Traumatic Events and Posttraumatic Stress in Childhood

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Context: Traumatic events are common and are related to psychiatric impairment in childhood. Little is known about the risk for posttraumatic stress disorder (PTSD) across different types of trauma exposure in children.

Objective: To examine the developmental epidemiology of potential trauma and posttraumatic stress (PTS) in a longitudinal community sample of children.

Methods: A representative population sample of 1420 children aged 9, 11, and 13 years at intake were followed up annually through 16 years of age.

Main Outcome Measure: Traumatic events and PTS were assessed from child and parent reports annually to 16 years of age. Risk factors and *DSM-IV* disorders were also assessed.

Results: More than two thirds of children reported at least 1 traumatic event by 16 years of age, with 13.4% of those children developing some PTS symptoms. Few PTS symptoms or psychiatric disorders were observed for individuals experiencing their first event, and any effects were short-lived. Less than 0.5% of children met the criteria for full-blown *DSM-IV* PTSD. Violent or sexual trauma were associated with the highest rates of symptoms. The PTS symptoms were predicted by previous exposure to multiple traumas, anxiety disorders, and family adversity. Lifetime co-occurrence of other psychiatric disorders with traumatic events and PTS symptoms was high, with the highest rates for anxiety and depressive disorders.

Conclusions: In the general population of children, potentially traumatic events are fairly common and do not often result in PTS symptoms, except after multiple traumas or a history of anxiety. The prognosis after the first lifetime trauma exposure was generally favorable. Apart from PTSD, traumatic events are related to many forms of psychopathology, with the strongest links being with anxiety and depressive disorders.

Arch Gen Psychiatry. 2007;64:577-584

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POSTTRAUMATIC STRESS DISORDER (PTSD) is distinct from most psychiatric disorders in requiring an initiating stressor.[1] Early PTSD research focused on Vietnam War veterans and rape victims, leading to a narrow definition of the stressor criteria in the *DSM-III* and *DSM-III-R*.[2] Increased attention to the subjective appraisal of potentially traumatic situations in the 1980s and early 1990s led to a broader view of what constituted a stressor. The criteria were amended in the *DSM-IV* to include a wider range of events such as serious illnesses, natural disasters, and exposure to community violence.[1] Research on PTSD with younger samples has added to the list of potentially traumatic events. For example, Giaconia and colleagues[3] found that a parent being sent to prison put adolescents at the same risk of PTSD as rape did. Additional childhood events identified as potentially traumatic include sudden separation from a loved one and learning of a traumatic event occurring to a parent or a loved one.[3,4] Many studies have looked at the risk for PTSD in children, given exposure to a specific trauma such as a natural disaster or a motor vehicle crash,[5-11] but few have assessed exposure to a full range of potentially traumatic events. Efficient treatment and prevention strategies require knowledge of the conditional risk for PTSD, given different event categories across the full range of potentially vulnerable groups. Only data from community samples can provide this information.[12]

The few studies that have looked at a range of events in representative samples supported moderate levels of PTSD in children exposed to traumatic events,[3,4] with some evidence of higher vulnerability in girls than in boys.[4] These studies provided the first look at the epidemiology of PTSD in adolescents, but each is limited

by 1 of the following issues: (1) reliance on retrospective report, which tends to underestimate levels of traumatic events[13-20]; (2) use of small samples; or (3) assessment of events and symptoms at a single time point only.

The present study examines the role of potentially traumatic events in childhood PTSD and other *DSM-IV* psychopathology. About one third of the sample participants reported their first event exposure subsequent to the first wave of data collection. This provided a within-subject natural experiment to study predictors of incident exposure to trauma and subsequent PTSD symptoms. The study seeks to answer the following questions:

1. What is the population prevalence of PTSD and posttraumatic stress (PTS) symptoms in childhood and adolescence?

2. Given the occurrence of a potentially traumatic event, what are the conditional probabilities associated with PTSD, related symptoms, and impairments?

3. What are the characteristics of children who are vulnerable to developing PTS symptoms in response to potentially traumatic events?

4. What is the lifetime co-occurrence of PTSD and PTS symptoms and other *DSM-IV* childhood disorders?

5. What predicts the first potentially traumatic experience and response to it?

METHODS

SETTING AND SAMPLING

The Great Smoky Mountains Study is a longitudinal study of psychopathology and use of medical services in childhood.[21,22] A multistage sample design was used to randomly select potential participants from a population of 20 000 children in 11 counties in western North Carolina. Three cohorts of children aged 9, 11, and 13 years were recruited at intake. Potential participants were randomly selected from the population using a household equal-probability, accelerated cohort design. This means that each cohort reaches a given age in a different year, thus controlling for cohort effects.[23] The initial random sample of 4067 yielded 3896 (95.8%) screening questionnaires consisting of the externalizing (behavioral) problems scale of the Child Behavior Checklist completed by the parent, on the telephone or in person. All children scoring above a predetermined cutoff point (the top 25% of the total scores), plus a 1-in-10 random sample of the rest, were recruited for detailed interviews. The contribution of each participant is weighted by the inverse of their selection probabilities, stratified by age, sex, and race or ethnicity, to provide accurate prevalence estimates for the population of the study area.

Of 1796 children recruited, 79.1% (n=1420) agreed to participate. Across annual waves, 83.4% of the 8002 possible interviews have been completed with 75.0% to 94.2% of the sample who participated in each wave. The data presented herein, based on the first 8 annual waves of the study (1993-2000), consist of 6674 interviews with participants through 16 years of age and 1 parent. Funding constraints prevented our interviewing the youngest cohort from January 1997 through June 1998. Because subjects were randomly selected, the cohort members interviewed from July to December 1998 are a random sample of the whole cohort. Data were collected on 1 cohort at 9 and 10 years of age; 2 cohorts at 11, 12, and 13 years of age; and all 3 cohorts at 14, 15, and 16 years of age.

The final sample consisted of 790 boys and 630 girls (weighted percentages, 51.1% and 49.0% respectively). In the unweighted sample, 69.2% (n=983) were white, 6.2% (n=88) were African American, and 24.6% (n=349) were American Indian. When weighted back to population probability of selection, the respective proportions were 89.5%, 6.9%, and 3.6%, respectively.

MEASURES

We based our analyses on the following 3 areas of information: (1) psychiatric disorders, (2) potentially traumatic events and associated PTS symptoms, and (3) risk factors. These areas were assessed using the Child and Adolescent Psychiatric Assessment (CAPA).[24,25] Symptoms are coded using an extensive glossary, and diagnoses are generated by computer algorithms. With the exception of attention-deficit/hyperactivity disorder symptoms, about which only the parent was interviewed, a symptom is counted as present if it was reported by the parent, the child, or both, as is standard clinical practice. The 2-week test-retest reliability of the CAPA diagnoses in children and adolescents aged 10 to 18 years is comparable to that of other highly structured interviews (κ range for individual disorders, 0.56-1.00).[25] The time frame of the CAPA for determining the presence of most psychiatric symptoms is the 3 months immediately preceding the interview to minimize recall bias.

Details of the construction and psychometric testing of the life events and PTS sections of the CAPA are contained in another report.[26] The life events section covered 17 areas of children's lives that could potentially induce PTS symptoms. The terms *trauma* and *traumatic events* are used to describe these events in reporting our results, but this is not meant to imply that the events are traumatic apart from the individual's response to the event. These events meet the *DSM-IV* PTSD criterion A, which stipulates that the event must involve "actual or threatened death or serious injury, or a threat to the physical integrity of self or others."[1(p427)] The parent or the child is queried about lifetime occurrence of each event and, where necessary, when it occurred. For each event, the interviewer administered a screen to determine whether the 3 key symptoms of PTSD (painful recall, avoidance, and hyperarousal) required for a *DSM-IV* diagnosis have been present during the past 3 months and are linked to the event under discussion. Painful recall/reexperience is assessed first and, if endorsed, the interviewer inquires about avoidance or hyperarousal. This procedure was put in place to avoid false-positive responses associated with the more common and less specific behavior patterns of hyperarousal and avoidance of painful stimuli. Painful recall is coded as present if the child or the parent reports unwanted, painful, and distressing recollections, memories, thoughts, or images of the life event, the occurrence of which the child cannot prevent (including childhood manifestations such as nightmares, reenactment, and repetitive play). *Avoidance* is defined as avoiding situations that might provoke recall of the event, and hyperarousal as an increased general level of awareness and alertness toward the subject's surroundings, in the absence of imminent danger. If minimal levels of all 3 symptoms are endorsed, then the detailed PTSD module is completed. Up to 2 detailed PTSD sections could be completed, 1 for the most upsetting event meeting 3 screens in the past 3 months, and 1 for the most upsetting lifetime event. A reliability study with 58 parents and children interviewed twice by different interviewers supported fair to excellent test-retest reliability (interclass correlations, 0.58-0.83, depending on the informant and type of event).[27] Discriminant validity was established through comparisons of general population and clinic-referred subjects.[27]

The PTSD section of the CAPA inquires in detail about the 3 main symptom clusters. Coping mechanisms such as normal, obsessional, and compulsive suppression are explored; questions are asked about autonomic effects such as panic attacks;

Table 1. Three-Month and Cumulative Prevalence Estimates of Traumatic Events, Posttraumatic Stress Disorder (PTSD), and Related Symptoms

| | Prevalence Estimate, Percentage (SE) | | | | | | | |
| | 3-Month | | | | Lifetime | | | |
Characteristic	Trauma	Painful Recall	Subclinical PTSD	PTSD	Trauma	Painful Recall	Subclinical PTSD	PTSD
Total	5.9 (0.5)	2.2 (0.3)	0.5 (0.1)	0.1 (0.1)	68.2 (2.1)	9.1 (1.2)	2.2 (0.6)	0.4 (0.2)
Age, y								
9-13	4.8 (0.7)	2.0 (0.4)	0.2 (0.1)	0.03 (0.02)	54.0 (2.4)	5.4 (1.0)	0.9 (0.4)	0.1 (0.04)
14-16	6.6 (0.7)	2.3 (0.4)	0.6 (.2)	0.1 (0.1)	68.2 (2.1)	9.1 (1.2)	2.2 (0.6)	0.4 (0.2)
Sex								
Male	5.9 (0.7)	2.1 (0.5)	0.6 (.2)	0.01 (0.01)	67.9 (2.9)	8.1 (1.7)	2.8 (1.1)	0.1 (0.1)
Female	6.0 (0.7)	2.4 (0.4)	0.3 (0.1)	0.2 (0.1)	68.4 (3.0)	10.2 (1.8)	1.6 (0.5)	0.7 (0.5)
Ethnicity								
White	5.7 (0.5)	2.2 (0.3)	0.4 (0.1)	0.1 (0.1)	67.1 (2.3)	9.1 (1.3)	2.1 (0.6)	0.4 (0.3)
Native American	5.0 (0.6)	2.3 (0.4)	0.4 (0.1)	*	73.9 (2.4)	9.7 (1.6)	1.7 (0.7)	*

*Indicates no cases.

and other associated features are queried (eg, "omen formation" and engagement in dangerous activities). Diagnoses of PTSD are computed using *DSM-IV* algorithms. Further details can be found in a previous publication.[27]

PROCEDURES

Children were first interviewed as closely as possible to their 9th, 11th, or 13th birthday and were interviewed annually until 16 years of age, at home or in a location convenient for them. Before the interviews began, the parent and child signed informed consent forms approved by the institutional review board of the Duke University Medical Center. They were then interviewed in separate rooms. Each parent and child was paid $10 after the interview was completed. Interviewers attempted to interview the primary caregiver, who was usually the biological mother (83.2% of the time).

Interviewers were residents of the area in which the study took place. All had at least bachelor's-level degrees. They received 1 month of training and constant quality control, maintained by postinterview reviews of each schedule by experienced interviewer supervisors and study faculty (A.A.). Interviewers were trained by the Department of Social Services staff per North Carolina's requirements for reporting abuse or neglect, and all suspected cases were referred to the appropriate agency.

DATA MANAGEMENT AND ANALYSIS

Scoring programs for the CAPA, written in SAS statistical software,[28] combined information about the date of onset, duration, and intensity of each symptom to create *DSM-IV* diagnoses. Prevalence estimates, odds ratios, and group comparisons were computed using the SAS program GENMOD.[28] We used general estimation equations to account for the sampling design and within-subject correlation. In general estimation equations, the subject is introduced as a cluster (class) variable, and the sampling weights are introduced as a scale vector that multiplies the subject's wave-to-wave correlation matrix. We also used the robust variance estimates (ie, sandwich-type estimates), together with sampling weights, to adjust the standard errors of the parameter estimates to account for the multiphase sampling design. The use of multiwave data with the appropriate sample weights thus capitalized on the multiple observation points over time while controlling for the effect on variance estimates of the repeated measures. For predictive analyses, logistic regression using the SAS procedure GLIMMIX[28] examined the effects of 1-year–lagged variables on current functioning. Models were evaluated with the Bayesian information criteria, an index that balances model fit with complexity.[29]

RESULTS

POPULATION PREVALENCE OF PTSD AND PTS SYMPTOMS

We derived the following 3 categories of PTS symptoms: (1) meeting all diagnostic criteria for *DSM-IV* PTSD; 2) endorsing at least 1 symptom each of painful recall, hyperarousal, and avoidance symptoms but not meeting full PTSD criteria (subclinical PTSD); and (3) reporting painful recall only. **Table 1** displays 3-month and cumulative prevalence estimates for trauma exposure and the 3 symptom categories. Two thirds of the sample reported exposure to 1 or more events by 16 years of age. Trauma exposure was more common in adolescence than childhood ($z=1.99$; $P=.05$).

Full-blown *DSM-IV* PTSD was rare across all sex, age, and ethnic groups (n=6; weighted prevalence, 0.5%). Painful recall and subclinical PTSD were more common, with cumulative rates of 9.1% and 2.2%, respectively, by 16 years of age in the full sample. Subclinical PTSD was more common in adolescence than childhood ($z=2.24$; $P=.02$). Rates of painful recall and subclinical PTSD did not differ across sex or ethnic groups. Because full-blown PTSD was so rare, the few cases were included in the painful recall and subclinical PTSD groups, but not analyzed separately.

CONDITIONAL PROBABILITIES

Table 2 displays the prevalence rates for all potentially traumatic events and the likelihood of developing symptoms when exposed to particular events. Events are grouped into the broad categories of violence, sexual trauma, other injury or trauma, witness to trauma, and learning about trauma. By 16 years of age, similarly sized groups of chil-

Table 2. Prevalence of Traumatic Events and Conditional Probabilities for PTS Symptoms

Type of Trauma	Lifetime			3-Month		
	Prevalence Rate, %	Painful Recall*	Subclinical PTSD*	Prevalence Rate, %	Painful Recall*	Subclinical PTSD*
Violence	24.7	15.5	3.7	1.4	7.4	2.5
Violent death of loved one	2.4	39.9	14.1	0.04	24.2	18.4
Violent death of sibling/peer	14.5	12.0	0.6	0.7	3.5	1.2
War, terrorism	0.1	0	0	0	0	0
Cause of death or severe harm	0.6	5.3	0	0.1	0	0
Victim of physical violence	3.1	13.0	9.1	0.2	6.6	0
Physical abuse by relative	7.2	13.5	2.2	0.5	20.0	4.3
Captivity	0.9	7.0	3.5	0	0	0
Sexual trauma	11.0	10.0	3.9	0.2	16.4	6.4
Sexual abuse	10.9	8.4	3.4	0.2	17.4	6.8
Rape	1.2	33.2	17.1	0.02	65.6	34.4
Coercion	4.3	21.9	7.9	0.05	32.2	16.1
Other injury or trauma	32.8	4.5	1.8	1.8	7.7	6.7
Diagnosis of physical illness	11.0	3.6	2.4	0.7	9.6	8.3
Serious accident	11.6	7.6	2.7	0.4	20.4	18.0
Natural disaster	11.1	0.8	0.3	0.2	0	0
Fire	5.9	1.5	0	0.3	0	0
Exposure to noxious agent	3.3	0.3	0	0.3	0	0
Witness to life event	23.7	10.2	1.7	1.3	11.8	1.3
Learned about life event	21.4	6.7	1.5	1.4	2.5	0
Any trauma	67.8	13.4	3.3	5.9	8.7	3.1
No. of traumatic events						
1	30.8	6.7	0.3	5.6	7.7	3.1
≥2	37.0	19.1	5.7	0.3	25.3	2.6

Abbreviations: PTS, posttraumatic stress; PTSD, PTS disorder.
*Reported as the percentage of individuals exposed to the event (ie, conditional probability). Lifetime probabilities refer to current responses to any previous event, whereas the 3-month probabilities are limited to current response to recent events.

dren reported no event exposure (32.2%), exposure to 1 event (30.8%), or exposure to multiple events (37.0%). The most common events were witnessing or learning about a traumatic event—so-called "vicarious" events.

Averaging 3-month and lifetime prevalance rates, about 1 of every 10 subjects exposed to trauma reported painful recall and about 3% reported subclinical PTSD. The highest rates of painful recall and subclinical PTSD were associated with violent events or sexual trauma. Three-month and lifetime conditional probabilities did not differ appreciably across most traumatic event categories. In addition to event characteristics, a lifetime history of multiple trauma exposures strongly predicted higher rates of painful recall and subclinical PTSD.

Subjects and their parents were queried about recent impairments related to their experience of the traumatic event. Impairments included a wide range of problems, including disruption of important relationships, school problems, physical problems, and exacerbation of emotional problems. Rates of impairments were generally double the rates of having any painful recall. Children with any traumatic event reported impairment 21.9% of the time. As with PTS symptoms, the rates of impairment increased with the number of traumatic events experienced. Impairment rates were 20.4% for those exposed to 1 event and 49.6% for children exposed to 2 or more events. Additional breakdowns by type of trauma and type of impairment are available by request from one of us (W.E.C.).

RISK FOR PTS SYMPTOMS

To test the predictors of PTS symptoms in the presence of a traumatic event, 4 sets of variables were entered into a model: (1) sex and current developmental period (ages 9-13 [childhood] vs 14-16 years [adolescence]); (2) previous emotional and behavioral disorders (anxiety disorders, depressive disorders, and disruptive behavior disorders); (3) previous negative events; and (4) previous environmental, family, and parental risk factors. All predictors except sex and developmental period were assessed 1 year prior to trauma exposure. Results of the logistic analyses are presented in **Table 3**. Adolescence was a strong predictor of both painful recall and subclinical PTSD, controlling for other predictor variables. Other significant predictors varied across symptom categories. Painful recall was predicted independently by exposure to a previous trauma and being previously diagnosed as having an anxiety disorder. Previous diagnosis with a depressive disorder did not independently predict trauma response. For subclinical PTSD, the best-fitting model included only sex and age. In the full model, previous environmental adversity, such as coming from an impoverished or poorly educated home, predicted subclinical PTSD, although the fit index supports a more parsimonious solution. These models suggest that age, prior anxiety, and previous trauma exposure are important determinants of trauma response in the next year.

Table 3. Logistic Regression Analyses Predicting Painful Recall and Subclinical PTSD From 1-Year–Lagged Risk Factors

	OR (95% CI)†							
	Painful Recall				**Subclinical PTSD**			
Variable*	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 1**	**Model 2**	**Model 3**	**Model 4**
Sex	0.8 (0.4-1.5)	0.8 (0.4-1.5)	0.8 (0.4-1.4)	0.8 (0.5-1.6)	2.2 (0.7-7.2)	2.4 (0.7-7.8)	2.3 (0.7-8.0)	2.4 (0.7-8.5)
Age	1.8 (0.9-3.4)	2.0 (1.0-3.8)‡	2.1 (1.1-3.9)‡	2.1 (1.1-4.1)‡	3.3 (1.4-9.5)‡	3.5 (1.2-10.2)‡	3.8 (1.4-10.6)‡	4.0 (1.4-11.5)‡
Trauma	§	2.4 (1.2-4.7)‡	2.3 (1.2-4.4)	2.2 (1.2-4.3)‡	§	3.0 (0.9-9.3)	2.5 (0.8-8.0)‡	2.6 (0.8-8.6)
Life events	§	1.9 (1.0-3.6)	1.7 (0.9-3.3)	1.7 (0.8-3.3)	§	2.9 (1.0-8.6)†	2.1 (0.7-6.3)	2.2 (0.7-6.8)
Environmental adversities	§	§	1.2 (0.5-2.4)	1.2 (1.0-1.4)	§	§	1.2 (1.0-1.5)	1.3 (1.0-1.5)‡
Family dysfunction	§	§	1.2 (0.7-2.2)	1.2 (0.8-1.4)	§	§	1.6 (0.8-3.3)	1.7 (0.8-3.8)
Parental psychopathology	§	§	1.3 (0.7-2.4)	1.2 (0.8-.14)	§	§	1.1 (0.7-1.8)	1.1 (0.7-1.8)
Anxiety disorder	§	§	§	2.7 (1.3-5.4)‡	§	§	§	2.1 (0.5-8.1)
Depressive disorder	§	§	§	1.5 (0.5-4.1)	§	§	§	0.2 (0.1-1.1)
Behavioral disorder	§	§	§	0.8 (0.4-1.6)	§	§	§	0.4 (0.1-2.2)
BIC	9355.6	9364.61	9348.8	9439.6	11 493.8	11 613.9	11 704.2	11 945.4

Abbreviations: BIC, Bayesian information criterion (lower is better); CI, confidence interval; OR, odds ratio; PTS, posttraumatic stress; PTSD, PTS disorder.

*All predictor variables except sex and age were assessed 1 year before trauma exposure. The age variable refers to childhood or adolescent status. Life events includes low-magnitude events that did not meet the criteria as a *DSM-IV* extreme stressor. Environmental adversities includes a range of variables such as poverty, having a single parent, having 1 or more unemployed parents, and living in a dangerous community. Family dysfunction refers to parenting problems, marital conflict, or frequent conflict between the child and a parent. The parental psychopathology variable includes mental health problems, substance abuse problems, and criminality.

†Models are described in the "Risk for PTS Symptoms" subsection of the "Results" section.

‡*P*<.05.

§Indicates variable not included in model.

Table 4. Lifetime Comorbidity Rates Between Psychiatric Disorders, Trauma Exposure, and Posttraumatic Stress Symptoms

	Trauma, %			**Painful Recall, %**			**Subclinical PTSD, %**		
Diagnosis	**None**	**Any**	**OR (95% CI)**	**None**	**Any**	**OR (95% CI)**	**None**	**Any**	**OR (95% CI)**
Any disorder	25.5	40.4	2.0 (1.3-3.0)*	33.9	52.6	2.2 (1.2-3.9)†	35.1	59.5	2.7 (0.8-9.2)‡
Affective disorders									
Major depressive episode	2.2	1.7	0.8 (0.2-3.0)	1.5	5.5	3.8 (1.1-13.1)‡	1.6	15.4	11.5 (2.3-57.8)†
Dysthymia	1.3	1.8	1.3 (0.3-6.3)	1.1	7.1	7.2 (1.8-29.1)†	1.1	26.3	33.4 (7.0-160.1)*
Depressive disorder NOS	3.2	9.5	3.2 (1.3-7.8)†	6.3	19.9	3.8 (1.8-7.8)*	6.9	35.6	7.5 (2.3-24.8)*
Any depressive disorder	3.3	9.8	3.2 (1.4-7.5)†	6.5	20.3	3.7 (1.8-7.6)*	7.1	37.1	7.7 (2.4-25.0)*
Anxiety disorders									
Separation anxiety disorder	1.1	6.7	6.3 (3.1-12.8)*	4.5	8.8	2.0 (1.1-3.8)‡	4.8	12.9	3.0 (1.1-7.8)‡
Generalized anxiety disorder	3.1	11.7	4.2 (1.7-10.1)*	6.7	31.4	6.4 (3.2-12.7)*	8.3	39.9	7.4 (2.3-23.3)*
Social anxiety disorder	1.2	2.6	2.2 (0.4-10.7)	1.7	6.2	3.8 (1.1-13.5)‡	2.1	4.1	2.0 (0.5-8.2)
Any anxiety disorder	3.0	12.1	4.5 (2.2-9.3)*	7.6	24.8	4.0 (2.1-7.7)*	8.4	43.0	8.2 (2.7-25.3)*
Substance use disorders	6.4	10.6	1.7 (0.8-3.6)	8.3	18.4	2.5 (1.1-5.4)‡	9.0	20.3	2.6 (0.9-10.9)
DBDs									
ADHD	2.3	3.8	1.7 (0.7-4.3)	3.2	4.0	1.3 (0.6-2.7)	3.2	10.2	3.5 (1.2-9.8)‡
Conduct disorder	3.9	10.8	3.0 (1.6-6.0)*	7.6	18.0	2.7 (1.3-5.3)†	8.2	23.2	3.4 (0.9-12.6)
ODD	5.6	11.7	2.2 (1.2-4.2)	9.6	11.6	1.2 (0.7-2.1)	9.5	19.5	2.3 (0.9-5.6)
Any DBD	9.4	19.2	2.3 (1.4-3.7)*	15.2	24.5	1.8 (1.0-3.3)‡	15.6	35.6	3.0 (1.0-8.9)‡

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; CI, confidence interval; DBD, disruptive behavior disorder; NOS, not otherwise specified; ODD, oppositional defiant disorder; OR, odds ratio; PTSD, posttraumatic stress disorder.

*P<.001.

†P<.01.

‡P<.05.

CO-OCCURRENCE OF PTS SYMPTOMS WITH OTHER PSYCHIATRIC DISORDERS

Co-occurrence rates between common psychiatric disorders, trauma exposure, and PTS symptoms are presented in **Table 4**. Children exposed to trauma had almost double the rates of psychiatric disorders of those not exposed. This effect was significant for all diagnostic groups except substance use disorders. Higher levels of PTS-related symptoms were associated with higher levels of psychiatric disorders, with rates of 52.6% and 59.5% for painful recall and subclinical PTSD, respectively. For
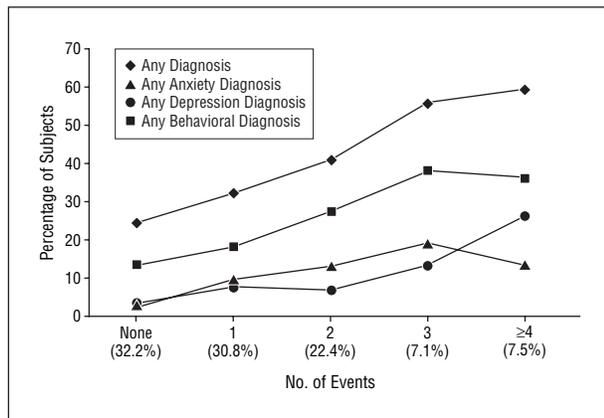
Figure. Effect of increasing trauma exposures on cumulative rates of psychiatric diagnoses by age 16 years.

both symptom groups, co-occurrence rates were significant across a range of disorders, although the strength of the association varied, suggesting both a general and a specific pattern. Co-occurence odds ratios (ORs) were highest for affective disorders and lower for substance use and disruptive behavior disorders. The **Figure** suggests that the dose-dependent relation between trauma and psychiatric disorders was similar to that observed for trauma and PTS symptoms. This pattern, significant for most diagnostic groups, was most pronounced for depressive disorders.

FIRST TRAUMATIC EXPERIENCE

Risk factors significantly predicting first trauma exposure in the model were previous (1-year–lagged) environmental adversity (OR, 2.2; 95% confidence interval [CI], 1.4-3.5), previous parenting problems (OR, 1.8; 95% CI, 1.1-2.8), and history of a depressive disorder (OR, 3.1; 95% CI, 1.0-9.8). The same constellation of vulnerability factors showed trends toward predicting painful recall, but only previous exposure to nontraumatic life events was significant (OR, 2.4; 95% CI, 1.6-9.6). Overall, 8.2% of the studied individuals reported painful recall and 1.4% reported subclinical PTSD in response to their first trauma exposure.

Rates of psychopathology were compared between individuals reporting PTS symptoms in response to their first trauma and those with no symptoms. One year before trauma exposure, psychopathology rates did not differ between groups (13.6% for trauma exposure only vs 17.6% for trauma exposure and PTS symptoms; F_1=0.81; P=.37). No differences were noted for anxiety or depressive disorders before trauma exposure. Immediately after trauma exposure, rates of psychiatric disorders were higher in the group experiencing painful recall (31.1% vs 14.5%; F_1=8.82; P=.003). The significant difference reflected increased levels of anxiety disorders in the group with painful recall (2.2% pretrauma vs 16.0% immediately following trauma exposure). One year after trauma exposure, the 2 groups did not differ on rates of psychopathology (F_1=0.05; P=.81). Overall, most children experienced few PTS symptoms in response to their initial trauma exposure,

and those experiencing PTS symptoms were also at highest risk of psychiatric morbidity.

COMMENT

The analyses of longitudinal data from a community-based sample of children and adolescents showed that, first, although exposure to traumatic events was almost commonplace, full-blown *DSM-IV* PTSD was rare across middle childhood and adolescence. Symptoms of PTS, including painful recall and subclinical PTSD, were more common, but very far from being expectable sequelae. Second, children displaying PTS symptoms in response to trauma exposure were more likely to be older, to have a history of exposure to trauma, to have a history of anxiety, and to come from an adverse family environment. Third, higher levels of trauma exposure were related to higher levels of most types of psychopathology, particularly anxiety and depressive disorders, as well as other impairments. Finally, the prognosis after a first lifetime trauma exposure was generally favorable.

In this report, as in an earlier report covering a more limited period,[30] our estimates of lifetime trauma exposure in childhood and adolescence are generally slightly higher than in previous community-based studies.[3,4] This is attributable to a number of study characteristics intended to improve accuracy of reporting. First, subjects were interviewed at least 4 times during childhood and adolescence about the immediate past, rather than relying on retrospective recall in adulthood. At each assessment point, both the parent and the child were interviewed. Each interviewee was asked about each type of traumatic event (17 in all) separately, whereas some studies have asked general questions about trauma exposure with a few examples.

The rate of PTSD after exposure to a traumatic event was lower than that reported in studies of adults.[31,32] At the same time, our results suggest that these children experienced PTS symptoms, higher rates of psychopathology, and additional impairments. One explanation for these findings has to do with the *DSM-IV* criteria themselves. These criteria were developed from the adult PTSD literature[33] and may not accurately reflect severe responses to trauma in children. Childhood studies indicate low reliability for PTS symptoms[34,35] and low diagnostic efficacy for the arousal symptoms,[36] and factor analytic studies have often failed to support the 3-symptom clusters of painful recall, arousal, and avoidance described in the adult literature.[37,38] Furthermore, research with children suggests that the optimal algorithm for PTSD may require substantially fewer symptoms than is required for diagnosis of the disorder in adults.[39-41] These studies suggest that different symptom clusters and different levels of symptoms are needed to predict impairment in childhood samples. Although the present study did not intend to evaluate the current *DSM-IV* PTSD criteria, the findings suggest that the current criteria, when applied to children, may not be developmentally sensitive or that childhood PTSD is rare.

Psychopathology is strongly interrelated with trauma and trauma symptoms. Across childhood, the children

who experience trauma are often those with anxiety, depressive, and disruptive behavior disorders, a finding supported in the present study. This likely reflects common liability conveyed from a limited set of family risk factors.[42] Furthermore, psychopathology, particularly depression and anxiety, can serve as a risk for and sequela of trauma exposure.[43,44] Our study indicated some specificity in the role of psychopathology as risk for trauma and trauma exposure. Past depression best predicted first trauma, but it was a history of anxiety disorders that best predicted PTS symptoms in response to trauma exposure. Both of these disorders are also common sequelae of trauma exposure, with rates increasing dramatically immediately after the first trauma exposure. This relationship is strongest in individuals who also display some PTS symptoms (ie, at least painful recall).

Among potential sources of bias, a previous report suggested little evidence of symptom attenuation (lower reported symptom levels in subsequent data waves), cohort differences, and differential dropout in this sample.[45] Conversely, if bias were inflating estimates of traumatic events, this would likely be reflected in similarly elevated psychopathology; however, psychiatric prevalence rates reported from this sample are consistent with rates obtained from other community-based studies.[46,47] On the other hand, the study may underestimate lifetime rates of traumatic events because interviews began when children were already in middle childhood.

A number of sources of bias may be specific to the life events and PTSD module. Our screening structure was intended to minimize cases in which the full PTSD module is completed unnecessarily. The screening structure requires the presence of painful recall symptoms (including nightmares, thoughts, or images) before inquiring about avoidance and hyperarousal symptoms. This decision, made to increase diagnostic efficacy, gave some primacy to painful recall during the less specific hyperarousal and avoidance symptoms. Subsequent studies support this decision, suggesting that symptoms involving bad dreams and repetitive thoughts have the highest diagnostic efficacy for predicting full-blown PTSD in children, along with behavioral and emotional avoidance symptoms.[48,49] Hyperarousal symptoms, by contrast, have the lowest levels of diagnostic efficacy.

There was no independent verification of the occurrence of the traumatic events reported; instead, we relied on information from the parent and/or the child. This could bias the estimate of the number of potentially traumatic events leading to PTSD because informants may forget events that had no emotional sequelae or suppress events that caused PTS symptoms. This is probably an unavoidable problem in community-based studies of PTSD that do not follow a specific event, such as a hurricane or flood, because it is practically impossible to verify not only the events reported but also exposure to events not reported. The advantage that, we believe, outweighs this drawback to general population studies is that we were able to examine the interplay of multiple different types of events, over time, on the risk of PTSD.

Severe events such as sexual abuse may be underreported. In studies assessing for events at multiple time points, it is not uncommon for an event reported at 1 time point to be followed by a false-negative report.[50,51] Those results support the methods used in the current study of assessing severe events at multiple time points. Also, mandated reporting requirements might suppress reporting for physical and sexual abuse, events associated with higher rates of PTS symptoms.

Finally, our study used subcategories of PTS symptoms (ie, subclinical PTSD and painful recall only) to identify children with 2 symptom levels that might influence functioning. Other categories could have been used (eg, *DSM-IV* criteria A, B, and E and criterion C or D), and prevalence rates and other results would vary depending on the stringency of the criteria. However, increasing evidence suggesting that children with an impairing response to trauma may be characterized by fewer symptoms supports the use of categories with relatively minimal requirements such as those used in this study.[37,40]

CONCLUSIONS

Studies of childhood trauma that use convenience samples of children exposed to specific events and undergoing assessment for PTS symptoms only provide incomplete answers to questions about how common trauma is in childhood and how children typically respond to potentially traumatic events. The present study followed up a large community sample of children through middle childhood to adolescence with repeated assessments for trauma exposure and a range of potential responses. The findings suggest that the effects of trauma are not symptom specific. Few children exposed to trauma develop PTSD, and the few who display PTS symptoms can be identified through information about their age, trauma history, anxiety history, and family environment. Children exposed to traumatic events also displayed higher rates of depression, anxiety disorders, and other impairments.

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REFERENCES

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition). Washington, DC: American Psychiatric Press; 1994.
2. March JS. What constitutes a stressor? The "Criterion A" issue. In: Davidson JRT, Foa E, eds. *Posttraumatic Stress Disorder: DSM-IV and Beyond*. Washington, DC: American Psychiatric Press Inc; 1993:37-54.
3. Giaconia RM, Reinherz HZ, Silverman AB, Pakiz B, Frost AK, Cohen E. Traumas and posttraumatic stress disorder in a community population of older adolescents. *J Am Acad Child Adolesc Psychiatry*. 1995;34:1369-1380.
4. Cuffe SP, Addy C, Garrison CZ, Waller JL, Jackson KL, McKeown RE, Chilappagari S. Prevalence of PTSD in a community sample of older adolescents. *J Am Acad Child Adolesc Psychiatry*. 1998;37:147-154.
5. Gold SL. *An Investigation of Posttraumatic Stress Disorder and the Lives of Formerly Homeless Children*. Fresno: California School of Professional Psychology; 1995.
6. Nader K, Pynoos R, Fairbanks L, Frederick C. Children's PTSD reactions one year after a sniper attack at their school. *Am J Psychiatry*. 1990;147:1526-1530.
7. Pynoos RS, Goenjian A, Tashjian M, Karakashian M, Manjikian R, Manoukian G, Steinberg AM, Fairbanks LA. Post-traumatic stress reactions in children after the 1988 Armenian earthquake. *Br J Psychiatry*. 1993;163:239-247.
8. Ahmad A, Sofi M, Sundelin-Wahlsten V, von Knorring A. Posttraumatic stress disorder in children after the military operation "Anfal" in Iraqi Kurdistan. *Eur Child Adolesc Psychiatry*. 2000;9:235-243.
9. March JS, Amaya-Jackson L, Terry R, Costanzo P. Posttraumatic symptomatology in children and adolescents after an industrial fire. *J Am Acad Child Adolesc Psychiatry*. 1997;36:1080-1088.
10. McDermott BM, Lee EM, Judd M, Gibbon P. Posttraumatic stress disorder and general psychopathology in children and adolescents following a wildfire disaster. *Can J Psychiatry*. 2005;50:137-143.
11. Sack WH, Clarke GN, Him C, Dickason D, Goff B, Lanham K, Kinzie JD. A 6-year follow-up study of Cambodian refugee adolescents traumatized as children. *J Am Acad Child Adolesc Psychiatry*. 1993;32:431-437.
12. Fairbank JA, Ebert L, Costello EJ. Epidemiology of traumatic events and posttraumatic stress disorder. In: Nutt D, Davidson JRT, Zohar J, eds. *Posttraumatic Stress Disorder: Diagnosis, Management and Treatment*. London, England: Martin Dunitz Ltd; 2000:17-27.
13. Maughan B, Rutter M. Retrospective reporting of childhood adversity: issues in assessing long-term recall. *J Personal Disord*. 1997;11:19-33.
14. Patten SB. Recall bias and major depression lifetime prevalence. *Soc Psychiatry Psychiatr Epidemiol*. 2003;38:290-296.
15. Coughlin SS. Recall bias in epidemiologic studies. *J Clin Epidemiol*. 1990;43:87-91.
16. Pillemer DB, White SH. Childhood events recalled by children and adults. *Adv Child Dev Behav*. 1989;21:297-340.
17. Harlow SD, Linet MS. Agreement between questionnaire data and medical records: the evidence for accuracy of recall. *Am J Epidemiol*. 1989;129:233-248.
18. Som RK, ed. *Recall Lapse in Demographic Enquiries*. New York, NY: Asia Publishing House; 1973.
19. Raphael KG, Cloitre M, Dohrenwend BP. Problems of recall and misclassification with checklist methods of measuring stressful life events. *Health Psychol*. 1991;10:62-74.
20. Widom CS, Shepard S. Accuracy of adult recollections of childhood victimization, I: childhood physical abuse. *Psychol Assess*. 1996;8:412-421.
21. Costello EJ, Angold A, Burns BJ, Stangl DK, Tweed DL, Erkanli A, Worthman CM. The Great Smoky Mountains Study of Youth: goals, designs, methods, and the prevalence of DSM-III-R disorders. *Arch Gen Psychiatry*. 1996;53:1129-1136.
22. Costello EJ, Farmer E, Angold A, Burns B, Erkanli A. Psychiatric disorders among American Indian and white youth in Appalachia: the Great Smoky Mountains Study. *Am J Public Health*. 1997;87:827-832.
23. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research: Principles and Quantitative Methods*. New York, NY: Van Nostrand Reinhold; 1982.
24. Angold A, Costello EJ. The Child and Adolescent Psychiatric Assessment (CAPA). *J Am Acad Child Adolesc Psychiatry*. 2000;39:39-48.
25. Angold A, Costello EJ. A test-retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment (CAPA-C). *Psychol Med*. 1995;25:755-762.
26. Costello EJ, Messer SC, Reinherz HZ, Cohen P, Bird HR. The prevalence of serious emotional disturbance: a re-analysis of community studies. *J Child Fam Stud*. 1998;7:411-432.
27. Costello EJ, Angold A, March J, Fairbank J. Life events and post-traumatic stress: The development of a new measure for children and adolescents. *Psychol Med*. 1998;28:1275-1288.
28. SAS Institute. *SAS/STAT User's Guide, Version 6*. Vol 1. 4th ed. Cary, NC: SAS Institute Inc; 1994.
29. Raferty A. Choosing models for cross-classifications. *Am Sociol Rev*. 1986;51:145-146.
30. Costello EJ, Erkanli A, Fairbank JA, Angold A. The prevalence of potentially traumatic events in childhood and adolescence. *J Trauma Stress*. 2002;15:99-112.
31. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson C. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry*. 1995;52:1048-1060.
32. Breslau N, Davis GC, Andreski P, Peterson EL, Schultz LR. Sex differences in posttraumatic stress disorder. *Arch Gen Psychiatry*. 1997;54:1044-1048.
33. Summary of the practice parameters for the assessment and treatment of children and adolescents with posttraumatic stress disorder. *J Am Acad Child Adolesc Psychiatry*. 1998;37:997-1001.
34. Shannon MP, Lonigan CJ, Finch A, Taylor CM. Children exposed to disaster, I: epidemiology of post-traumatic symptoms and symptom profiles. *J Am Acad Child Adolesc Psychiatry*. 1994;33:80-93.
35. Vernberg EM, La Greca A, Silverman W, Prinstein M. Prediction of posttraumatic stress symptoms in children after Hurricane Andrew. *J Abnorm Psychol*. 1996;105:237-248.
36. Sack WH, Seeley JR, Clarke GN. Does PTSD transcend cultural barriers: a study from the Khmer Adolescent Refugee Project. *J Am Acad Child Adolesc Psychiatry*. 1997;36:49-54.
37. Anthony JL, Lonigan C, Vermbeg E, La Greca A, Silverman W, Prinstein M. Multisample cross-validation of a model of childhood posttraumatic stress disorder symptomatology. *J Trauma Stress*. 2005;18:667-676.
38. Anthony JL, Lonigan CJ, Hecht SA. Dimensionality of posttraumatic stress disorder symptoms in children exposed to disaster: results from confirmatory factor analyses. *J Abnorm Psychol*. 1999;108:326-336.
39. Scheeringa MS, Peebles CD, Cook CA, Zeanah CH. Toward establishing procedural, criterion, and discriminant validity for PTSD in early childhood. *J Am Acad Child Adolesc Psychiatry*. 2001;40:52-60.
40. Scheeringa MS, Zeanah C, Myers L, Putnam F. New findings on alternative criteria for PTSD in preschool children. *J Am Acad Child Adolesc Psychiatry*. 2003;42:561-571.
41. Carrion VG, Weems CF, Ray R, Reiss AL. Toward an empirical definition of pediatric PTSD: the phenomenology of PTSD symptoms in youth. *J Am Acad Child Adolesc Psychiatry*. 2002;41:166-173.
42. Kendler KS, Gardner C, Prescott C. Personality and the experience of environmental adversity. *Psychol Med*. 2003;33:1193-1202.
43. Kendler KS, Kuhn J, Prescott CA. The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *Am J Psychiatry*. 2004;161:631-636.
44. Bolton D, Hill J, O'Ryan D, Udwin O, Boyle S, Yule W. Long-term effects of psychological trauma on psychosocial functioning. *J Child Psychol Psychiatry*. 2004;45:1007-1014.
45. Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. Prevalence and development of psychiatric disorders in childhood and adolescence. *Arch Gen Psychiatry*. 2003;60:837-844.
46. Angold A, Erkanli A, Farmer EMZ, Fairbank JA, Burns BJ, Keeler G, Costello EJ. Psychiatric disorder, impairment, and service use in rural African American and white youth. *Arch Gen Psychiatry*. 2002;59:893-901.
47. Shaffer D, Fisher P, Dulcan MK, Davies M. The NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC 2.3): description, acceptability, prevalence rates, and performance in the MECA study: Methods for the Epidemiology of Child and Adolescent Mental Disorders Study. *J Am Acad Child Adolesc Psychiatry*. 1996;35:865-877.
48. Lonigan CJ, Anthony JL, Shannon MP. Diagnostic efficacy of posttraumatic symptoms in children exposed to disaster. *J Clin Child Psychol*. 1998;27:255-267.
49. Pynoos RS, Goenjian A, Tashjian M, Karakashian M, Manjikian R, Manoukian G, Steinberg AM, Fairbanks LA. Post-traumatic stress reactions in children after the 1988 Armenian earthquake. *Br J Psychiatry*. 1993;163:239-247.
50. Fergusson DM, Horwood LJ, Woodward LJ. The stability of child abuse reports: a longitudinal study of the reporting behaviour of young adults. *Psychol Med*. 2000;30:529-544.
51. Johnson JG, Cohen P, Brown J, Smailes EM, Bernstein DP. Childhood maltreatment increases risk for personality disorders during early adulthood. *Arch Gen Psychiatry*. 1999;56:600-606.