerned in other epidemiologic reports.<sup>1,31,43</sup> For example, the prevalence of postdisaster comorbid disorders in survivors of the Oklahoma City bombing with PTSD was 63%, but in the absence of PTSD, it was only 9%.<sup>31</sup> In the National Vietnam Veterans Readjustment Study,<sup>1</sup> the prevalence of other disorders reached a markedly high level only among veterans with PTSD,

not in the entire subset of veterans exposed to high war zone stress.

Epidemiologic studies have consistently reported that only a small subset of persons exposed to traumatic events develop PTSD; most persons exposed to trauma do not. This study shows that persons who experience trauma and who do not develop PTSD (ie, most of those exposed) are not at a markedly elevated risk for the subsequent onset of major depression and substance use disorders, compared with unexposed persons. The excess incidence of other disorders in persons exposed to trauma is concentrated primarily in the small subset of persons who meet the diagnostic criteria for PTSD. These findings suggest that PTSD identifies a subset of persons who experience trauma who are at considerable risk for a range of disorders. The extent to which a shared diathesis with PTSD accounts for the elevated risk of these disorders might vary across disorders. The PTSD-depression association might primarily reflect a shared diathesis,<sup>26,42</sup> whereas the PTSD–drug use disorder might primarily reflect a causal effect of PTSD.<sup>17</sup> Family and twin studies, designed to examine genetic and environmental factors, are needed to further elucidate these relationships.

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Corresponding author and reprints: Naomi Breslau, PhD, Department of Psychiatry, Henry Ford Health System, 1 Ford Pl, Room 3A, Detroit, MI 48202-3450 (e-mail: nbresla1@hfh.org).

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