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

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Abstract

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. Both longitudinal and cross-sectional papers published in the past 15 years were included.

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

Words for adolescence have been around since the 15th 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 and 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 hundred years sexual maturation has begun ever earlier, while legal independence varies by country or state and topic (e.g., drinking, marrying, voting)(Parent et al., 2003, Liu et al., 2000). At the same time, employers' demands for a more educated labour force have expanded the years spent in education, lengethening 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

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caused by different disorders at various developmental stages (Murray and Lopez, 1996). This "public health" aspect of research 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 (1) childhood depression; (2) persistence of childhood depression into adolescence; (3) adolescent depression; (4) persistence of adolescent depression into adulthood; and (5) adult-onset depression. Furthermore, there may be times, and adolescence is an example (Copeland et al., 2009a), 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 (Kleinbaum et al., 1982, Kapphahn et al., 2006, Zuvekas and Taliaferro, 2003, Costello et al., 1998). 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 et al., 2002)). Third, if a "case" is defined in distributional terms (as, for example, 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. Since these have been reviewed elsewhere(Costello et al., 2005, Costello et al., 2006b, Costello and Angold, 2009), and in any case mainly use different taxonomies (DSM-III or IIIR or ICD-9), the present review concentrates on work published in the past 15 years.

[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.

Methods

We reviewed the data available in published form, and also included unpublished data from own ongoing longitudinal study, about (1) the prevalence of psychiatric disorders in adolescence; (2) how these rates change as young people move into and out of adolescence; (3) 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 data sets 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 (for example, differences in drug use observed in a youth of the same age some of whom were interviewed before and some after the attacks of 9/11/2001 (Costello et al., 2004)).

In addition to longitudinal studies, cross-sectional studies that present their data separately for children, adolescents, and adults can also be used. Since 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 et al., 2006a). In this paper, 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 data sets 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 specifically for adolescence – which we define as spanning the 12-19 age-range. Studies that include adolescence but do not provide information specifically for a group in the age-range 12-19 were excluded. Across these studies, the average rate of any adolescent psychiatric disorder was 21.8%, with an interquartile range of 14.8% to 22.8%. Drug abuse or dependence was the most common diagnosis in this age group (mean 12.1%, interquartile range 3.3% to 18.3%), followed by anxiety disorders (mean 10.7%, interquartile range 5.5% to 14.9%) and depressive disorders

(6.1%, interquartile range 3.1% to 7.2%). On average, studies identified between 3% and 4% of adolescents as suffering from any one of the behavioral disorders (conduct disorder, oppositional defiant disorder, or ADHD).

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) had one or more "less common disorders" (PDD, psychotic disorders, tic disorders, eating disorders) (Ford et al., 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 intervales around the estimate tend to be very wide.

Psychosis—The British National Survey of Psychiatric Morbidity (Johns et al., 2004) found that 5.0% of a sample (N=8,580) age 17 to 74 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=2127) at age 12 (Polanczyk 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 bilumia 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 et al., 2007). However, rates for the youngest cohort (age 17-34 years) were double those for the older sample members. In a younger sample, Rowe at al. found that only 2 of a state-wide sample of 2,790 adolescent twins met full criteria for bulimia (Rowe et al., 2002), but 46% of girls and 15% of boys had one or more symptoms. In a German sample, Wittchen and colleagues (Wittchen et al., 1998) found that 0.3% of 3,021 young people (14-24) had a diagnosis of anorexia nervosa in the past 12 months, and 0.7% 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 GSMS. Wittchen et al. found that about 3 times as many young people reported panic attacks as met criteria for panic disorder (Reed and Wittchen, 1998).

Bipolar disorder—Increased rates of early-onset bipolar disorder have been widely discussed in the clinical literature in recent years, but empirial epidemiologic data are hard to find. In 1995 Lewinsohn et al. reported that 1% of youth aged 14-18 had met criteria for bipolar disorder (mainly bipolar II and cyclothymia) during their lifetime (Lewinsohn et al., 1995). 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 age 21. The 12-month prevalence in the German sample was 1.6%, but most cases were over 17. Since 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 sentences with early onset (American Psychiatric Association, 1994), so rates should be similar in childhood and adolescence. Although there are several studies of the

prevalence of both autism and PDD, they tend to use sampling methods based on referral for special education or other services, assuming that there are no unidentified cases, despite evidence that up to 40% of cases of autism-spectrum conditions are undiagnosed by age 9 (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 5-fold (0.1% to 0.6%) across that period. In contrast, rates of developmental delay rose little (3.4% to 4.2%). The British national study of 1999 reported pervasive developmental disorders in 0.2% of youth aged 11-15 (Meltzer et al., 1999).

Tic disorders—Most studies distinguish between transient vocal and motor tics and the more chronic and impairing tic disorders, including Gilles de la Tourette disorder. By adolescence rates of the latter are quite low, probably less than 1% (Peterson et al., 2001, Hirtz et al., 2007, Mustillo et al., 2003).

These estimates come with a lot of caveats. The age of participants ranged from 12 through 19, 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 one month, 3 months, six months, or a year. Most studies used the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV: (American Psychiatric Association, 1994)) as their taxonomy, but a few used the earlier version (DSM-III-R(American Psychiatric Association, 1987)) or the International Classification of Diseases, 10th Edition (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., (Roberts et al., 1998, Costello et al., 2005, Costello, 2009, Fombonne, 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 age 15-16 rose between 1974 and 1999, and mean hyperactive and emotional problem scores between 1986 and 1999 (Collishaw et al., 2004).

Changes in prevalence from childhood to adolescence and adolescence to adulthood

Childhood to adolescence—Table 2 present 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 to 15. Rates of

DSM-IV psychiatric disorder in BCAMHS rose from 8.6% at 8-10 to 9.6% at 11-12 and 12.2% AT 13-15.

This and other studies were unanimous in showing increased rates of depression as children moved into adolescence, although in two studies the increase was “significant” only for girls. They also showed increasing drug abuse, panic disorder and agoraphobia, and decreasing ADHD, and separation anxiety disorders (SAD). Conduct disorder and Oppositional Defiant Disorder (ODD) followed 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 separation anxiety disorder, social phobia, specific phobias, and GAD. 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 Great Smoky Mountains Study (GSMS) showed a modest decrease (7.2% to 5.2%). For diagnoses available in more than one data set, 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.

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 oppositional defiant disorder 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 adolescence—Homotypic prediction has been identified in most studies predicting from childhood to adolescence in rates of disorder (e.g., Bittner et al., 2007, Costello et al., 2003, and continuous measures {e.g., Burke et al., 2005, Fergusson et al., 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 (Keenan et al., 2009, Burke, 2008, Steinhausen, 2006, Sterba et al., 2010, Cohen et al., 1993a).

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 et al., 1999, Ford et al., 2003). For example, in GSMS prediction from depression to ADHD was found to diappear in the absence of comorbid anxiety, 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 (Kim-Cohen et al., 2003)). There is widespread evidence of homotypic prediction (Goodwin et al., 2004, Ferdinand and Verhulst, 1995, Ferdinand et al., 1995, Hofstra et al., 2002, Kim-Cohen et al., 2003, Lewinsohn et al., 1999, Newman et al., 1996, Haarasilta et al., 2001, Rohde et al., 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 et al., 1998); support in one sex only (Bittner et al., 2007, Haarasilta et al., 2001, Hofstra et al., 2002, Orvaschel et al., 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, Kim-Cohen et al., 2003, Hofstra et al., 2002, Zoccolillo, 1992, Capaldi, 1992, Loeber and Keenan, 1994), but not vice versa (Orvaschel et al., 1995, Costello et al., 2003, Kim-Cohen et al., 2003) (see (Hofstra et al., 2000) for an exception). Third, adolescent conduct disorder predicts adult SUD (Kim-Cohen et al., 2003)

Accounting for concurrent comorbidities among adolescent disorders in the Great Smoky Mountains data (Copeland et al., 2009b) homotypic patterns were common. However, the path from adolescent to young adult depression was entirely accounted for by ODD, GAD, and adolescent substance disorders. A single behavioral disorder, adolescent ODD, preceded most young adult anxiety and depressive disorders.

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 non-aggressive symptoms of conduct disorder in boys (Rowe et al., 2004) we found no difference in aggressive symptoms from ages 9 to 15, but increasing rates of non-aggressive 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 non-aggressive symptoms. In another example Beiderman et al. (Biederman et al., 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 et al., 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 20s (Dawson et al., 2005). Another group of anxiety disorders (separation anxiety disorder, 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 Conduct Disorder, 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 et al., 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 et al., 2007), and increasing levels of sex steroids such as estrogen and testosterone (Angold et al., 1998). However, appeal to these mechanism is oversimplified: In the case of pubertal timing, an environmentally-influenced (Moffitt et al., 1992) biological transition affects sex-specific risk for depression through effects on social context (De Bernardo et al., 2002) mediated by cognitive processing (Weichold et al., 2003). Carefully dissecting such mechanisms requires prospective, longitudinal studies that assess for 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 et al., 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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Table 1

Prevalence of any psychiatric disorder in adolescents, from studies published since 1997

Study	Year of Publication	Nationality	Instrument	Time frame	Taxonomy	Age range	Number of subjects	Prevalence of any psychiatric disorder
(Verhulst et al., 1997)	1997	Holland	DISC	6 months	DSM-IIIR	13-18	780	35.5
(Beals et al., 1997)	1997	USA (American Indian)	DISC	6 months	DSM-IIIR	14--16	109	21.1
(Canals et al., 1997)	1997	Spain	SCAN	12 months	DSM-IIIR	18	290	20.6
(Krueger et al., 1998)	1998	New Zealand	DIS	12 months	DSM-IIIR	18	930	44
(Lewinsohn et al., 1998)	1998	USA	K-SADS	current	DSM-IIIR	14-18	1709	9.6
(Lewinsohn et al., 1998)	1998	USA	K-SADS	current	DSM-IIIR	15-19	1507	7.8
(Meltzer et al., 1999)	1999	UK	DAWBA	current	DSM-IV	11--15	4609	11.2
(Rueter et al., 1999)	1999	USA	UM-CIDI	5 years	DSM-IIIR	15--19	303	~*
(Johnson et al., 2000)	2000	USA	DISC	6 months	DSM-IIIR	12--16	688	~**
(Fergusson and Horwood, 2001)	2001	New Zealand	CIDI*	12 months	DSM-IIIR	15	1000	22
(Fergusson and Horwood, 2001)	2001	New Zealand	CIDI*	12 months	DSM-IIIR	18	1000	42
(Romano et al., 2001)	2001	Canada	DISC	6 months	DSM_IIIR	14--17	2000	20.1
[Costello, 2003 #15807}	2003	USA	CAPA	3 months	DSM-IV	13-16	1420	22.8
(Gau et al., 2005)	2005	Taiwan	K-SADS-E	3months	DSM-IV	13--15	1070	20.3
(Gau et al., 2005)	2005	Taiwan	K-SADS-E	3months	DSM-IV	14-16	1051	22.7
(Gau et al., 2005)	2005	Taiwan	K-SADS-E	3months	DSM-IV	13	1051	22.7
(Gau et al., 2005)	2005	Taiwan	K-SADS-E	3months	DSM-IV	14	1035	14.8
(Green et al., 2005)	2005	UK	DAWBA	current	DSM-IV	11--16	4051	11.5
(Lynch et al., 2006)	2006	Ireland	K-SADS	current	DSM-IV	12--15	723	15.6
(Shear et al., 2006)	2006	US	CIDI	childhood	DSM-IV	15-17	479	~***
(Ehringer et al., 2006)	2007	USA	DISC	12 months	ICD-10	12--19	2750	6.0
Benjet, 2009 #23894	2009	Mexico	CIDI	12 months	DSM-IV	12--17	3005	39.4
Kessler	2011	USA	CIDI	1month	DSM-IV	13--17	10,123	25.5

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, Robins et al., 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 and Peters, 1998)

CIDI* CIDI adapted for this study (Goodwin et al., 2004)

CAPA Child and Adolescen Psychiatric Assessment (Angold et al., 1995)

~* Anxiety and depression only

~** Alcohol problems only

~*** Separation anxiety disorder only

Table 2

Changes in prevalence of disorders from childhood to adolescence*

Diagnosis	Direction of change	Study
Any anxiety disorder	Increase Increase (F, M)	BCAMHS[1] GSMS[2]
Panic disorder	**Increase** **Increase (F, M)**	BCAMHS GSMS
Agoraphobia	**Increase** **Increase (F, M)**	BCAMHS GSMS
Generalized anxiety disorder	**Decrease** Increase (F) Decrease (M) Increase	BCAMHS GSMS GSMS Dunedin[3]
Separation anxiety disorder	Decrease **Decrease (F, M)** **Decrease**	BCAMHS GSMS CCS[4]
Social phobia	Same **Increase (F)** Decrease (M)	BCAMHS GSMS GSMS
Specific phobia	Decrease **Decrease (F)** Same (M)	BCAMHS GSMS GSMS
Any depressive disorder	**Increase (F,** M) Increase **Increase** **Increase (F,** M)	GSMS Brazil[5] BCAMHS CCS
Conduct Disorder	**Increase** Same (F) Decrease (M) Decrease (F) Increase (M) Same	BCAMHS GSMS GSMS CCS CCS Dunedin[6]
Oppositional disruptive disorder	Decrease Same (F) Decrease (M) Increase (F) Same (M) Same	BCAMHS GSMS GSMS CCS CCS Dunedin
ADHD	**Decrease (F, M)** Decrease Decrease	GSMS BCAMHS CCS
Any substance use disorder	**Increase** **Increase (F, M)**	CCS GSMS
Any disorder	Increase Increase (F) Decrease (M) Same	BCAMHS GSMS GSMS Dunedin

[1] British Child and Adolescent Mental Health Survey (BCAMHS) 11-12 vs. 13-15 (Ford et al., 2003), 13-16 (Green et al., 2005)

[2] Great Smoky Mountains Study (GSMS) 9-12 vs. 13-16 (Costello et al., 2003)

[3] Dunedin: Dunedin Multidisciplinary Health and Development Study (Arseneault et al., 2000, Moffitt et al., 2007) (Newman et al., 1996)

[4] CCS: Caring for Children Study 10-13 vs. 14-16 (Cohen et al., 1993b)

[5] Brazil: Isla de Maré (Fleitlich-Bilyk and Goodman, 2004)

Table 3

Changes in prevalence of disorders from adolescence to adulthood

Diagnosis	Direction of change	
Any anxiety disorder	Increase (F, **M**) Increase Decrease	GSMS[1] EDSP[2] Dunedin [3]
Panic disorder	**Increase (F, M)**	GSMS
Agoraphobia	**Increase (F, M)**	GSMS
Generalized anxiety disorder	Decrease (F, M)	GSMS
Separation anxiety disorder	**Decrease (F, M)**	GSMS
Social phobia	**Decrease(F, M)**	GSMS
Specific phobia	**Decrease (**F, **M)**	GSMS
Any depressive disorder	Decrease (F, M) Increase **Increase**	GSMS Dunedin EDSP
Major depression	Increase (F) Increase (M) Decrease (F)	Dunedin NCS[4] NCS
Depression NOS/Minor depression	Decrease	NCS
Conduct Disorder	**Decrease (F, M)**	GSMS
Oppositional disruptive disorder	**Decrease (F.M)**	GSMS
ADHD	**Decrease (F, M)**	GSMS
Any substance use disorder	**Increase (**F, **M)** **Increase**	GSMS EDPS
Any hard drugs	**Increase (F, M)**	GSMS
Nicotine dependence	**Increase** Increase (F, M)	EDPS GSMS
Alcohol abuse/dependence	**Increase** **Increase (F, M)**	EDPS GSMS
Eating disorder	**Increase**	EDPS
Any disorder	**Increase (F,** M) **Increase**	GSMS Dunedin

[1]GSMS: Great Smoky Mountains Study (GSMS) (Copeland et al., 2009b) 13-16 vs 19-21

[2]EDSP: Early Developmental Stages of Psychopathology (Wittchen et al., 1998) 14-17 vs 18-20

[3]Dunedin: Dunedin Study (Richardson et al., 2003, Jaffee et al., 2002); (Moffitt et al., 2007); (Newman et al., 1996)

[4]NCS: National Comorbidity Study (Kessler and Walters, 1998)