Patterns of incidence and psychiatric risk factors for traumatic events

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ABSTRACT *Individual differences are thought to influence the propensity for exposure to trauma and the subsequent development of post-traumatic stress disorder (PTSD) symptoms. Prior research has identified pre-existing mood disorders as one such individual risk factor for traumatic events as well as for PTSD. The present study reports the incidence of traumatic events (and PTSD) and examines psychiatric risk factors for trauma exposure in a prospective community sample.*

Data come from a prospective, longitudinal epidemiological study of adolescents and young adults (age 14–24) in Munich, Germany. Respondent diagnoses (N = 2,548) at baseline and at follow-up 34–50 months later were considered. Psychiatric diagnoses at baseline were examined as predictors of qualifying trauma.

Baseline prevalence of persons having experienced trauma meeting DSM-IV A1 and A2 criteria ('qualifying trauma') was 16.7%; during the follow-up period, 20.3% persons had experienced incident (new) qualifying traumata. The prevalence of PTSD, including subthreshold cases, at baseline was 5.6%; by the end of the follow-up period this had increased to 10.3%. Presence of an anxiety disorder at baseline predicted exposure to qualifying traumas during the follow-up period (adjusted ORs ranging from 1.36 for any trauma type to 3.00 for sexual trauma); this association was apparently due to an increased tendency to report events as being particularly horrific (meeting A2 criteria). In contrast, presence of illicit drug use predicted the onset of traumatic events (specifically assaultive and sexual trauma) meeting at least A1 criteria, suggesting an actual exposure to these types of traumatic events for this class of disorders.

In this prospective study of urban adolescents and young adults, certain classes of pre-existing psychiatric disorders (most notably anxiety disorders and illicit drug use disorders) were associated with increased risk for qualifying traumatic events. The mechanisms by which premorbid psychiatric disorders promote exposure to traumatic events are unknown. Better understanding of these pathways may lead to novel strategies for primary and secondary prevention of PTSD.

Key words: trauma, post-traumatic stress disorder, epidemiology, anxiety, drug abuse

Introduction

Post-traumatic stress disorder (PTSD), previously thought to be exclusively a disorder of combat veterans, is now recognized as one of the more prevalent mental disorders in the general population (Breslau, Davis, Andreski and Peterson, 1991; Resnick, Kilpatrick, Dansky, Saunders and Best 1993; Kessler, Sonnega, Bromet, Hughes and Nelson 1995; Stein, Walker, Hazen and Forde 1997; Kessler, Sonnega, Bromet, Hughes, Nelson and Breslau 1999; Perkonigg and Wittchen 1999; Perkonigg, Kessler, Storz and Wittchen 2000; Creamer, Burgess, and McFarlane

2001). Recent events (such as the terrorist attacks of 11 September 2001) have intensified interest in traumatic events and PTSD, in light of observations that a large segment of the population is at risk from these events and the development of PTSD symptoms (Schuster, Stein, Jaycox, Collins, Marshall, Elliott, Zhou, Kanouse, Morrison and Berry, 2001; Galea, Ahern, Resnick, Kilpatrick, Bucuvalas, Gold and Vlahov, 2002). These observations underscore the importance of developing a better understanding of factors that influence exposure to traumatic events and susceptibility to PTSD.

Traumatic events are defined by the DSM-IV PTSD A1 criterion that involves experience of actual or threatened death or serious injury, or a threat to the physical integrity of self or others; and the DSM-IV PTSD A2 criterion that involves an intense fear response with helplessness, or horror. Qualifying traumatic events include, for example, military combat, violent personal assaults, disasters, accidents or life-threatening illness. These qualifying traumatic events differ in their prevalence, as well as in their conditional risks for PTSD (Breslau, Kessler, Chilcoat, Schultz, Davis and Andreski, 1998; Bromet, Sonnega, and Kessler, 1998; Kessler, Sonnega, Bromet, Hughes, Nelson and Breslau, 1999). For example, events characterized by the element of interpersonal assault (such as rape) are more likely to be associated with PTSD than non-assaultive events (such as motor vehicle accidents) (Breslau, Kessler, Chilcoat, Schultz, Davis and Andreski, 1998). Within uniform types of trauma (such as combat), duration or intensity of exposure to the trauma tends to increase risk for PTSD (a dose-response relationship is evident) (Green, Grace, Lindy, Gleser and Leonard, 1990; Goldberg, True, Eisen and Henderson, 1990; King, King, Foy, Keane and Fairbank, 2000; Wolfe, Erickson, Sharkansky, King and King, 2000; King, King, Foy, Keane and Fairbank, 2000). Prior trauma is also a risk factor for development of PTSD after exposure to new trauma(ta) (Breslau, Chilcoat, Kessler and Davis, 1999).

Several studies of risk factors for traumatic events have shown that gender, age and social class predict specific types of events (for example sexual assault in women and military combat trauma in men). Prior mental disorders, or family history thereof, are also hypothesized to confer increased risk for traumatic events (as well as for PTSD once trauma exposure has occurred) (Davidson, Swartz, Storck, Krishnan and Hammett, 1985; Davidson, Smith and Kudler, 1989; Reich, Lyons and Cai, 1996; Yehuda, Halligan and Bierer, 2001). Some studies, for example, have found that pre-existing depressive disorders increase risk for PTSD (Breslau, Davis, Peterson and Schultz, 1997; Bromet, Sonnega and Kessler, 1998; Acierno, Resnick, Kilpatrick, Saunders and Best, 1999; Perkonigg, Kessler, Storz and Wittchen, 2000; Mayou, Bryant and Ehlers, 2001). Limiting interpretation of some of these studies, however, is the fact that a history of prior psychiatric disorder(s) and traumatic events as well as PTSD are often assessed contemporaneously, raising the possibility that concurrent symptoms might influence recall of traumatic events or otherwise confound their assessment.

Identification of individuals susceptible to traumatic events and PTSD (and mediators of this vulnerability) should be a cornerstone of any efforts directed at reducing the public health burden associated with this disorder (Galea, Ahern, Resnick, Kilpatrick, Bucuvalas, Gold and Vlahov, 2002). The purpose of the present study was to prospectively examine the role of pre-existing mental disorders as risk factors for traumatic events in a representative community sample of adolescents and young adults. As part of this investigation, we will report incidence rates of traumatic events and PTSD in this sample.

Methods

Sample and overall design

Data were collected as part of the Early Developmental Stages of Psychopathology (EDSP) study (Wittchen, Nelson and Lachner, 1998; Wittchen, Perkonigg, Lachner and Nelson, 1998). The EDSP is designed to explore the prevalence and incidence, familial and other risk factors, comorbidity and course of mental disorders in a representative population sample of adolescents and young adults. The study is divided into three waves, the first conducted in 1995 (baseline with all 14-to-24 year olds; N = 3,021), the second in 1996/1997 (T1, only with the 14-to-17 year olds at baseline: N = 1,228), and the third in 1998/1999 (T2, again with all 14-to-24 year olds at baseline; N = 2,548) and contains a family history component.

The baseline sample was drawn in 1994 from government registries in metropolitan Munich, Germany, of registrants expected to be 14–24 years of age at the time of the baseline interview in 1995. The

study was designed as a longitudinal panel with special emphasis on early developmental stages of psychopathology, 14–15 year olds were sampled at twice the probability of people 16–21 years of age, and 22–24 year olds were sampled at half this probability. At baseline (T0), a total of 3,021 interviews were completed, resulting in a response rate of 71%. The two follow-up assessments were completed after the initial baseline investigation, covering an overall period of three to four years (depending on the timing of the follow-up interviews, which were conducted after intervals of approximately 18 months). The first follow-up study (T1) was conducted on average 19.7 months (range = 15 to 25.6 months) after the baseline study with a response rate of 88%. Only the younger cohort (14–17 year olds at baseline) was included in this wave. The second follow-up (T2), in which all baseline respondents were included again, was conducted in 1998/99 on average of 42 months (range = 34 to 50 months) after baseline with a response rate of 84% (N = 2,548). A more detailed description of the study is available elsewhere (Wittchen, Nelson and Lachner, 1998; Lieb, Isensee, von Sydow and Wittchen, 2000).

Sociodemographic characteristics of the baseline (T0) and entire follow-up (T0-T2) sample have been published (Wittchen, Nelson, and Lachner, 1998; Lieb, Isensee, von Sydow, and Wittchen, 2000). Briefly, at baseline, most of the respondents were attending school (89%) and living with their parents (98%). About 10% were in job training. The majority was classified as belonging to the middle class (61%). Noteworthy changes in sociodemographic characteristics from baseline to second follow-up were found for school (follow-up: 42% attended school) and employment status (follow-up: 24% were in job training program and 12% were employed).

For those probands aged 14 to 17 years at baseline, the follow-up status in this report is assessed from the aggregation of information obtained from the first and second follow-up interviews. For those older than 17 years at baseline, the follow-up status is assessed from the second set of follow-up questions, which refer to the time between baseline and the second follow-up.

Diagnostic assessment
Detailed descriptions of the training procedures and field work have been reported elsewhere (Wittchen, Nelson and Lachner, 1998; Perkonigg, Kessler, Storz and Wittchen, 2000). Briefly, the highly experienced survey staff consisted of 57 clinical interviewers, most of whom were clinical psychologists. At baseline, 25 full-time professional health research interviewers were additionally included. Most of the interviewers had extensive experience in diagnostic interviewing, including the CIDI, and in the developmental work of the M-CIDI. All interviewers received two full weeks of training. In the fieldwork, most interviews were conducted in the home of the probands. To minimize errors and missing values, especially trained and clinical experienced M-CIDI editors closely supervised all interviewers.

Diagnostic assessments in the baseline and the two follow-up investigations of the respondents were based on the computer-assisted personal version of the Munich-Composite International Diagnostic Interview that allows for the assessment of symptoms, syndromes and diagnoses of 48 mental disorders according to the DSM-IV along with information about onset, duration, severity and psychosocial impairment. Diagnostic findings are obtained by using the M-CIDI/DSM-IV algorithms (Wittchen and Pfister, 1997). At baseline, the lifetime version of the M-CIDI was used. At each follow-up, the M-CIDI interval-version was applied. Test-retest reliability and validity findings of the full M-CIDI along with descriptions of the M-CIDI format and coding conventions have been reported in detail elsewhere (Wittchen, Lachner, Wunderlich and Pfister, 1998).

Post-traumatic stress disorder (PTSD) is defined here as meeting DSM-IV criteria per the M-CIDI diagnostic algorithm (Wittchen and Pfister, 1997); details have been presented in a previous PTSD-paper (Perkonigg, Kessler, Storz and Wittchen, 2000). Briefly, several modifications were necessary to match DSM-IV criteria and to take into account more recent methodological innovations:

- a screening question and a respondent list with 10 groups of specified events and an open-ended question about any other traumatic events,
- a question for each event to determine if the DSM-IV A2 criterion (intense fear, helplessness, or horror) had been met, and
- further probing for the most severe events as well as linkages between events.

The latter was used to determine exactly the number of qualifying events. If the respondent indicated several qualifying events (A1 and A2 criterion: events involv-

ing actual or threatened death or serious injury, or a threat to the physical integrity of self or others and a response with intense fear, helplessness, or horror) that did not cluster, only the criteria for the worst and most distressing event were assessed. For the purpose of this paper the 10 specific event types plus the 'other' category were grouped into two categories each with two mutually exclusive groups:

- assaultive versus non-assaultive: horrific experience during war, imprisoned, or taken hostage or kidnapped, physical attacks, sexual abuse and rape versus serious accidents, natural catastrophes, sudden (threat of) death of associates or witnessing traumatic events happen to others;
- sexual versus non-sexual traumas: rape, sexual abuse versus all other trauma types.

Similar categories have been used in other studies (Breslau, Chilcoat, Kessler, Peterson and Lucia, 1999; Stein, Walker and Forde, 2000). One-week test-retest reliability of PTSD was acceptable (kappa = 0.79) (Wittchen, Lachner, Wunderlich and Pfister, 1998) as was the validity (kappa = 0.85) (Reed, Gander, Pfister and Wittchen, 1998).

A number of studies have shown that the DSM-IV criteria for PTSD are too narrow in that they fail to include large numbers of persons who are clearly impaired in the aftermath of trauma. This has led to the frequent reference in research studies to persons

with so-called 'partial' or 'subthreshold' PTSD (Carlier and Gersons, 1995; Stein, Walker, Hazen and Forde, 1997; Schützwohl and Maercker, 1999; Marshall, Olfson, Hellman, Blanco, Guardino and Struening, 2001) referring to those individuals who fall a symptom or several short of DSM-IV syndromal PTSD, yet are often indistinguishable from those with full PTSD in terms of functional impairment. For the purpose of this paper we specified a category of partial PTSD, referring to persons who fulfilled the A, B (traumatic event, fear and persistent re-experiencing) and E criterion (duration) of DSM-IV but did not completely fulfil the C (avoidance or numbing of general responsiveness) and/or D criterion (hyperarousal) but did report at least one symptom from each of these two criteria with a duration of more than one month. The DSM-IV criterion of impairment was not necessary for membership in this category.

Table 1 shows a comparison of persons with and without qualifying trauma exposure, as well as threshold (full) and partial PTSD, by impairment. We found that persons with qualifying trauma exposure (regardless of PTSD status) showed a trend towards reporting greater functional impairment due to mental illness than persons with no qualifying trauma exposure. Persons with full and partial PTSD did not differ significantly in the mean number of days totally or even mildly disabled due to mental illness. Grouped together, persons with full or partial PTSD differed significantly from persons without PTSD in the number of days

Table 1. Relationship of trauma and PTSD to reported impairment in previous four weeks

	N	Number of Days Totally Disabled* Mean	mean ratio** (95% CI)	p-value	Number of Days At Least Mildy Disabled* Mean	mean ratio** (95% CI)	p-value
Lifetime trauma status							
Qualifying trauma	783	0.22			1.03		
No qualifying trauma	1501	0.13	2.01 (0.95-4.20)	0.064	0.83	1.30 (0.96-1.77)	0.082
Full PTSD	52	1.21			2.74		
Subthreshold PTSD	210	0.41	2.46 (0.94-6.41)	0.750	2.07	1.12 (0.40-2.26)	0.750
Full or Subthreshold PTSD	262	0.57			2.21		
No PTSD	2284	0.17	3.04 (1.65-5.63)	0.000	0.9	2.41 (1.79-3.24)	0.000

*Disability reported in relation to mental illness.
** Mean ratios from negative binomial regressions, adjusted for age and gender differences.

totally or mildly disabled due to mental illness (Table 1). We therefore combined these two categories for purposes of further reporting in this paper.

Statistical analysis

Data were weighted to consider different sampling probabilities as well as systematic non-response at baseline. The Stata Software package (StataCorp, 2001) was used for robust inference of statistical precision in the case of weighted data. Logistic regressions with odds ratios (OR) were used to describe associations between psychiatric predictor variables and new onset of qualified trauma and PTSD during the follow-up period, taking into account non-specific risk factors such as subject's age and sex. This report uses 95% confidence intervals (CIs) throughout. Analyses concerning associations between psychiatric predictor variables and subsequent qualifying trauma exposure and PTSD were conducted in the subsample of persons with new incident traumas (those not already reported at baseline) to avoid the confounding of predictors of new traumatic events and predictors of PTSD. We did not find selective attrition for persons with PTSD from baseline to follow-up relevant to our categories of DSM-IV PTSD and partial PTSD. Associations with the number of disability days were analysed with mean ratios from negative binomial regressions (Cox, 1983).

Results

Prevalence and incidence rates of qualifying trauma and PTSD

T2 follow-up data were available in 2,548 respondents. Prevalence rates of qualifying trauma (trauma meeting the DSM-IV A1 and A2 criteria) and full and partial (subthreshold) PTSD at baseline (T0), follow-up (T2), and cumulative incidence (from baseline to follow-up) are summarized in Table 2. Whereas only 16.7% (N_w = 425) of respondents had experienced a qualifying trauma at baseline, 20.3% (N_w = 430) experienced a new incident trauma during the period between baseline and follow-up. Post-traumatic stress disorder (full or partial) was present in 5.6% (N_w = 143) of respondents at baseline; this rate increased to 10.3% (N_w = 262) by the end of the follow-up period resulting from a cumulative incidence of 5.0% (N_w = 119) during follow-up (see Table 2).

Likelihood of experiencing a qualifying trauma during the follow-up period

Sociodemographic characteristics at baseline in relation to onset of qualifying trauma during the follow-up period were examined in respondents who had not already experienced a qualifying trauma at baseline (N = 2,155: N_w = 2,121). Of these persons, 20.3% (N_w = 430) experienced new qualifying trauma during the follow-up period. None of gender, employment or school status, living arrangements, educational level, or social class significantly predicted onset of qualifying trauma in these individuals. In contrast, the presence at baseline of certain classes of psychiatric disorder emerged as a moderately strong predictor of qualifying trauma during the follow-up period. These included presence at baseline of an anxiety disorder (OR 1.41, 95% CI 1.08–1.83) nicotine dependence (OR 1.42, 95% CI 1.02–1.95), or illicit drug use disorder (OR 1.85, 95% CI 1.04–3.26)). After controlling these associations for one another using multiple logistic regression analysis (Table 3), the psychiatric predictors of qualifying trauma during the follow-up period were reduced to baseline history of an anxiety disorder (OR = 1.36, 95% CI 1.04–1.78).

Asscociation with trauma type

It has been suggested that certain kinds of trauma (for example, assaultive and/or sexual trauma) are more likely than others to result in PTSD (Breslau, Kessler, Chilcoat, Schultz, Davis and Andreski, 1998; Stein, Walker and Forde, 2000; McQuaid, Pedrelli, McCahill and Stein, 2001). It would therefore be important to know if certain pre-existing mental disorders determined risk for either (or both) of these classes of trauma. Several factors were differentially associated (using multiple logistic regression analyses) with exposure to assaultive trauma (versus no trauma) and sexual trauma (versus no trauma) during the follow-up period (Table 3). Older age was associated with decreased risk for assaultive trauma (OR = 0.84, 95% CI 0.79–0.90 per year of age) (but not for sexual trauma; OR = 1.02, 95% CI 0.89–1.17) whereas female gender was associated with markedly increased risk for sexual trauma (OR = 11.11, 95% CI 2.12–58.20) but not for assaultive trauma (OR = 0.77, 95% CI 0.52–1.14). In addition to baseline presence of anxiety disorders, baseline presence of nicotine dependence or illicit drug use disorder (which were risk factors for any qualifying trauma in the preceding univariate but not the multivariate analyses) were also

Table 2. Prevalence of traumatic events and PTSD (full or partial) at baseline and cumulative incidence during follow-up*

	Male		Weighted results Female		Total	
	N_w	$%_w$	N_w	$%_w$	N_w	$%_w$
Baseline: any traumatic event (at least A1)						
no	950	75.27	1062	82.45	2012	78.99
yes	312	24.72	222	17.24	534	20.97
total	1262	100.00	1288	100.00	2547	100.00
Follow-up interval (T2): cumulative incidence of any new traumatic event (at least Al)*						
no	453	47.67	524	49.37	977	48.57
yes	497	52.33	538	50.63	1035	51.43
total	950	100.00	1062	100.00	2012	100.00
Baseline: qualified trauma lifetime						
no	1031	81.71	1090	84.87	2121	83.30
yes	231	18.29	194	15.13	425	16.70
total	1262	100.00	1284	100.00	2547	100.100
Follow-up interval (T2): cumulative incidence of new qualifying trauma*						
no	828	80.29	863	79.21	1691	79.73
yes	203	19.71	227	20.79	430	20.27
total	1031	100.00	1090	100.00	2121	100.00
Baseline: PTSD lifetime (includes subthreshold PTSD)						
no	1207	95.65	1196	93.11	2403	94.37
yes	55	4.35	88	6.89	143	5.63
total	1262	100	1284	100	2547	100.00
Follow-up (T2): PTSD cumulative incidence (includes subthreshold PTSD)						
no	1157	95.82	1127	94.26	2284	95.05
yes	50	4.18	69	5.74	119	4.95
total	1207	100.00	1196	100.00	2403	100.00

Results for cumulative incidence might reflect lower bound estimates because we excluded N = 84 cases from this analysis with baseline traumas and unclear onset information to identify the follow-up trauma as incident trauma.

associated with exposure to assaultive or sexual trauma exposure. Among these risk factors, it should be noted that illicit drug-use disorder was the strongest, associated with odds ratios ranging from approximately three to seven for these forms of trauma exposure.

Likelihood of events fulfilling A1 criterion of PTSD
We considered the possibility that anxiety disorders at baseline might increase the incidence of qualifying trauma if persons with anxiety disorders were prone to experience traumatic events as particularly frightening or horrific (leading them to endorse A2 criteria). We tested this hypothesis indirectly by determining if anxiety disorders were a risk factor for event(s) that specifically fulfil at least the A1 criterion (for which we felt that our data were most reliable). Although anxiety disorders, as shown in Table 3, are a risk factor for qualifying events in general, Table 4 shows that they are not a risk factor for event(s) that fulfil at least the A1 criterion. This suggests that the increase in liability for qualifying trauma associated with anxiety disorders reflects an increase in the tendency to experience (or endorse) the A2 criterion. On the other hand, illicit drug-use disorders did predict traumatic events fulfilling at least A1-criteria (Table 4).

Table 3. Baseline factors associated with onset of new qualifying trauma during the follow-up period*

	Odds ratio	95% CI	p-value
1. Onset of any new qualifying trauma			
Age at baseline (in years)	0.97	0.93–1.00	0.047
female gender	1.05	0.83–1.33	0.671
anxiety disorder	1.36	1.04–1.78	0.026
depressive disorder	1.01	0.68–1.48	0.975
nicotine dependence	1.26	0.90–1.77	0.178
alcohol-use disorder (abuse or dependence)	1.10	0.75–1.61	0.641
any illicit drug-use disorder	1.58	0.86–2.91	0.139
2. Onset assaultive trauma ($N_w = 100$)			
Age at baseline (in years)	0.84	0.79–0.90	0.000
female gender	0.77	0.52–1.14	0.191
anxiety disorder	1.73	1.11–2.69	0.016
depressive disorder	1.00	0.50–2.02	0.991
nicotine dependence	2.57	1.55–4.27	0.000
alcohol-use disorder (abuse or dependence)	0.60	0.27–1.34	0.213
any illicit drug-use disorder	3.25	1.31–8.10	0.011
3. Onset sexual trauma ($N_w = 27$)			
Age at baseline (in years)	1.02	0.89–1.17	0.744
female gender	11.11	2.12–58.20	0.004
anxiety disorder	3.00	1.20–7.51	0.019
depressive disorder	0.94	0.32–2.81	0.915
nicotine dependence	2.95	1.18–7.39	0.021
alcohol-use disorder (abuse or dependence)	0.44	0.09–2.26	0.327
any illicit drug-use disorder	7.00	2.00–24.44	0.002

*Among $N_w = 2121$ respondents without qualifying trauma at baseline.
**Versus no qualifying trauma.

Table 4. Baseline factors associated with onset of any new A1 -trauma during the follow-up period

	Odds ratio	95% CI	p value
Onset of any new A1 trauma			
anxiety disorder	1.21	0.95–1.53	0.122
depressive disorder	1.09	0.78–1.51	0.622
nicotine dependence	1.04	0.77–1.40	0.808
alcohol-use disorder (abuse or dependence)	0.76	0.54–1.05	0.098
any illicit drug-use disorder	1.89	1.04–3.44	0.037

Comment

In this prospective study we found that the presence of certain mental disorders in adolescence or early adulthood are risk factors for the experience of traumatic events. Specifically, we found that a pre-existing anxiety disorder (and, for certain forms of trauma such as assaultive or sexual trauma, or a pre-existing nicotine or illicit drug-use disorder) moderately increases risk for qualifying traumatic events. These findings are generally consistent with the extant literature that has

highlighted a relationship between prior mental disorders (most consistently major depression), traumatic events and subsequent PTSD (Breslau, Davis, Peterson and Schultz 1997; Bromet, Sonnega and Kessler, 1998; Acierno, Resnick, Kilpatrick, Saunders and Best, 1999; Perkonigg, Kessler, Storz and Wittchen, 2000; Mayou, Bryant and Ehlers, 2001). But our findings are novel in uncovering an association with pre-existing anxiety disorders, as well as with pre-existing nicotine dependence or illicit drug use for traumatic events (for assaultive and sexual trauma).

Importantly, however, we found that the association between pre-existing anxiety disorders and qualifying traumatic events is probably conveyed indirectly, through the increased risk that anxiety disorders convey for anxious responses (A2-PTSD criterion) after exposure to traumatic events. These observations are consistent with other data supporting the notion that anxiety may increase the risk for traumatic events (or, as our data imply, the perception of certain events as being sufficiently traumatic to meet the A2 criterion). In their study of 1,007 young adults age 21–30 from a large health maintenance organization, neuroticism was among the individual differences that conferred greater risk for trauma exposure, thereby putting some persons at increased risk for PTSD (Breslau, Davis and Andreski, 1995). In a recent study of phobic twins, Kendler et al. also noted that neuroticism was associated with increased likelihood of reporting a traumatic event in association with onset of the phobia (Kendler, Myers and Prescott, 2002). In light of the fact that neuroticism is generally viewed as a trait marker (or liability factor) for anxiety disorders (Andrews, Stewart, Morris-Yates, Holt and Henderson, 1990; Clark, Watson and Mineka, 1994) our finding that pre-existing anxiety disorders are associated with increased rates of subsequent traumatic events may be broadly consistent with these observations. It remains to be seen if the presence of full-blown anxiety disorders confers risk independent from that of anxiety-related traits (for example, neuroticism). Furthermore, the possibility that attributional differences influence the perception of negative events as 'traumatic' bears further study.

On the other hand, the associations with illicit drug use disorders (and, to a lesser extent, with nicotine dependence) and assaultive and sexual trauma exposure seem to represent a genuine increase in exposure to these events (rather than merely to their perception

as horrific, and so forth). However, these findings are generally contrary to those of other investigators who found that PTSD increased the risk for subsequent substance-use disorders but not vice-versa (Chilcoat and Breslau, 1998a; Chilcoat and Breslau, 1998b). Geographic differences (for example, Detroit versus Munich) in patterns of drug use may explain this inconsistency. Other research groups have reported evidence of a common vulnerability for PTSD and illicit drug use disorders (Dierker and Merikangas, 2001). Further longitudinal research will be necessary to better understand the directionality and importance of this pathway to (or from) PTSD.

It is important to emphasize that causal inferences cannot be drawn from these observational data. Nonetheless, some cautious interpretations of the data are in order. The association between pre-existing illicit drug-use disorder and trauma exposure seems obvious (although the association with nicotine dependence is less so). Persons who abuse drugs are likely to frequent environments where the risk of sexual or other assaultive violence is high. We must speculate, however, about mechanism(s) through which pre-existing anxiety disorders and nicotine dependence would increase the risk for subsequent exposure to traumatic events. One possible explanation is the definition and the methods we use to assess traumatic events. By DSM-IV definition (criterion A2), traumatic events that yield PTSD must involve intense fear, helplessness or horror. As such, the individual's cognitive appraisal of the event largely determines whether or not this criterion is met. Investigators have shown that certain factors (such as being female) increase the likelihood of endorsing the A2 criterion following trauma exposure (Breslau and Kessler, 2001). It is possible that anxious persons are more likely than non-anxious persons to perceive certain events as meeting these criteria. Hypotheses concerning reporting biases can be tested in future prospective studies, where a broader array of potentially 'traumatic' events is assayed, at the same time paying close attention to the cognitive appraisal processes of traumatized individuals.

Additional limitations of our study should be considered. It is possible that persons most impaired by psychiatric disorders did not participate in our study. It is also possible that our findings from this urban German sample, consisting of persons who are well educated with relatively high economic status, may not generalize to other populations. Diagnoses at baseline are

retrospective and therefore subject to possible recall problems or biases, although these should be attenuated in this relatively young sample. Finally, we wish to remind the reader that our findings may not generalize to exposure to other types of traumatic events that were rare or did not occur at all in the present sample (such as natural catastrophes or terrorist attacks).

Efforts directed at lessening the public-health burden of PTSD through preventive measures will be informed by studies that delineate susceptible populations. Previous studies have identified, for example, youth exposed to violence as being at particularly high risk of PTSD (Berton and Stabb, 1996; Campbell and Schwarz, 1996; Cooley-Quille, Boyd, Frantz and Walsh, 2001; Mazza and Reynolds, 1999; Schwab-Stone, Chen, Greenberger, Silver, Lichtman and Voyce, 1999; Ward, Flisher, Zissis, Muller and Lombard, 2001). Our findings suggest that persons with certain pre-existing mental disorders – anxiety disorders and illicit drug-use disorders, in particular, are at increased risk for exposure to traumatic stressors (and/or increased likelihood of experiencing traumatic events as inherently life-threatening). The mechanism by which these premorbid psychiatric disorders promote exposure to traumatic events (that have the potential to lead to PTSD) is unclear. Better understanding of these pathways is likely to lead to novel mechanisms for primary prevention of PTSD. Furthermore, future work should focus on psychiatric risk factors for developing PTSD (and other forms of common post-traumatic psychopathology, such as major depression) conditional upon trauma exposure (Breslau, Chase and Anthony, 2002) in order to identify possible avenues for secondary prevention.

Acknowledgements

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