Trends in psychopathology across the adolescent years: What changes when children become adolescents, and when adolescents become adults?

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Background: Little is known about changes in the prevalence of psychiatric disorders between childhood and adolescence, and adolescence and adulthood. Methods: We reviewed papers reporting prevalence rates of psychiatric disorders separately for childhood, adolescence, and early adulthood. Results: About one adolescent in five has a psychiatric disorder. From childhood to adolescence there is an increase in rates of depression, panic disorder, agoraphobia, and substance use disorders (SUD), and a decrease in separation anxiety disorder (SAD) and attention-deficit hyperactivity disorder (ADHD). From adolescence to early adulthood there is a further increase in panic disorder, agoraphobia, and SUD, and a further decrease in SAD and ADHD. Other phobias and disruptive behavior disorders also fall. Conclusions: Further study of changes in rates of disorder across developmental stages could inform etiological research and guide interventions. Keywords: Epidemiology, adolescence, psychiatric disorders, continuity.

Introduction
Words for adolescence have been around since the fifteenth century to describe the period of growing to maturity, but the concept of adolescence as a special phase of life, different from both childhood and adulthood, has been seriously examined only in the last 100 years (Dahl & Hariri, 2005). Adolescence is generally agreed to be the period between puberty and legal adulthood, but neither of these occurs at a fixed point. Over the past 100 years sexual maturation has begun ever earlier, while legal independence varies by country or state and topic (e.g., drinking, marrying, voting; Liu, Albertsson Wikland, & Karlberg, 2000; Parent et al., 2003). At the same time, employers’ demands for a more educated labor force have expanded the years spent in education, lengthening dependence on parents. This means that the gap between sexual and social maturation – a definition of adolescence that parents will certainly recognize – has grown not only longer but also more of a challenge both for adolescents themselves and for the community.

Understanding the impact of passing through adolescence on rates of psychiatric disorder can be helpful in two ways. First, it can be informative about the degree of societal burden caused by different disorders at various developmental stages (Murray & Lopez, 1996). This ‘public health’ aspect of epidemiology can help with allocating resources for treatment and prevention. Second, learning more about the causes of developmental change in rates of psychopathology can help to provide ideas for interventions, and in the process can fine-tune our research questions about etiology. For example, there may be different risk factors associated with (a) childhood depression; (b) persistence of childhood depression into adolescence; (c) adolescent depression; (d) persistence of adolescent depression into adulthood; and (e) adult-onset depression. Furthermore, there may be times, and adolescence is an example (Copeland, Shanahan, Costello, & Angold, 2009), when the normal patterns of risk–disorder association are temporarily different from the patterns seen before and after. If we understood more about these transitions, we might be in a better position to try out developmentally sensitive methods of prevention and treatment. At the same time, because every intervention is, implicitly or explicitly, a test of a causal hypothesis, we could learn more about the etiology, course, and consequences of different disorders by testing the effects of different interventions at different developmental stages. A fascinating example is the research from 30 years ago showing that depressed adolescents are, like adults, responsive to monoamine oxidase inhibitors but not, unlike adults, to tricyclic antidepressants (Ryan et al., 1987).

This review of trends in psychopathology across adolescence is intended to provide some background.
information on the epidemiology of psychiatric disorders across the transitions into and out of adolescence. There are three main reasons for the focus on epidemiologic studies. First, it is rarely possible to generalize from clinical studies to either prevalence rates or causal explanations, beyond that particular study. This is because the patients who attend for treatment are often very different from members of the general population with the same disorder who have either not sought or not been able to gain access to treatment (Costello, PescoSolido, Angold, & Burns, 1998; Kapphahn, Morreale, Rickert, & Walker, 2006; Kleinbaum, Kupper, & Morgenstern, 1982; Zuvekas & Taliaferro, 2003). Second, it is very rare for clinical research to cover a long enough period of time to uncover developmental changes. Third, developmental changes may be obscured by treatment effects.

Our focus has been narrowed further by the decision to concentrate on studies that make psychiatric diagnoses, rather than simply reporting symptoms. Scale scores (some studies of course provide both). The reason for this is that the interpretation of change in mean symptom scores is often unclear. A fall in mean symptom scores from one developmental period to another may occur because everyone’s score is lower, or because scores have fallen for particular groups (e.g., the lowest or highest scoring group; see Angold, Erkanli, Silberg, Eaves, & Costello, 2002). Third, if a ‘case’ is defined in distributional terms (as, e.g., someone scoring in the top 10% of the range), the case rate in a longitudinal study will stay the same from one developmental period to another.1

One result of these decisions is greatly to reduce the number of studies published before about 1995 that meet criteria for this review. As these have been reviewed elsewhere (Costello & Angold, 2009; Costello, Egger, & Angold, 2005; Costello, Foley, & Angold, 2006b), and in any case mainly use different taxonomies [Diagnostic and Statistical Manual of Mental Disorders (DSM–III or DSM–IIIR or International Classification of Diseases (ICD)-9], the present review concentrates on work published in the past 15 years.

### Methods

We review the data available in published form, and also include unpublished data from our own ongoing longitudinal study, about (a) the prevalence of psychiatric disorders in adolescence; (b) how these rates change as young people move into and out of adolescence; (c) patterns of continuity and discontinuity across adolescence. Finally, we raise the question of what might explain the observed changes in rates.

Ideally, we would study change and persistence of psychiatric disorders by following representative samples from childhood into adolescence, and from adolescence into adulthood. Unfortunately there are few datasets that do this, and there are even fewer whose data is published in a way that enables us to study these transitions. There is also the risk that historical events occurring as participants in longitudinal studies grow up may bias rates of disorder (e.g., differences in drug use observed in a youth of the same age some of whom were interviewed before and some after the attacks of September 11, 2001; Costello, Erkanli, Keeler, & Angold, 2004).

In addition to longitudinal studies, cross-sectional studies that present their data separately for children, adolescents, and adults can also be used. As these data refer to different samples at different ages assessed at the same time, such studies do not risk ‘period’ effects but do run the risk of differences in rates caused by the fact that these are different people. A third possibility is to compare child, adolescent, and adult rates from different studies. Unfortunately, reported rates vary so widely across studies (Costello, 2009) that the results of such comparisons are unreliable. This review of developmental change is thus restricted to longitudinal and cross-sectional studies that present their data in such a way that we can examine rates for children and adolescents, or adolescents and adults, or both.

Different methods of data collection can generate very different rates of the same disorder in the same age group (Costello, Erkanli, & Angold, 2006a). In this article, rather than presenting actual reported prevalence rates (which are in any case often not available) we have noted for each study whether rates increased, decreased, or stayed the same across the age transition. It is usually not possible to test the size of the increase or decrease, because most studies have not reported confidence intervals or standard errors. To provide a general sense of the significance of increases or decreases, differences that halve or double the previous rate are indicated in bold.

For transitions from childhood to adolescence, for which there are more data, we have reported results if we could find at least two datasets covering a diagnosis. For the transitions from adolescence to adulthood this was not always possible. Where available we have shown whether the size of the difference from one developmental stage to the next was similar for both boys and girls.

### Results

**Prevalence of psychiatric disorders in adolescence**

Table 1 summarizes prevalence rates of the more common disorders reported in the past 15 years by epidemiological studies providing estimates

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1 This caveat does not necessarily apply to cross-sectional studies with a wide age range in the sample, but it does apply to the methodologically stronger studies with repeated measures of the same subjects.
specifically for adolescence – which we define as spanning the age range of 12–19 years. Studies that include adolescence but do not provide information specifically for a group in the age range of 2–19 years were excluded. Across these studies, the average rate of any adolescent psychiatric disorder was 21.8%, with an interquartile range of 14.8–22.8%. Drug abuse or dependence was the most common diagnosis in this age group (M: 12.1%; interquartile range: 3.3–18.3%), followed by anxiety disorders (M: 10.7%; interquartile range: 5.5–14.9%) and depressive disorders (6.1%; interquartile range: 3.1–7.2%). On average, studies identified between 3% and 4% of adolescents as suffering from any one of the behavioral disorders [conduct disorder (CD), oppositional defiant disorder (ODD), or attention-deficit hyperactivity disorder (ADHD)].

Psychosis. The British National Survey of Psychiatric Morbidity (Johns et al., 2004) found that 5.0% of a sample (N = 8,580) aged 17–74 years had one or more psychotic symptoms, which is similar to the prevalence of any definite psychotic symptoms (5.9%) reported from the Environmental Risk (E-Risk) Longitudinal Twin Study (N = 2,127) at age 12 (Polanczyk et al., 2007). The prevalence of rare disorders (those with a point prevalence of less than 1%) is hard to estimate unless samples are very large, which few adolescent epidemiological samples are. It is even harder to find estimates of change in prevalence levels of disorders such as eating disorders, panic disorder with or without agoraphobia, developmental, psychotic, or bipolar disorders. Thus, the British Child and Adolescent Mental Health Survey of 1999, with a sample of over 10,000, found that only 0.5% of adolescents (11–15 years) had one or more ‘less common disorders’ (PDD (pervasive developmental disorders), psychotic disorders, tic disorders, and eating disorders; Ford, Goodman, & Meltzer, 2003). However, the ‘rare disorders’ tend to be impairing and of clinical concern. Here we very briefly review estimates of these rare disorders in adolescence, with the caveat that confidence intervals around the estimate tend to be very wide.

### Table 1: Prevalence of any psychiatric disorder in adolescents, from studies published since 1997

<table>
<thead>
<tr>
<th>Study</th>
<th>Year of Publication</th>
<th>Nationality</th>
<th>Instrument</th>
<th>Time frame</th>
<th>Taxonomy</th>
<th>Age range</th>
<th>Number of subjects</th>
<th>Prevalence of any psychiatric disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verhulst, Van Der Ende, Ferdinand, and Kasius, 1997</td>
<td>1997</td>
<td>Holland</td>
<td>DISC</td>
<td>6 months</td>
<td>DSM-III-R</td>
<td>13–18</td>
<td>780</td>
<td>35.5</td>
</tr>
<tr>
<td>Canals et al., 1997</td>
<td>1997</td>
<td>Spain</td>
<td>SCAN</td>
<td>12 months</td>
<td>DSM-III-R</td>
<td>18</td>
<td>290</td>
<td>20.6</td>
</tr>
<tr>
<td>Krueger et al., 1998</td>
<td>1998</td>
<td>New Zealand</td>
<td>DIS</td>
<td>12 months</td>
<td>DSM-III-R</td>
<td>18</td>
<td>930</td>
<td>44</td>
</tr>
<tr>
<td>Lewinsohn et al., 1998</td>
<td>1998</td>
<td>United States</td>
<td>K-SADS</td>
<td>Current</td>
<td>DSM-III-R</td>
<td>14–18</td>
<td>1,709</td>
<td>9.6</td>
</tr>
<tr>
<td>Lewinsohn et al., 1998</td>
<td>1998</td>
<td>United States</td>
<td>K-SADS</td>
<td>Current</td>
<td>DSM-III-R</td>
<td>15–19</td>
<td>1,507</td>
<td>7.8</td>
</tr>
<tr>
<td>Meltzer et al., 1999</td>
<td>1999</td>
<td>United Kingdom</td>
<td>DAWBA</td>
<td>Current</td>
<td>DSM-IV</td>
<td>11–15</td>
<td>4,609</td>
<td>11.2</td>
</tr>
<tr>
<td>Rueter et al., 1999</td>
<td>1999</td>
<td>United States</td>
<td>UM-CIDI</td>
<td>5 years</td>
<td>DSM-III-R</td>
<td>15–19</td>
<td>303</td>
<td>1</td>
</tr>
<tr>
<td>Johnson et al., 2000</td>
<td>2000</td>
<td>United States</td>
<td>DISC</td>
<td>6 months</td>
<td>DSM-III-R</td>
<td>12–16</td>
<td>688</td>
<td>2</td>
</tr>
<tr>
<td>Ferguson and Horwood, 2001</td>
<td>2001</td>
<td>New Zealand</td>
<td>CIDI</td>
<td>12 months</td>
<td>DSM-III-R</td>
<td>15</td>
<td>1,000</td>
<td>22</td>
</tr>
<tr>
<td>Ferguson and Horwood, 2001</td>
<td>2001</td>
<td>New Zealand</td>
<td>CIDI</td>
<td>12 months</td>
<td>DSM-III-R</td>
<td>18</td>
<td>1,000</td>
<td>42</td>
</tr>
<tr>
<td>Romano et al., 2001</td>
<td>2001</td>
<td>Canada</td>
<td>DISC</td>
<td>6 months</td>
<td>DSM-III-R</td>
<td>14–17</td>
<td>2,000</td>
<td>20.1</td>
</tr>
<tr>
<td>Costello et al., 2003</td>
<td>2003</td>
<td>United States</td>
<td>CAPA</td>
<td>3 months</td>
<td>DSM-IV</td>
<td>13–16</td>
<td>1,420</td>
<td>22.8</td>
</tr>
<tr>
<td>Gau et al., 2005</td>
<td>2005</td>
<td>Taiwan</td>
<td>K-SADS-E</td>
<td>3 months</td>
<td>DSM-IV</td>
<td>13–15</td>
<td>1,070</td>
<td>20.3</td>
</tr>
<tr>
<td>Gau et al., 2005</td>
<td>2005</td>
<td>Taiwan</td>
<td>K-SADS-E</td>
<td>3 months</td>
<td>DSM-IV</td>
<td>14–16</td>
<td>1,051</td>
<td>22.7</td>
</tr>
<tr>
<td>Green et al., 2005</td>
<td>2005</td>
<td>United Kingdom</td>
<td>DAWBA</td>
<td>Current</td>
<td>DSM-IV</td>
<td>11–16</td>
<td>4,051</td>
<td>11.5</td>
</tr>
<tr>
<td>Lynch et al., 2006</td>
<td>2006</td>
<td>Ireland</td>
<td>K-SADS</td>
<td>Current</td>
<td>DSM-IV</td>
<td>12–15</td>
<td>723</td>
<td>15.6</td>
</tr>
<tr>
<td>Shear et al., 2006</td>
<td>2006</td>
<td>United States</td>
<td>CIDI</td>
<td>Childhood</td>
<td>DSM-IV</td>
<td>15–17</td>
<td>479</td>
<td>1</td>
</tr>
<tr>
<td>Ehringer et al., 2006</td>
<td>2006</td>
<td>United States</td>
<td>DISC</td>
<td>12 months</td>
<td>ICD-10</td>
<td>12–19</td>
<td>2,750</td>
<td>6.0</td>
</tr>
<tr>
<td>Benjet, 2009</td>
<td>2009</td>
<td>Mexico</td>
<td>CIDI</td>
<td>12 months</td>
<td>DSM-IV</td>
<td>12–17</td>
<td>3,005</td>
<td>39.4</td>
</tr>
</tbody>
</table>

DISC, Diagnostic Interview Schedule for Children (Shaffer et al., 1989) (Shaffer et al., 2000); SCAN, Schedules for Clinical Assessment in Neuropsychiatry (Wing et al., 1992); DIS, Diagnostic Interview schedule (Robins et al., 1981, 1982); K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Aged Children (Kaufman et al., 1997); DAWBA, Development and Well-Being Assessment (Goodman et al., 2000); CIDI, Composite International Diagnostic Interview (Andrews & Peters, 1998); CAPA, Child and Adolescent Psychiatric Assessment (Angold et al., 1995).

*Anxiety and depression only.
*Alcohol problems only.
*CID adapted for this study (Goodwin et al., 2004).
*Separation anxiety disorder only.
et al., 2010). However, full DSM–IV psychotic disorders are extremely rare until late adolescence.

**Eating disorders.** Rates of eating disorders vary widely depending on what is included. However, when only anorexia nervosa and bulimia nervosa are included there is greater consistence. The National Comorbidity Study Replication reported that no one in the sample (N = 2,980) met criteria for anorexia nervosa; the 12-month rate for bulimia was 0.3% (Hudson, Hiripi, Pope, & Kessler, 2007). However, rates for the youngest cohort (17–34 years of age) were double those for the older sample members. In a younger sample, Rowe, Pickles, Simonoff, Bulik, and Silberg (2002) found that only 2 of a state-wide sample of 2,790 adolescent twins met full criteria for bulimia, but 46% of girls and 15% of boys had one or more symptoms. In a German sample, Wittchen, Nelson, and Lachner, (1998) found that 0.3% of 3,021 young people (14–24 years) had a diagnosis of anorexia nervosa in the past 12 months, and 0.7% had a diagnosis of bulimia.

**Panic disorder with or without agoraphobia.** The 12-month prevalence was 1.2% in the German study, 0.6% in the Dunedin study through age 21, and 0.3% in the 13–16-year-olds in Great Smoky Mountains Study (GSMS). Wittchen et al. (Reed & Wittchen, 1998) found that about three times as many young people reported panic attacks as met criteria for panic disorder.

**Bipolar disorder.** Increased rates of early-onset bipolar disorder have been widely discussed in the clinical literature in recent years, but empirical epidemiologic data are hard to find. In 1995, Lewinsohn, Klein, and Seeley reported that 1% of youth aged 14–18 years had met criteria for bipolar disorder (mainly bipolar II and cyclothymia) during their lifetime. Four individuals out of 1,420 in GSMS and no one in the Dunedin sample of nearly 1,000 met criteria for bipolar disorder by 21 years of age. The 12-month prevalence in the German sample was 1.6%, but most cases were over 17 years. As there are no large-scale epidemiologic studies of adolescents before the 1990s that included bipolar disorder it is impossible to say whether prevalence has increased compared with earlier decades.

**Autism and pervasive developmental disorders.** Both autism and PDD are considered life spectrum conditions are undiagnosed by 9 years of age (Baron-Cohen et al., 2009). One study using population sampling is the National Health Interview Survey, an annual population survey conducted by the Centers for Disease Control (Boyle et al., 2011). Using parental reports that a child had autism or developmental delay, it has looked at both age and secular differences. Prevalence of autism at ages 10–17 between 1997 and 2008 was 0.4%, increasing more than fivefold (0.1–0.6%) across that period. In contrast, rates of developmental delay increased little (3.4–4.2%). The British National Study of 1999 reported pervasive developmental disorders in 0.2% of youth aged 11–15 years (Meltzer, Gatward, Goodman, & Ford, 1999).

These estimates come with a lot of caveats. The age of participants ranged from 12 through 19 years, and it is rare to find results presented separately by year of age. The time frame of the study interviews also varied, encompassing current (e.g., ‘Are you currently depressed?’) to 1 month, 3 months, 6 months, or a year. Most studies used DSM–IV (American Psychiatric Association, 1994) as their taxonomy, but a few used the earlier version (DSM–IIIR; American Psychiatric Association, 1987) or the ICD–10 (World Health Organization, 1992). Different taxonomies are likely to lead to different prevalence rates. The studies listed used 10 different assessment instruments, each of which has its own way of turning a diagnostic symptom into a question or set of questions, and different computer algorithms and/or clinical reviews for fusing symptoms, functional impairment, duration, severity, and age of onset into a diagnosis. Different studies included and excluded a different range of disorders. While most studies collected information from the study participants themselves, they varied in the extent to which they made use of other informants, such as parents and teachers, and the system that they used to combine information from different informants. There is wide variation in study size, from several thousand participants down to a hundred, as well as differences in the number of times that participants were assessed, and in the methods used to aggregate information across assessments. This said, the overall estimate that at any given time one adolescent in five has a psychiatric disorder is consistent with earlier reviews based on different samples of studies (e.g., Costello, 2009; Costello et al., 2005; Fombonne, 1998; Roberts, Attkisson, & Rosenblatt, 1998; Verhulst, 1995). Nothing in the literature suggests any dramatic secular changes in

overall rates of adolescent psychiatric disorder, although mean symptom scores for conduct problems at ages 15–16 increased between 1974 and 1999, and mean hyperactive and emotional problem scores between 1986 and 1999 (Collishaw, Mau-ghan, Goodman, & Pickles, 2004).

### Changes in prevalence from childhood to adolescence and adolescence to adulthood

#### Childhood to adolescence

Table 2 presents results for the transition from childhood to adolescence. For this review, we take the years between 10 and 15 as those during which the transition from childhood to adolescence occurs. The largest epidemiological study to cover both childhood and early adolescence is the British Child and Adolescent Mental Health Survey (BCAMHS), conducted in 1999 with over 10,000 children aged 5–15 years. Rates of DSM–IV psychiatric disorder in BCAMHS increased from 8.6% at 8–10 years to 9.6% at 11–12 years and 12.2% at 13–15 years.

This and other studies were unanimous in showing increased rates of depression as children move into adolescence, although in two studies the increase was ‘significant’ only for girls. They also show increasing drug abuse, panic disorder and agoraphobia, and decreasing ADHD and separation anxiety disorders (SAD). CD and ODD follow different courses in different studies. BCAMHS found a modest increase in adolescent prevalence, while there was little difference in the rates from Dunedin. In GSMS, rates of both disorders remained the same in girls, and fell slightly in boys. Across all disorders, there was a modest increase in prevalence between childhood and adolescence. Among the ‘rare disorders’ autism spectrum disorders and tic disorders tended to show fewer or less impairing symptoms, while panic and bipolar disorders, psychoses, and eating disorders all began to increase in prevalence across this transition. Apart from SAD the disorders that diminish in adolescence – tics, ADHD, autism spectrum disorders – tend to be more common in boys. One group of those that emerge or increase in adolescence – depression, anxiety, panic – are more common in girls, while psychosis and SUD are more common in boys.

#### Adolescence to adulthood

Table 3 shows changes observed as adolescents became adults (up to age 30). The transition from adolescence to adulthood was marked by an increase in overall rates of disorder. This was led by the surge in substance use disorders (abuse or dependence: SUD), as well as panic disorder, agoraphobia, and eating disorders. Disruptive behavior disorders and ADHD continued to fall, as did SAD, social phobia, specific phobias, and GAD (Generalized Anxiety Disorder). The story about depression is more difficult to interpret; Dunedin and the National Comorbidity Study (NCS) reported a modest increase in prevalence as adolescents moved into adulthood, while the GSMS showed a modest decrease (7.2–5.2%). For diagnoses available in more than one dataset, there was consistent evidence for increases in any disorder, attributable to drug abuse and dependence, including alcohol and nicotine. ADHD and tic disorders continued to fall.

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**Table 2**: Changes in prevalence of disorders from childhood to adolescence

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Direction of change</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any anxiety disorder</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Panic disorder</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>Decrease BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Separation anxiety disorder</td>
<td>Decrease GSMS</td>
<td></td>
</tr>
<tr>
<td>Social phobia</td>
<td>Same BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Specific phobia</td>
<td>Increase GSMS</td>
<td></td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>Increase (M) CCS</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Oppositional disruptive disorder</td>
<td>Decrease BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Attention-deficit hyperactivity disorder</td>
<td>Decrease BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Any substance use disorder</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
<tr>
<td>Any disorder</td>
<td>Increase BCAMHS</td>
<td></td>
</tr>
</tbody>
</table>

BCAMHS, British Child and Adolescent Mental Health Survey: 11–12 years versus 13–15 years (Ford et al., 2003), 13–16 years (Green et al., 2005); GSMS, Great Smoky Mountains Study: 9–12 years versus 13–16 years (Costello et al., 2003); Dunedin, Dunedin Multidisciplinary Health and Development Study (Arseneault et al., 2000; Moffitt et al., 2007; Newman et al., 1996); CCS, Caring for Children Study: 10–13 years versus 14–16 years (Cohen et al., 1993); Brazil, Isla de Maré (Fleitlich-Bilyk & Goodman, 2004). Differences that halve or double the previous rate are indicated in bold.
Homotypic and heterotypic continuity in adolescence

Homotypic prediction refers to a disorder predicting itself over time (e.g., earlier depression predicting later depression). This supports the idea that a single disease process expresses itself robustly across developmental contexts.

Heterotypic prediction refers to different disorders predicting one another over time (e.g., earlier ODD predicting later depression). Such patterns may suggest that the different disorders reflect a general disease process that has specific phenotypic expressions in different developmental contexts.

Childhood to adulthood

Homotypic prediction has been identified in most studies predicting from childhood to adolescence in rates of disorder (e.g., Bittner et al., 2007; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; and continuous measures (e.g., Burke, Loeber, Lahey, & Rathouz, 2005; Fergusson, Lynskey, & Horwood, 1996). In GSMS, homotypic continuity was strongest for SUD, with odds ratios (OR) over 20, adjusted for other comorbidities, and weakest (but still significant) for anxiety, with ORs around 2. The same study showed modest levels of heterotypic continuity in this age range: past depression predicted anxiety disorders (adjusted OR: 2.8) while past anxiety predicted depression (adjusted OR: 2.7). Earlier anxiety also predicted SUDs. Other studies confirm that homotypic continuity is stronger than heterotypic continuity in this age range (Cohen, Cohen, & Brook, 1993; Keenan, Feng, Hipwell, & Klostermann, 2009; Steinhausen, 2006; Sterba et al., 2010).

Few studies account for concurrent comorbidity among disorders in assessing continuity. When comorbidity is not taken into account, pairwise associations may simply represent indirect effects rather than direct associations (Angold, Costello, & Erkanli, 1999; Ford et al., 2003). For example, in GSMS prediction from depression to ADHD was found to disappear in the absence of comorbid anxious, CD, or ODD (Angold et al., 1999).

Adolescence to adulthood

More than three quarters of young adults with psychiatric disorders first had a diagnosis between the ages of 11 and 18 years (Kim-Cohen et al., 2003). There is widespread evidence of homotypic prediction (Ferdinand & Verhulst, 1995; Ferdinand, Verhulst, & Wznitzer, 1995; Goodwin, Fergusson, & Horwood, 2004; Haarasilta, Marttunen, Kaprio, & Aro, 2001; Hofstra, Van Der Ende, & Verhulst, 2002; Kim-Cohen et al., 2003; Lewinsohn, Rohde, Klein, & Seeley, 1999; Newman, Moffitt, Silva, Avshalom, & Magdol, 1996; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). Indeed, prior disorder status is typically the strongest predictor of having the same disorder later. Although typically less common than homotypic prediction, three patterns of heterotypic prediction have received consistent support. First, anxiety and depression tend to cross-predict from childhood/adolescence to adulthood; anxiety predicting depression: full support (Burke et al., 2005; Costello et al., 2003; Kim-Cohen et al., 2003; Moffitt et al., 2007; Pine, Cohen, Gurley, Brook, & Ma, 1998); support in one sex only (Bittner et al., 2007; Haarasilta et al., 2001; Hofstra et al., 2002; Orvaschel, Lewinsohn, & Seeley, 1995); depression predicting anxiety: full support (Burke et al., 2005; Costello et al., 2003; Kim-Cohen et al., 2003; Moffitt et al., 2007; Pine et al., 1998); support in one sex only (Hofstra et al., 2002; Orvaschel et al., 1995). Second, childhood/adolescent conduct/oppositional problems tend to precede adult anxiety and depression (Burke et al., 2005; Costello et al., 2003; Kim-Cohen et al., 2003; Moffitt et al., 2007; Pine et al., 1998). Table 3 Changes in prevalence of disorders from adolescence to adulthood

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Direction of change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any anxiety disorder</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Social phobia</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Specific phobia</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Any depressive disorder</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Major depression</td>
<td>Increase Dunedin</td>
</tr>
<tr>
<td>Depression NOS/Minor depression</td>
<td>Decrease NCS</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Oppositional disruptive disorder</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Attention-deficit hyperactivity disorder</td>
<td>Decrease (F, M) GSMS</td>
</tr>
<tr>
<td>Any substance use disorder</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Nicotine dependence</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Alcohol abuse/dependence</td>
<td>Increase (F, M) GSMS</td>
</tr>
<tr>
<td>Eating disorder</td>
<td>Increase EDPS</td>
</tr>
<tr>
<td>Any disorder</td>
<td>Increase EDPS</td>
</tr>
</tbody>
</table>

GSMS, Great Smoky Mountains Study: 13–16 years versus 19–21 years (Copeland et al., 2009); EDSP, Early Developmental Stages of Psychopathology: 14–17 years versus 18–20 years (Wittchen et al., 1998); Dunedin, Dunedin Study (Laufer et al., 2002; Richardson et al., 2003; Moffitt et al., 2007; Newman et al., 1996); NCS, National Comorbidity Study (Kessler & Walters, 1998); NOS, Not Otherwise Specified; Differences that halve or double the previous rate are indicated in bold.
Homotypic and heterotypic continuity at the symptom level

Recently there has been an interest in which symptoms stay stable and which ones diminish or increase with these changing prevalence rates. A couple of examples illustrate the key issues. In a study using GSMS to look at aggressive and nonaggressive symptoms of CD in boys (Rowe, Maughan, Worthman, Angold, & Costello, 2004), we found no difference in aggressive symptoms from 9 to 15 years of age, but increasing rates of nonaggressive symptoms (lying, stealing without confrontation, breaking in, property damage, fire setting, running away, truancy) between 12 and 15 years. Increasing testosterone levels were correlated with high nonaggressive symptoms. In another example, Biederman, Petty, Evans, Small, and Faraone (2010) used 10 years of data on 100 clinically treated boys with ADHD to show that whereas inattentive symptoms were more common than hyperactive symptoms in adolescence; by adulthood, both types were equally rare. Further studies at the symptom level are clearly needed.

Discussion

Adolescence, widely believed to be a turbulent developmental stage (Rutter, Graham, Chadwick, & Yule, 1976) between the relative calm of childhood and adulthood, might be expected to be accompanied by rates of psychiatric disorder that differ from either. A review of the available literature reveals a more nuanced picture. Some anxiety disorders (panic, agoraphobia), depression, and SUD begin to increase in adolescence and continue to increase into early adulthood, although SUD begins to decrease again markedly by the middle twenties (Dawson et al., 2005). Another group of anxiety disorders (SAD, specific phobias, social phobia), along with ADHD, begin to decrease in late childhood and continue to do so into adulthood. Trajectories for other disorders remain unclear. In the GSMS data, GAD shows an adolescence-limited increase in girls, but a general decrease in boys. Although the decline in rates of CD and ODD from adolescence to adulthood is consistent across studies, the pattern of change from childhood to adolescence is less clear. The persuasive idea of childhood-onset and adolescence-limited antisocial behavior (Moffitt, 1993), a construct reflecting conflicts with the law, is not wholly consistent with the data on CD, a syndrome that includes several symptoms that go beyond law-breaking and in any case become irrelevant in adulthood (e.g., truancy, running away; Loeber, Lahey, Winters, & Zera, 2000).

These patterns raise questions about the causal factors that might produce them. We need studies that test hypotheses about predictors of childhood, adolescent, and adult psychiatric disorders, preferably in the same (longitudinal) samples. For example, to what extent are changes in prevalence between childhood and adolescence affected by the biological and psychosocial changes of puberty? Adolescence begins with the biologically driven developmental transition of puberty, which has secondary effects on social, emotional, and sexual development (Hayward, 2003). Which of these myriad effects mediate the observed changes in rates of depression from childhood and adolescence? A full review of such mechanisms is beyond the scope of the current manuscript, not least because even simple effects have multiple determinants.

Taking a single example – depression – increases in adolescent females have been linked independently to early pubertal timing (Copeland et al., 2010), low birthweight (Costello, Worthman, Erkanli, & Angold, 2007), and increasing levels of sex steroids such as estrogen and testosterone (Angold, Costello, & Worthman, 1998). However, appeal to these mechanisms is oversimplified: in the case of pubertal timing, an environmentally influenced (Moffitt, Caspi, Belsky, & Silva, 1992) biological transition affects sex-specific risk for depression through effects on social context (De Bernardo, Newcomb, Toth, Richey, & Mendoza, 2002) mediated by cognitive processing (Weichold, Silbereisen, & Schmott-Rodermund, 2003). Carefully dissecting such mechanisms requires prospective, longitudinal studies that assess a range of potential biological, cognitive, and social mediators and moderators. The goal of this epidemiological review of adolescent transitions is to direct researchers to the changes and transitions relevant to such process-based study (see Shanahan, Copeland, Costello, & Angold, 2008, for a recent partial review). It is clear, however, that a concerted program of research on what predicts these rises and falls in prevalence over development could well bring to light robust and potentially causal pathways that could increase our understanding of the origins of mental illness.
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Key points
- Changes in the prevalence of psychiatric disorders between childhood and adolescence, and between adolescence and adulthood, provide opportunities for etiologic research as well as for targeting interventions.
- Recent epidemiological studies of the transition from childhood to adolescence show an increase in panic disorder, agoraphobia, depression, and substance use disorders, and a decrease in separation anxiety disorder and attention deficit hyperactivity disorder.
- Studies of the transition from adolescence to adulthood show further increases in panic disorder, agoraphobia, substance use disorders, and eating disorders, and decreases in several anxiety disorders and behavioral disorders.

References


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