Prevalence and Development of Psychiatric Disorders in Childhood and Adolescence

E. Jane Costello, PhD; Sarah Mustillo, PhD; Alaattin Erkanli, PhD; Gordon Keeler, MS; Adrian Angold, MRCPsych

**Background:** This longitudinal community study assessed the prevalence and development of psychiatric disorders from age 9 through 16 years and examined homotypic and heterotypic continuity.

**Methods:** A representative population sample of 1420 children aged 9 to 13 years at intake were assessed annually for **DSM-IV** disorders until age 16 years.

**Results:** Although 3-month prevalence of any disorder averaged 13.3% (95% confidence interval [CI], 11.7%-15.0%), during the study period 36.7% of participants (31% of girls and 42% of boys) had at least 1 psychiatric disorder. Some disorders (social anxiety, panic, depression, and substance abuse) increased in prevalence, whereas others, including separation anxiety disorder and attention-deficit/hyperactivity disorder (ADHD), decreased. Lagged analyses showed that children with a history of psychiatric disorder were 3 times more likely than those with no previous disorder to have a diagnosis at any subsequent wave (odds ratio, 3.7; 95% CI, 2.9-4.9; P<.001). Risk from a previous diagnosis was high among both girls and boys, but it was significantly higher among girls. Continuity of the same disorder (homotypic) was significant for all disorders except specific phobias. Continuity from one diagnosis to another (heterotypic) was significant from depression to anxiety and anxiety to depression, from ADHD to oppositional defiant disorder, and from anxiety and conduct disorder to substance abuse. Almost all the heterotypic continuity was seen in girls.

**Conclusions:** The risk of having at least 1 psychiatric disorder by age 16 years is much higher than point estimates would suggest. Concurrent comorbidity and homotypic and heterotypic continuity are more marked in girls than in boys.

**Arch Gen Psychiatry. 2003;60:837-844**

Studies that follow the same subjects as they grow up are the best source of information about the prevalence and causes of continuity and discontinuity of psychiatric disorders. A review of the few studies that cover both child and adolescent psychiatric disorders showed that between 23% and 61% of children with a diagnosis at one wave had a diagnosis, although not necessarily the same one, at a subsequent wave. This suggests quite a high level of continuity. However, few studies have the power to distinguish between homotypic continuity (the same diagnosis at different assessments) and heterotypic continuity (continuity of disorder but a different diagnosis). Homotypic continuity is evidence for a disorder that has a similar manifestation across the age range of the study, whereas heterotypic continuity suggests an underlying vulnerability to psychiatric illness that may expose children to different disorders at different ages or an underlying disorder that has different manifestations at different ages. The clinical and research implications of homotypic and heterotypic continuity are quite different.

In this article we address the following questions about continuity for the age range 9 to 16 years: What is the prevalence of **DSM-IV** disorders at different ages? (Because **DSM-IV** is inconsistent in whether impairment is required for a diagnosis, we include the prevalence of severe emotional disturbance (SED), the term used by the federal government for psychiatric disorder accompanied by significant impairment in the child's functioning.) Does the prevalence of these disorders increase or decrease as children grow up? What are the patterns of comorbidity? Is there continuity of disorder across this developmental period? Which disorders predict which other disorders? And are there significant sex differences in any of these estimates?
The Great Smoky Mountains Study (GSMS) is a longitudinal study of the development of psychiatric disorder and the need for mental health services.12-17 Three cohorts of children, aged 9, 11, and 13 years at intake, were recruited from 11 counties in western North Carolina. A multistage sampling design was used, with weighting that returned the weighted sample to its original size.18 Potential participants were randomly selected from the population of some 20000 children using a household equal probability, accelerated cohort design.19 This means that each cohort reaches a given age in a different year, thus controlling for cohort effects.20 The initial random sample of 4067 yielded 3896 screening questionnaires (95%) consisting of most psychiatric symptoms is the past 3 months. In the Time frame of the CAPA for determining the presence of most psychiatric symptoms is the past 3 months. In the case of a few rare and severe acts, such as fire-setting or assault, a lifetime frame of reference is used, as required by DSM-IV. Two-week test-retest reliability of CAPA diagnoses in children aged 10 through 18 years is comparable to that of other highly structured child psychiatric interviews.19-23

**METHODS**

Interviewers were residents of the study area and had at least bachelor’s-level degrees. They received 1 month of training, constant monitoring of quality control, and instruction by Department of Social Services staff in North Carolina’s requirements for reporting abuse or neglect. All suspected cases were referred to the appropriate agency.

**DATA MANAGEMENT AND ANALYSIS**

Scoring programs for the CAPA, written in SAS statistical software (SAS Institute Inc, Cary, NC), combined information about the date of onset, duration, and intensity of each symptom to create diagnoses according to the fourth edition of the DSM-IV.28

**GENERATING POPULATION PREVALENCE AND VARIANCE ESTIMATES**

To generate population prevalence estimates from a multi-stage sampling design subjects were assigned a weight inversely proportional to their probability of selection. Correlation matrices were introduced to account for within-subject correlations. We used general estimation equations (GEEs)29 to account for both the sampling design and within-subject correlation. In GEE, subject is introduced as a cluster (class) variable, and the sampling weights are introduced as a scale vec-

---

**Table 1. Participant Age and Date of Interviews by Cohort**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>9</td>
<td>A1</td>
<td>95.2</td>
<td>90.5</td>
<td>88.0</td>
<td>78.0</td>
<td>80.3</td>
<td>75</td>
<td>75.2</td>
<td>80.7</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
<td>A2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>11</td>
<td>B1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>B2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>C1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>C2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>C3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>C4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Response rate, %

©2003 American Medical Association. All rights reserved.
tor 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 repeated measures. Identical results can be generated with SUDAAN using primary sampling units as the subject identifiers, along with sampling weights. To obtain the prevalence of each of the various psychiatric disorders we fitted logistic regression models, again using GEE, with no predictors but including the intercept only. We used the Delta method (Taylor series expansion) to generate overall and time-averaged prevalence estimates and standard errors, corrected for within-subject correlations and design effects. Statistical tests such as comparison of prevalence between sexes used the same approach.

Lagged analyses were used to answer the questions about homotypic and heterotypic continuity. Two sets of analyses were done, one looking at the effect of a diagnosis in the year immediately preceding any annual interview and one looking at the effect of a diagnosis at any interview before the current one. Although the effects were stronger when the 1-year lag was used, results are more generalizable using the entire preceding period, and so the latter are reported here. Logistic regression analyses regressed this year’s diagnosis on all past years, with other period, and so the latter are reported here. Logistic regression analyses regressed this year’s diagnosis on all past years, with other

tor 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 repeated measures. Identical results can be generated with SUDAAN using primary sampling units as the subject identifiers, along with sampling weights. To obtain the prevalence of each of the various psychiatric disorders we fitted logistic regression models, again using GEE, with no predictors but including the intercept only. We used the Delta method (Taylor series expansion) to generate overall and time-averaged prevalence estimates and standard errors, corrected for within-subject correlations and design effects. Statistical tests such as comparison of prevalence between sexes used the same approach.

Lagged analyses were used to answer the questions about homotypic and heterotypic continuity. Two sets of analyses were done, one looking at the effect of a diagnosis in the year immediately preceding any annual interview and one looking at the effect of a diagnosis at any interview before the current one. Although the effects were stronger when the 1-year lag was used, results are more generalizable using the entire preceding period, and so the latter are reported here. Logistic regression analyses regressed this year’s diagnosis on all past years, with other

Table 2 presents the 3-month prevalence of a range of DSM-IV diagnoses by age and sex. A fuller version of the table can be found on our Web site (http://www.devepi.mc.duke.edu; Web Table 1). In the case of depressive disorders, data are also presented on depressive disorder not otherwise specified, which includes the DSM-IV experimental category of minor depression. The overall prevalence of any disorder was highest in 9- to 10-year-olds, falling to its lowest level in 12-year-olds and then rising slowly (Figure). Twelve years was the age at which many of the disorders of childhood (ie, ADHD, separation anxiety disorder [SAD], enuresis and encopresis, and motor and verbal tic disorders) had almost disappeared, especially in boys, while those of adolescence and adulthood had not yet developed. Childhood disorders also decreased in girls across the same period but were less frequent to start with.
Concurrent comorbidity refers to the co-occurrence of cases of depression and anxiety disorders, conduct disorder (CD) and ADHD. Girls accumulated more disorders. The estimate for boys greatly exceeded that for girls, children had met cumulative prevalence by age 16 years. By then, 36.7% of diagnosed boys and 58% of diagnosed girls had SED. We estimated the cumulative prevalence of disorders and 31% of girls with a psychiatric disorder qualified as specifically in boys. At age 9 and 10 years only 20% of boys and 31% of girls with a psychiatric disorder qualified as significantly impaired by SED. By age 16 years, 79% of diagnosed boys and 58% of diagnosed girls had SED.

**ONSET OF DISORDERS BETWEEN AGES 9 AND 16 YEARS**

We estimated the cumulative prevalence of disorders across the age range of the study; that is, the accumulation of new cases in previously unaffected children. (Note that the individual may have had an episode before entry into the study.) Table 3 presents the predicted cumulative prevalence by age 16 years. By then, 36.7% of children had met DSM-IV criteria for 1 or more disorders. The estimate for boys greatly exceeded that for girls, as a result of a much higher cumulative prevalence of conduct disorder (CD) and ADHD. Girls accumulated more cases of depression and anxiety disorders.

**CONCURRENT COMORBIDITY**

Concurrent comorbidity refers to the co-occurrence of 2 or more diagnoses at the time of measurement, in this case, within the same 3-month period (A.A., A.E., E.J.C., and Helen Egger, MD, unpublished research, 2001). Table 4 presents the major types of concurrent comorbidity by sex. In bivariate analyses, 25 of 30 comparisons showed significant comorbidity. This fell to 16 of 30 after controlling for other comorbidities. There was significant comorbidity among the behavioral disorders and between anxiety and depression, as expected. The strong bivariate association between anxiety disorders and the behavioral disorders fell markedly and in most cases became nonsignificant when controlling for other forms of comorbidity. This was mainly because of the strong association between oppositional defiant disorder (ODD) and depression, on the one hand, and depression and anxiety, on the other.

There were 2 marked sex differences in patterns of concurrent comorbidity, both involving depression. After controlling for other comorbidity, depression was comorbid with CD in girls but not boys. Conversely, depression was comorbid with SUD in boys but not girls.

**HOMOTYPIC AND HETEROYPIC CONTINUITY**

Children with a history of psychiatric disorder were 3 times more likely than those with no previous disorder to have a diagnosis at any subsequent wave of data collection (28.0% vs 9.3%; odds ratio [OR], 3.7; 95% confidence interval [CI], 2.9-4.9; P<.001). Risk from a previous diagnosis was high in girls (OR, 5.2; 95% CI, 3.4-7.9; P<.001) and boys (OR, 2.9; 95% CI, 2.0-4.1; P<.001), but it was significantly higher in girls (interaction OR, 1.8; 95% CI, 1.0-3.7; P=.03). Within this general picture, there were marked differences in continuity from one diagnosis to the same one (homotypic continuity) or to another (heterotypic continuity).

Looking first at homotypic continuity, this was significant for every diagnosis except for specific phobias. The details can be found on our Web site (http://www.devepi.mc.duke.edu; Web Table 2). The disorders showing the highest level of continuity were panic disorders, psychosomatics, verbal tics, encopresis and enuresis, and SUDs. Girls showed more continuity than boys, even though they had fewer psychiatric disorders. Prediction from past episodes was significantly higher in girls for depression, GAD, social phobia, and specific phobia. On the other hand, encopresis showed continuity only in boys. There were no significant sex differences in continuity for the behavioral disorders or SUDs, despite their greater frequency in boys.

We next tested whether there were cases in which the presence of one disorder earlier in life increased the risk of a different diagnosis later, net of the effects of concurrent comorbidity. Models included both homotypic and heterotypic continuity and then tested whether adding a term for concurrent comorbidity affected heterotypic continuity. Results are summarized in Table 5.

There was strong heterotypic continuity from depression to anxiety, which was not greatly affected by the high level of concurrent comorbidity between the 2 disorders. Prediction from anxiety to depression also occurred, and remained significant, although with a lower OR when controlling for concurrent comorbidity. Anxiety predicted later SUDs, but depression did not. Among the disruptive behavior disorders, ADHD showed a modest but significant prediction to later ODD, even controlling for the high level of concurrent comorbidity between the two. Conduct disorder predicted both ADHD and SUD in the bivariate model, but the very high level of concurrent comorbidity explained this.

Separate analyses by sex showed that heterotypic continuity was much more common in girls. Anxiety predicted depression, and vice versa, even controlling for concurrent comorbidity, but this was not the case for boys. Similarly, the link between past anxiety or CD and cur-

---

**Table 3. Predicted Cumulative Prevalence of Psychiatric Disorders by Age 16 Years***

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Total</th>
<th>Girls</th>
<th>Boys</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any disorder</td>
<td>36.7 (.7)</td>
<td>31.0 (.3)</td>
<td>42.3 (.1)</td>
</tr>
<tr>
<td>Any emotional disorder</td>
<td>15.0 (.7)</td>
<td>17.1 (.7)</td>
<td>13.0 (.6)</td>
</tr>
<tr>
<td>Any anxiety disorder</td>
<td>9.9 (.5)</td>
<td>12.1 (.5)</td>
<td>7.7 (.4)</td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>9.5 (.1)</td>
<td>11.7 (.2)</td>
<td>7.3 (.1)</td>
</tr>
<tr>
<td>Any behavior disorder</td>
<td>23.0 (.7)</td>
<td>16.1 (.2)</td>
<td>29.9 (.2)</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>9.0 (.2)</td>
<td>3.8 (.7)</td>
<td>14.1 (.8)</td>
</tr>
<tr>
<td>Oppositional defiant disorder</td>
<td>11.3 (.0)</td>
<td>9.1 (.0)</td>
<td>13.4 (.0)</td>
</tr>
<tr>
<td>ADHD</td>
<td>4.1 (.7)</td>
<td>1.1 (.2)</td>
<td>7.0 (.1)</td>
</tr>
<tr>
<td>SUDs</td>
<td>12.2 (.6)</td>
<td>10.1 (.5)</td>
<td>14.3 (.7)</td>
</tr>
</tbody>
</table>

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; SUDs, substance use disorders.

*Data are given as the percentage of participants (SE).
in 5 will have a psychiatric disorder. This was also the conclusion that we reached on the basis of the first wave of data from GSMS, published in 1996.\textsuperscript{12}

Analyses of further waves from the same data set, presented here, lead us to revise this picture. The 1-in-5 estimate was true of the youngest children, but as they grew up the 3-month prevalence fell to a low of 8.3\% at age 12 years, before beginning to rise again in adolescence. This is consistent with the one other (cross-sectional) study that provides annual data by age group.\textsuperscript{30}

Earlier epidemiologic studies of children and adolescents in the 1980s and early 1990s tended to generate very varied, and sometimes very high, rates of some disorders, in particular anxiety disorders, ADHD, and in some cases depression.\textsuperscript{32,34} However, recent studies have tended toward greater agreement and, on the whole, lower population estimates.\textsuperscript{33,38,42}

Although there has never been a representative population survey of child psychiatric disorders in the US comparable with the National Comorbidity Survey of 15- to 54-year-olds,\textsuperscript{39} or the recent British national survey of 5- to 15-year-olds,\textsuperscript{39} researchers have patched together a picture of the prevalence of psychiatric disorders in American children of different ethnic groups and ages. This research has been reviewed several times,\textsuperscript{34-37} and a consensus has been reached that at any given time 1 child

**Table 4. Concurrent Comorbidity**\textsuperscript{†}

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Any Anxiety Disorder</th>
<th>Any Depressive Disorder</th>
<th>ADHD</th>
<th>Conduct Disorder</th>
<th>Oppositional Defiant Disorder</th>
<th>SUDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any anxiety disorder</td>
<td>25.1 (9.9-63.3)|</td>
<td>6.6 (2.9-15.4)|</td>
<td>2.5 (1.1-5.5)|</td>
<td>3.3 (1.7-6.5)|</td>
<td>0.6 (0.2-1.9)</td>
<td></td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>28.4 (14.3-55.9)|</td>
<td>8.0 (3.4-16.5)|</td>
<td>5.0 (2.3-11.2)|</td>
<td>20.7 (8.8-48.8)|</td>
<td>9.9 (3.1-31.5)</td>
<td></td>
</tr>
<tr>
<td>ADHD</td>
<td>7.7 (2.7-22.2)|</td>
<td>3.3 (0.7-15.1)|</td>
<td>3.7 (1.8-7.6)|</td>
<td>8.7 (4.6-16.4)|</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>3.5 (1.3-9.5)|</td>
<td>21.3 (9.0-50.5)|</td>
<td>41.3 (14.0-121.9)|</td>
<td>9.6 (3.3-17.3)|</td>
<td>7.2 (3.1-16.7)</td>
<td></td>
</tr>
<tr>
<td>Oppositional defiant disorder</td>
<td>0.3 (0.06-1.2)|</td>
<td>10.6 (2.0-54.7)|</td>
<td>3.9 (1.1-13.6)|</td>
<td>8.0 (4.5-14.0)|</td>
<td>5.7 (2.3-13.9)</td>
<td></td>
</tr>
<tr>
<td>SUDs</td>
<td>0.4 (0.9-1.9)|</td>
<td>3.5 (1.1-11.1)|</td>
<td>0.3 (0.04-2.4)|</td>
<td>23.4 (9.1-60.3)|</td>
<td>2.4 (1.1-5.2)</td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>2.9 (0.6-12.9)|</td>
<td>NC |</td>
<td>30.7 (12.7-73.8)|</td>
<td>0.4 (0.04-2.7)|</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 5. Homotypic and Heterotypic Continuity With and Without Controls for Comorbidity**\textsuperscript{*}

<table>
<thead>
<tr>
<th>Predicting to:</th>
<th>Past Depression</th>
<th>Past Anxiety</th>
<th>Past Conduct Disorder</th>
<th>Past ODD</th>
<th>Past ADHD</th>
<th>Past SUDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>7.0 (3.1-15.9)|</td>
<td>3.0 (1.7-5.4)|</td>
<td>11.2 (5.9-21.1)|</td>
<td>10.3 (4.3-24.7)|</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>Controlling for comorbidity</td>
<td>4.2 (2.1-8.3)|</td>
<td>2.7 (1.8-5.2)|</td>
<td>2.0 (1.1-3.8)|</td>
<td>2.1 (1.1-4.2)|</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.7 (2.2-14.5)|</td>
<td>2.4 (1.6-7.1)|</td>
<td>4.1 (1.7-10.2)|</td>
<td>29.4 (6.9-126.0)|</td>
<td>1.9 (0.7-4.6)</td>
<td></td>
</tr>
<tr>
<td>Controlling for comorbidity</td>
<td>2.8 (1.2-6.5)|</td>
<td>2.0 (1.2-3.4)|</td>
<td>841</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>10.3 (4.3-24.7)|</td>
<td>3.7 (2.2-6.2)|</td>
<td>10.7 (5.2-22.3)|</td>
<td>21.3 (6.3-72.5)|</td>
<td>25.7 (7.8-85.4)</td>
<td></td>
</tr>
<tr>
<td>SUDs</td>
<td>2.0 (1.2-3.5)|</td>
<td>2.7 (1.2-6.5)|</td>
<td>9.6 (4.4-21.2)|</td>
<td>21.3 (6.3-72.5)|</td>
<td>25.7 (7.8-85.4)</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** ADHD, attention-deficit/hyperactivity disorder; NC, model would not converge; SUDs, substance use disorders.

\*Data are given as the odds ratio (95\% confidence interval). Empty cells indicate that continuity without controls for comorbidity was not significant. Boldface type indicates homotypic continuity.

\[P<.05.\]

\[P<.01.\]

\[P<.001.\]
METHODOLOGICAL CONSIDERATIONS

The GSMS sample comes from a small (11-county) and predominantly rural area of the southeastern United States. There were no Asian American subjects, and only 8% were African American. Thus, it does not represent the American population. A recent study of another part of North Carolina, a largely rural area including a large percentage of African American youth, found remarkably similar rates of disorder in white and African American children. A recent British population study of youth aged 5 to 15 years produced quite similar results; for example, the prevalence of any anxiety disorder was 3.8% (GSMS, 2.4%); depression, 0.9% (GSMS, 2.2%); ADHD, 1.4% (GSMS, 0.9%); ODD, 2.9% (GSMS, 2.7%); and tic disorders, 0.1% (GSMS, 0.1%).

Despite the overlapping cohorts design, which used 3 age cohorts to permit age X cohort comparisons, there is no escaping the fact that in the later data waves of GSMS the children’s mean age is greater than it was in the earlier waves and the participants have been study subjects for longer. Thus, lower prevalence relative to cross-sectional studies may reflect (1) a tendency for subjects to report fewer symptoms in later interviews (i.e., “symptom attenuation”)13-35; (2) cohort differences, with the youngest cohort having more symptoms and the oldest cohort the fewest, controlling for age; and (3) differential dropout of children with and without psychiatric disorders. Another type of methodological challenge lies in the fact that the CAPA uses a 3-month window to estimate prevalence. If none of these methodological challenges is upheld, then we need to look for substantive reasons for the differences between this and earlier studies.

Symptom Attenuation

In a test-retest reliability study of 77 children aged 10 to 18 years using the CAPA, there was a very modest amount of attenuation between the first and second interviews; the difference was significant only for CD symptoms. The sample, however, had no 10-year-olds, so attenuation remains a possibility for the youngest children.

Cohort Differences

Analyses of symptom and diagnostic counts by cohort in GSMS (unpublished data) showed that the cohort differences were nonsignificant.

Differential Dropout

The response rates for the different waves of the study are reported in Table 1. Of 7944 possible interviews during the study period, 6675 (84%) were completed. Four subjects died, and 5.9% of participants only completed a single interview; these participants were no more or less likely than those who completed multiple interviews to have a diagnosis. Subjects who refused to be interviewed at one wave frequently returned later. This suggests that biased nonresponse is not a major reason for the observed prevalence rates.

Because the CAPA inquires only about the 3-month period preceding the annual interview, our estimate of cumulative prevalence may underestimate the number of cases occurring since the last interview, and thus the “burden of disease” across childhood and adolescence. There is no question that the CAPA underestimates cumulative prevalence. It misses cases that began and ended in the 9 months between interviews, including those for which some symptoms were present during the 3-month window, but not enough to reach the diagnostic threshold. Many psychiatric interviews adopt a 6-month, 12-month, or even lifetime time frame for estimating the prevalence of psychiatric disorders. However, the evidence from studies using longer time frames suggests that a lot of forgetting occurs; for example, in the National Comorbidity Survey the lifetime rate for 15- to 54-year-olds (48.0%) was less than double the 12-month rate (29.5%). Clearly, both approaches underestimate the burden of psychiatric disorder. The CAPA uses a 3-month time frame because of the strong psychometric evidence that memory for the type of phenomena sought in psychiatric interviews is highly unreliable further back than 3 months. Ideally, study subjects would be interviewed every 3 months, but the burden imposed on subjects would introduce another set of biases.

Substantive Reasons

Controlling for wave, the 9- and 10-year-olds had significantly more symptoms and diagnoses than any other age. This was particularly true of the younger boys. As noted earlier, 9- and 10-year-old boys had very high rates of enuresis (9.5%), ADHD (3.6%), tic disorders (5.5%), and SAD (4.1%), all of them disorders of childhood that had fallen by more than 50% by age 11 years and disappeared by age 16 years. Excluding these diagnoses, the 3-month prevalence of psychiatric disorder for 9- and 10-year-old boys would fall to 8.3% (95% CI, 5.2%-12.9%).

In summary, bias is always a risk in survey research designed to estimate the population prevalence of a disorder. We have guarded against it to some extent by choosing an interview that shows minimal attenuation effects, maintaining a good response rate over 8 years of data collection, and adopting appropriate statistical controls for the sampling design and the use of correlated data across waves. The result is an estimate of the 3-month prevalence of psychiatric disorder that is lower than our wave 1 estimate largely because of the fall in disorders of childhood: enuresis and encopresis, ADHD, tic disorders, and SAD.

PREVALENCE OVER TIME: THE BURDEN OF DISEASE

Multiple waves of data show that although only 13.3% of children, on average, had a diagnosis at any measurement point, almost 3 times this number had 1 or more disorders over the period of the study. This means that single-wave, cross-sectional studies are likely to under-
estimate the burden of disease\textsuperscript{46} in the general population of children over time.

**COMORBIDITY AND DEVELOPMENTAL CONTINUITY**

As with other cross-sectional\textsuperscript{30,38,39} and longitudinal\textsuperscript{5,9} studies, we found considerable concurrent comorbidity: 25.5\% of children with a diagnosis had 2 or more. It appears that much of the association between CD/ODD and ADHD is in fact carried by ODD rather than CD.\textsuperscript{30} In both sexes, the high level of association between CD and ADHD fell noticeably when other diagnoses were included in the model, whereas the association between ADHD and ODD did not. Concurrent comorbidity between anxiety and depression was, as expected, strong in both sexes. Comorbidity between depression and ODD was also strong, but comorbidity between depression and CD was significant only for girls, when other types of comorbidity were controlled.

The data presented here on continuity of disorder across time suffer from the same shortcomings as do the cumulative prevalence figures: we have only a series of snapshots on which to base our estimates. Given that, the degree of homotypic continuity is remarkable. In bivariate analyses, every DSM-IV diagnosis examined showed significant homotypic continuity with the exception of specific phobias. Apart from encopresis, which rarely occurred in girls, continuity was higher in girls than in boys.

Compared with the level of homotypic continuity, there were few cases of heterotypic continuity. Heterotypic continuity was much stronger in girls than in boys. This suggests that the DSM-IV taxonomy may fit boys’ developmental patterns better than those of girls. There is no evidence that boys with an emotional disorder were at increased risk of a behavioral disorder, or vice versa, whereas girls with anxiety disorders had increased risk for later SUDs. (See Kaplow et al\textsuperscript{57} for a more detailed examination of this issue.) Robins and Price’s prediction\textsuperscript{59} that later SUDs will be predicted by CDs holds here for girls but not for boys. This sample is too young to test the prediction of Zoccolillo et al\textsuperscript{59} that CD in girls is likely to lead to later depression and somatization disorders.

An important issue that exceeded the scope of this article is developmental continuity among the anxiety disorders and between specific anxiety disorders and other diagnoses. Continuity was much higher for some anxiety disorders (panic disorder and posttraumatic stress disorder) than for others (specific phobia and GAD) (http://www.devepi.mc.duke.edu; Web Table 2). Much more work needs to be done on comorbidity and heterotypic continuity among these disorders and with depression.

In summary, data on a representative population of children and adolescents growing up in the 1990s show that at any time 1 in 6 will have a psychiatric disorder and at least 1 in 3 will have 1 or more psychiatric disorders by age 16 years. As children grow older, psychiatric disorders are more and more likely to be accompanied by significant functional impairment. Once children, particularly girls, develop a psychiatric disorder their chances of continuing to have one, or of developing another episode after remission, are much higher than those of their unaffected peers. By mid-adolescence, although some disorders of childhood have disappeared, impairing adult disorders such as depression, panic disorder, and SUDs are becoming the most prevalent problems. Much more work on the childhood antecedents of these disorders is needed if prevention programs are to be effective.

Submitted for publication September 4, 2002; final revision received January 13, 2003; accepted January 22, 2003.

This study was supported by grants DA11301 and MH57761 and Independent Scientist Award MH01167 from the National Institutes of Health, Bethesda, Md, and Faculty Scholar awards from the William T. Grant Foundation, New York, NY (Drs Costello and Angold).

Corresponding author and reprints: E. Jane Costello, PhD, Center for Developmental Epidemiology, Department of Psychiatry and Behavioral Sciences, Duke University Medical School, DUMC Box 3454, Durham, NC 27710 (e-mail: jcostello@psych.mc.duke.edu).

**REFERENCES**


27. Wing JK, Babor T, Brugha T, Burke J, Cooper JE, Giel R, Jablenski A, Regier D, Sartorius N, SCAN: Schedules for Clinical Assessment in Neuropsychiatry. Arch Gen Psychiatry. 1990;47:589-593.


