

Is there an epidemic of child or adolescent depression?

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Background: Both the professional and the general media have recently published concerns about an ‘epidemic’ of child and adolescent depression. Reasons for this concern include (1) increases in antidepressant prescriptions, (2) retrospective recall by successive birth cohorts of adults, (3) rising adolescent suicide rates until 1990, and (4) evidence of an increase in emotional problems across three cohorts of British adolescents. **Methods:** Epidemiologic studies of children born between 1965 and 1996 were reviewed and a meta-analysis conducted of all studies that used structured diagnostic interviews to make formal diagnoses of depression on representative population samples of participants up to age 18. The effect of year of birth on prevalence was estimated, controlling for age, sex, sample size, taxonomy (e.g., DSM vs. ICD), measurement instrument, and time-frame of the interview (current, 3 months, 6 months, 12 months). **Results:** Twenty-six studies were identified, generating close to 60,000 observations on children born between 1965 and 1996 who had received at least one structured psychiatric interview capable of making a formal diagnosis of depression. Rates of depression showed no effect of year of birth. There was little effect of taxonomy, measurement instrument, or time-frame of interview. The overall prevalence estimates were: under 13, 2.8% (standard error (SE) .5%); 13–18 5.6% (SE .3%); 13–18 girls: 5.9% (SE .3%); 13–18 boys: 4.6% (SE .3%). **Conclusions:** When concurrent assessment rather than retrospective recall is used, there is no evidence for an increased prevalence of child or adolescent depression over the past 30 years. Public perception of an ‘epidemic’ may arise from heightened awareness of a disorder that was long under-diagnosed by clinicians. **Keywords:** Depression, child, adolescent, prevalence, meta-analysis, epidemic. **Abbreviations:** DISC: Diagnostic Interview Schedule for Children; CIDI: Composite International Diagnostic Interview; SDI: Short Depression Interview; K-SADS: Schedule for Affective Disorders and Schizophrenia, child version; CAS: Child and Adolescent Schedule; CAPA: Child and Adolescent Psychiatric Assessment; IOW: Isle of Wight interview; DAWBA: Development and Well-Being Assessment.

Concern about an ‘epidemic’ of child and adolescent depression has found expression not only in academic journals (Fombonne, 1998; Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn, Rohde, Seeley, & Fischer, 1993), but also in the popular press (Clausen, 1999; Healy, 2003; McGrath, 1992), in journals and television programs for the general public, and on many websites. Examples (from a list available on request from the first author) include <http://www.pbs.org/thesilentepidemic> (‘A silent epidemic is ravaging the nation and killing our kids’), http://www.clinical-depression.co.uk/Depression_Information/teen.htm (‘the fastest rate of increase in depression is among young people’), and <http://news.bbc.co.uk/1/hi/health/3532572.stm> (‘Teen depression on the increase. The numbers of young people suffering from depression in the last 10 years has risen worryingly, an expert says’).

This concern has been fueled by four sets of empirical evidence. First, there is evidence for increased numbers of prescriptions for antidepressants for children and adolescents (Zito et al., 2003). Second, there was an increase in teen suicides in the

United States between 1950 and 1990 (<http://www.cdc.gov/nchs/data/hus/04trend.pdf#topic>), although since 1990 the rate has fallen by 30%, to levels last seen in the mid-1970s (Gould, Greenberg, Velting, & Shaffer, 2003). Third, a study of three British birth cohorts assessed at age 15–16 in 1974, 1986, and 1999 found a trend toward increasing levels of ‘emotional problems’ (anxiety and depression) over time (Collishaw, Maughan, Goodman, & Pickles, 2004). Fourth, many epidemiologic studies of adults have reported increasing lifetime rates of depression in later-born cohorts (Burke, Burke, Rae, & Regier, 1991; Cross-National Collaborative Group, 1992; Hagnell, Lanke, Rorsman, & Öjesjö, 1982; Joyce, Oakley-Browne, Wells, Bushnell, & Hornblow, 1990; Kessler et al., 2003; Kessler et al., 1994; Klerman et al., 1985; Lewinsohn et al., 1993; Weissman, Leaf, Holzer, Myers, & Tischler, 1984; Wickramaratne, Weissman, Leaf, & Holford, 1989). Others, however, find no such cohort effect (Murphy, Laird, Monson, Sobol, & Leighton, 2000), or argue that any such effect may be explained more parsimoniously as a methodological artifact (Eaton et al., 1989; Giuffra & Risch, 1994; Patten, 2003; Paykel, 2000; Simon et al., 1995; Wittchen,

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Knauper, & Kessler, 1994). The problem with drawing conclusions about childhood depression from cross-sectional studies like most of those cited above is that, in order to remember early episodes of depression, older participants have to look back over a much longer period of time than do younger ones. Several papers have demonstrated that even a modest degree of 'recall failure' could give an impression of increasing rates in later cohorts (Giuffra & Risch, 1994; Patten, 2003).

One way to get around this problem is to look at concurrent rather than retrospective psychiatric information, provided by people of the same age from successive birth cohorts. These reports will not be as prone to 'recall failure' because the symptoms are current or occurred in the recent past.

Beginning in the 1970s, data on rates of childhood depression have been collected on representative population samples of children and adolescents, using structured interviews and standard algorithms to define cases. In this report we use prevalence estimates from such studies, with subjects born between 1965 and 1996, to test the belief that today's children and adolescents are more likely to suffer from depression than were earlier generations at the same age.

Methods

Following recommendations for meta-analysis of observational studies (Stroup et al., 2000), we first identified epidemiologic studies of child and adolescent psychiatric disorders using Medline and PsycINFO, and through email messages to relevant professional societies. We also made personal contact with investigators, on occasion asking them to perform extra analyses for this paper (e.g., generating separate depression estimates for childhood and adolescence), which they graciously did.

The selection criteria for inclusion in the report were:

1. Participants aged up to 18.
2. Formal psychiatric diagnoses of depressive disorders using an established taxonomy and a structured or semi-structured psychiatric interview of adequate reliability. Studies using only symptom questionnaires were excluded.
3. Information available about participants' date of birth, sex, and age at interview.
4. Information available about the time-frame of the interview used.

During the period since the first cohorts were born in the 1960s there have been several taxonomies defining a range of depressive diagnoses (American Psychiatric Association, 1980, 1987, 1994; U.S. Department of Health and Human Services, 1980; World Health Organization, 1992). These included dysthymia, minor depression or depression not otherwise specified (NOS), major depressive episode

(MDE), major depressive disorder (MDD), and in some studies bipolar disorder, which has a very low prevalence in young samples (Costello et al., 1996; Kessler et al., 2001; Lewinsohn, Klein, & Seeley, 1995) and was rarely reported separately. Studies varied in the depressive diagnoses they included, and whether they provided data on each one separately or as a group. Many reported prevalence only for 'any depressive disorder'. Many studies only provide figures for 'any depression'. We make the assumption that this refers to any depressive diagnosis that can be made using the taxonomy (ICD-9, ICD-10, DSM-III, DSM-III-R, DSM-IV) adopted for that particular study. In order to retain the largest possible number of studies for meta-analysis we included all those reporting any depression, MDD, or MDE. Separate analyses for MDD or MDE only (available from the first author) showed very similar results to those presented here.

Several factors could influence estimates of the prevalence of child and adolescent depression and needed to be included as covariates in the meta-analysis. The first is the age-range and sex of the participants. The prevalence of depression is low in both boys and girls before puberty, but after that it increases, especially in girls (Angold, Worthman, & Costello, 2003). We ran separate analyses for under-13s and those aged 13 and over, and in the latter age group separate analyses for boys and girls. Where the data could not be split at 13, we allocated a study according to the age of the majority of subjects.

Second, the time-frame of the psychiatric interview used in a study can vary from instantaneous (current) to one, three, six, or twelve months. If earlier studies used a shorter time-frame than later studies, this could artificially inflate later estimates, or vice versa. The models included a covariate to estimate the effect of time-frame. Third, prevalence estimates may be affected by the taxonomy used to define depression (ICD-9, ICD-10, DSM-III or DSM-IV), or the methods used to collect the necessary information about symptoms, and to define a diagnosis. For example, changes in the number of symptoms used to mark the cut-point for a diagnosis could produce the appearance of secular rate changes. Diagnostic system was included as a covariate in all models, as was number of informants (parent, child, teacher).

Methods for meta-analysis

We used fixed-effects linear regression models to combine information from available published results and to estimate the effects of birth cohort and time-frame. Where the subjects in a study were born in different years (e.g., were aged 13 to 15 at recruitment) the median was used as the measure of date of birth. We transformed the prevalence of depression to logit scales which were then used as dependent variables. The standard errors for the logits were estimated by the

delta method using a mean imputation technique¹ because not all studies provided standard errors.

The number of studies providing information for each estimate varied depending on the age-range for which each study was informative (see Table 1). Where studies gave prevalence rates separately by age these are listed separately in Table 1.

For each analysis, we first tested for heterogeneity of the prevalence rates using the chi-square test. None of the tests provided evidence of heterogeneity so we used fixed-effects rather than the more computationally intensive random-effects models. Each model included an intercept and slope parameters measuring the effects of birth cohort, time-frame, taxonomy and instrument. Computations were performed in WinBUGS using a Gibbs sampling approach based on non-informative (flat) priors.

Results

Table 1 lists the studies that contributed to the meta-analysis, and the prevalence estimates that they provide. For each data set, Table 1 shows the taxonomy and diagnostic interview used, the time-frame, birth dates of the participants and their age at the time of interview, the informants (e.g., parent, child, teacher), the number of subjects interviewed, the number of study waves, the setting, the race/ethnicity of the participants, the definition of depression used, and the prevalence estimates. Where possible, data are provided separately by race/ethnicity. In total, 59,703 observations were available for analysis. There were 15 data sets that contributed information on children under 13, and 20 for those aged 13–18. Sixteen data sets contained separate information for adolescent boys, and 18 for adolescent girls.

The earliest cohorts included subjects born in 1965 and interviewed at age 9, while the most recent had subjects born in 1996. Thus, data on children born up to 30 years apart were available to test the reality of the 'epidemic'. Studies came from around the world, but they were heavily weighted toward white samples. For this reason, no attempt was made to test for race/ethnic differences in cohort effects.

Results of the meta-analysis are shown in Table 2. The table shows the parameter estimate, standard error, and 95% credible interval (the Bayesian equivalent of the 95% confidence interval) for under-13s, 13–18-year-olds, and the latter separately by sex.

¹For a sufficiently large sample size, the logit transform of the prevalence estimator \hat{p}_i for the i -th study is normally distributed with mean $\mu_i = \log(p_i/[1 - p_i])$ and standard error $se_i = se(\hat{p}_i)/[p_i(1 - p_i)]$, where $se(\hat{p}_i)$ is the standard error of \hat{p}_i . Since not all of the studies published $se(\hat{p}_i)$, we used the mean of the available ones. Additional model fitting using an imputation model for $se(\hat{p}_i)$ (assuming a gamma distribution with unknown scale and shape parameters, and non-informative prior distributions) did not change the findings. Computational details are available from the second author.

There was no effect of birth cohort in any of the models fitted, for any of the age or age-by-sex groups. Interviews with a longer time-frame generated significantly higher prevalence estimates than those with a shorter time-frame in analyses involving under-13s, and boys and girls 13–18. The effect was in the same direction but not significant in the analysis that included the additional data sets on 13–18-year-olds for whom the data were not available separately by sex. Planned contrasts showed that this effect was accounted for by higher prevalence rates generated by a 6-month than by 3-month time-frame; there were no significant increases in prevalence estimates when interviews used a 12-month rather than a 6-month time-frame. There were too few data sets to permit a comparison of current versus 3-month time-frames.

There was an effect of taxonomy for younger children, with DSM-IV generating higher rates than earlier systems, but not for adolescents. There was no effect of the interview used except (marginally) for adolescent boys, where the studies reporting the highest estimates used an early version of the Diagnostic Interview Schedule for Children (DISC).

As expected, prevalence estimates based on model predictions averaged over studies were higher for adolescents (5.7%, standard error (SE) .3%) than for children (2.8%, SE .5%), and for adolescent girls (5.9%, SE .3%) than for adolescent boys (4.6%, SE .3%).

Discussion

This meta-analysis of studies involving close to 60,000 observations of children born over the past 30 years provided no evidence of increasing rates of depression in later-born cohorts. The model showed, as expected, higher rates of depression in adolescents than children, and in adolescent girls than adolescent boys, across the whole time-period of the studies. It also showed, as expected, that interviews using a longer time-frame produced higher prevalence estimates than those using a shorter time-frame. The fact that these findings were significant argues that lack of power was unlikely to explain the absence of a significant cohort effect.

The analysis of time-frame effects shown here suggests that recall tends to deteriorate after six months; the estimates were not significantly higher for 12-month than for 6-month interviews. This calls into question the likelihood that lifetime recall over decades will be accurate enough to provide good evidence for cohort differences.

The meta-analysis also provided an opportunity to estimate the prevalence of depression in children and adolescents based on a large number of observations, using many different taxonomies, interviews and study time-frames. The estimates for adolescents were somewhat lower than those generated by

Table 1 Studies available for meta-analysis

Key author and reference	Year of birth	Age at interview	N. of observations	Waves providing data	Taxonomy	Interview	Time frame (months)	Informants: P = parent, C = child, T = teacher	Prevalence of depression (percent)		
									<13	13-18	13-18
									Girls	Boys	Boys
Rutter (Graham & Rutter, 1973)	54-55	14-15	483	2		IOW*	1	P, C, T	3.2		
Cohen (Velez, Johnson, & Cohen, 1989)	65-74	10-20	776	1	DSM-III-R	DISC-1	12	P, C, T	2.5	7.6	1.6
Cohen (Velez et al., 1989)	65-74	10-20	776	2	DSM-III-R	DISC-1	12	P	3.1		
Kessler(Kessler & Walters, 1998)	66-75	15-24	1769	1	DSM-III-R	CIDI	1	C	7	12.4	1.5
Kessler (Kessler & Walters, 1998)	66-75	15-24	1769	1	DSM-III-R	CIDI	12	C	13	21.5	4.4
Fleming (Fleming, Offord, & Boyle, 1989)	66-79	6-16	2852	1	DSM-III-R	SDI	6	P, C, T	.6	1.8	1.2
Bird (Bird et al., 1988)	68-80	4-16	386	1	DSM-III-R	DISC-1	6	P, C			
Lewinsohn (Lewinsohn et al., 1993)	69-74	14-18	1710	1	DSM-III-R	K-SADS	1	C	2.9	3.8	2
Lewinsohn (Lewinsohn, Rohde, & Seeley, 1998)	69-74	14-18	1710	2	DSM-III-R	K-SADS	1	C	3.2	3.7	2.6
Anderson (Anderson, Williams, McGee, & Silva, 1987)	71-72	13	792	1	DSM-III-R	DISC-C, P and T	12	P, C, T	1.8	2.1	2.1
Hankin (Hankin et al., 1998)	71-72	15	792	1	DSM-III-R	questionnaires DISC-C, P and T	12	P, C, T	2.8	4.4	1.2
Kashani (Kashani, Orvaschel, Rosenberg, & Reid, 1989)	71	8	70	1	DSM-III-R	CAS	1	P, C	1.5		
Kashani (Kashani et al., 1989)	76	12	70	1	DSM-III-R	CAS	1	P, C	1.5		
Kashani (Kashani et al., 1989)	80	17	70	1	DSM-III-R	CAS	1	P, C		5.7	
Costello (Costello et al., 1988b)	73-77	7-11	300	1	DSM-III	DISC-1	12	P, C	.8		
Costello (Costello, Angold, & Keeler, 1999)	73-77	12-17	300	2	DSM-III-R	DISC-2.3	6	P, C	5.4		
Angold (Angold et al., 2002)	73-81	9-17	336	1	DSM-IV	CAPA	3	P, C	3.4	6	6.4
Angold (Angold et al., 2002)	73-81	9-17	542	1	DSM-IV	CAPA	3	P, C	.7	2.1	.8
Shaffer (Shaffer et al., 1996)	74-82	9-17	1285	1	DSM-III-R	DISC-2.3	6	P, C			
Simonoff (Simonoff et al., 1997)	74-83	8-16	2762	1	DSM-III-R	CAPA	3	P, C	.4		2.1
Verhulst (Verhulst, van der Ende, Ferdinand, & Kasius, 1997)	75-80	13-18	780	1	DSM-III-R	DISC 2.3	6	P, C		3.6	
Cairney (Cairney, 1998)	75-82	12-19	1847	1	DSM-III-R	CIDI	12	C	2.6		
Fergusson (Fergusson, Horwood, & Lynskey, 1993)	77	15	986	1	DSM-III-R	DISC-1	12	P, C	6.3	9.2	3.3
Fergusson (Fergusson & Horwood, 2001)	77	18	1011	1	DSM-III-R	DISC	12	P, C	18.2	26.5	9.7
Steinhausen (Steinhausen & Winkler Metzke, 2003)	78-82	15-19	203	1	DSM-III-R	DISC	12	P, C			
Kilpatrick (Kilpatrick et al., 2003)	78-83	12-17	4023	1	DSM-III-R	DISC-2.3	6	P, C	.6	5.3	1.1
Steinhausen(Steinhausen & Winkler Metzke, 2003)	78-87	7-16	1964	1	DSM-III-R	Phone interview DISC-2.3	6	C		13.9	7.4
Olsson (Olsson & von Knorring, 1999)	79-81	16-17	231	1	DSM-III-R	DICA	6	C	.3	2.4	.0
Doi (Doi, Roberts, Takeuchi, & Suzuki, 2001)	79-82	12-15	558	1	DSM-IV	DISC	12	C	1.4	2.2	.6
Doi (Doi et al., 2001)	79-82	12-15	665	1	DSM-IV	DISC	1	C	4.3	4.5	4.0
Doi (Doi et al., 2001)	79-82	12-15	429	1	DSM-IV	DISC	1	C	6.1	6.5	5.7
Doi (Doi et al., 2001)	79-82	12-15	494	1	DSM-IV	DISC	1	C	9.0	11.4	6.3
Doi (Doi et al., 2001)	79-82	12-15	494	1	DSM-IV	DISC	1	C	1.3	.9	1.8
Oldehinkel (Oldehinkel, Wittchen, & Schuster, 1999)	80-83	14-16, 16-19	1395	2	DSM-IV	CIDI	12	C	8.0	10.2	5.8
Costello (Costello et al., 1996)	80-84	9-16	4984	4	DSM-IV	CAPA	3	P, C	1.9	3.1	4.2
Costello (Costello et al., 1996)	80-84	9-16	1691	4	DSM-IV	CAPA	3	P, C	1.1	3.0	4.2
Almqvist (Almqvist, Kumpulainen et al., 1999)	81	8-9	278	1	DSM-III-R	DISC, IOW	9	P	3.2		
Almqvist (Almqvist, Kumpulainen et al., 1999)	81	8-9	278	1	DSM-III-R	DISC, IOW	9	C	5.9		
Almqvist (Almqvist, Puura et al., 1999)	81	8-9	255	1	DSM-III-R	IOW	3	P (Boys)	7.8		
Almqvist (Almqvist, Puura et al., 1999)	81	8-9	180	1	DSM-III-R	IOW	3	P (Girls)	4.7		

Table 1 (Continued)

Key author and reference	Year of birth	Age at interview	N. of observations	Waves providing data	Taxonomy	Interview	Time frame (months)	Informants:			Prevalence of depression (percent)				
								P = parent,	C = child,	T = teacher	<13	13-18	13-18	13-18	Boys
								P	P, C	P, C	Girls	Boys	Girls	Boys	
Sawyer(Sawyer et al., 2001)	81-92	6-17	3597	1	DSM-IV	DISC-IV	12	P	2.3	4.0	4.7	3.4			
Canino (Canino et al., 2004) (and personal communication)	82-96	4-17	1886	1	DSM-IV	DISC-IV	12	P, C (11-17)	2.1	5.8	9.7	2.0			
Ford (Ford, Goodman, & Meltzer, 2003)	84-94	5-15	10438	1	DSM-IV	DAWBA	1	P, C(11-15)	.3	2.5					
Fleitlich-Bilyk(Fleitlich-Bilyk & Goodman, 2004)	86-89	11-14	625	1	DSM-IV	DAWBA	1	P, C	.2	1.9					
Fleitlich-Bilyk (Fleitlich-Bilyk & Goodman, 2004)	90-93	7-10	625	1	DSM-IV	DAWBA	1	P	.2						
Van der Stoep (personal communication)	90-93	11-12	508	1	DSM-IV	DISC-IV	12	P, C	3.0						

*Abbreviations: IOW = Isle of Wight interview. DISC = Diagnostic Interview Schedule for Children. CIDI = Composite International Diagnostic Interview. SDI = Short Depression Interview; K-SADS = Schedule for Affective Disorders and Schizophrenia, child version. CAS = Child and Adolescent Schedule. CAPA = Child and Adolescent Psychiatric Assessment. DAWBA = Development and Well-Being Assessment. DSM = Diagnostic and Statistical Manual.

Table 2 Percent prevalence estimates based on model predictions, for different interview time-frames

	Parameter estimate	Standard error	95% Credible interval
Under 13 (15 datasets)			
Birth cohort	-.009	.013	-.036, .018
Time frame (overall)	.142	.022	.016, .192
Taxonomy	.74	.26	.220, 1.260
Instrument	-.12	.07	-.260, .020
Intercept	-4.79	1.13	-7.01, -2.59
13-18 (20 datasets)			
Birth cohort	-.005	.012	-.028, .017
Time frame (overall)	.029	.021	-.11, .069
Taxonomy	-.01	.36	-.139, .119
Instrument	-.09	.04	-.250, .070
Intercept	-3.07	.89	-4.81, -1.32
13-18 Girls (18 datasets)			
Birth cohort	.003	.011	-.018, .025
Time frame (overall)	.049	.013	.024, .076
Taxonomy	.65	.36	-.07, 1.37
Instrument	-.014	.04	-.094, .066
Intercept	-3.49	.85	-5.16, -1.83
13-18 Boys (16 datasets)			
Birth cohort	.026	.018	-.01, .06
Time frame	.032	.017	.001, .070
Taxonomy	.55	.48	-.41, 1.51
Instrument	-.10	.04	-.18, -.02
Intercept	-5.30	-1.41	-8.08, -2.54

recent surveys and reviews of adult depression (Kessler et al., 2003; Wittchen et al., 1994), and the sex difference for adolescents, while significant, was less extreme than some reviews based on smaller samples have indicated (Hayward, 2003).

Belief in the ‘epidemic of depression’ is so strong, as shown in the articles and websites listed in the introduction, that it needs some explanation. Here we discuss four of the most plausible.

First, because pubertal factors (such as increases in sex steroid hormones) have been associated with the adolescent increase in rates of depression in girls (Angold, Costello, & Worthman, 1999), and because the age of puberty fell during the twentieth century, this could be seen as putting a greater proportion of the pediatric population at risk. However, most of the fall in age at puberty appears to have occurred prior to the 1960s. Current estimates suggest that between the 1960s and the early 1990s age at menarche dropped by only 2½ months; hardly enough to have had a substantial influence on rates of depression overall (Parent et al., 2003).

A second compelling argument for an epidemic of depression is the increase in suicide noted in several countries between 1970 and 1990 (followed by declining rates since then) (<http://www.child-trends.databank.org/figures/70-Figure-1.gif>). There is no question that many youth who kill themselves are also depressed; a recent review estimates that between 49% and 64% have a depressive disorder (Gould et al., 2003). However, depression is more likely in suicidal girls than boys (Brent et al., 1993; Shaffer et al., 1996), whereas nearly all the increase in youth suicides between the 1960s and the 1990s

occurred in males, particularly African American males (Gould et al., 2003; Shaffer, Gould, & Hicks, 1994). Given that drug abuse followed a somewhat similar curve to that of suicide, increasing until the 1990s and then falling (<http://www.drugabuse.gov/newsroom/05/NR12-19.html>), this might explain trends in suicide better than does depression.

Third, several studies of *clinical* samples have reported secular trends in the prevalence of depression (Kovacs, Goldston, & Gatsonis, 1993; Ryan et al., 1992). There are several reasons to be cautious about conclusions based on clinical samples. First, children who make their way to clinical settings are likely to be different in many ways from the untreated cases that make up the majority of children with psychiatric disorders (Berkson, 1946; Costello et al., 1988a). They are, for example, more likely to be children of depressed parents, who may have a heightened sensitivity to depression in their children (Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). The development of effective pharmacotherapies and psychotherapies for childhood depression is likely to have increased the number of parents seeking treatment for their depressed children. Insurance companies have also been more willing to pay for treatments of proven effectiveness (Burns, Hoagwood, & Mrazek, 1999; Compton, Burns, Egger, & Robertson, 2002), and these have become more available in recent years. In order to demonstrate that an increase in treated cases was caused by an increase in the general population, it would be necessary to account for these competing explanations.

Fourth, rates of childhood depression might have appeared to increase because more recent taxonomies (DSM-IV, ICD-10) could have changed the criteria so as to include more children. This could, of course, co-occur with a real increase in depression. Fortunately there was considerable overlap among studies in the use of the various taxonomies; for example, some continued to use DSM-III-R while others moved to DSM-IV. Thus, it was possible to look for changes in prevalence controlling for taxonomy in the meta-analysis.

Limitations

Since childhood depression was scarcely recognized before the 1970s (Angold et al., 2003), this review goes back as far as is possible in charting changes in prevalence using current rather than retrospective data. Thus, the possibility remains that there was an increase in the prevalence of childhood depression before 1970, when clinical recognition and epidemiological measurement of childhood depression began (Angold & Costello, 2001).

It would be more compelling if we were able to chart incidence rather than prevalence, but

published incidence studies of child or adolescent depression are too few for conclusions to be drawn (Essau, Conradt, & Petermann, 2002; Kashani & Simonds, 1979; P.M. Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Meltzer, Gatward, Corbin, Goodman, & Ford, 2003). In generating the prevalence estimates presented here, we had to impute standard errors from the few studies that published them. Meta-analysis would be much more accurate if every study published standard errors or confidence intervals around its estimates.

We were unable to treat age as a continuous measure, because the data were not available. The best that could be done was to run separate analyses for children and adolescents, and in the case of adolescents, where power was adequate, to look separately at boys and girls. Also, there were too few studies to look separately at possible effects of using different classifications of depression (MDD, MDE, etc.).

Studies using symptom questionnaires were excluded from these analyses because they seldom include questions about severity, duration, onset, etc. that form part of the diagnostic algorithms for depressive disorders. Also, making a diagnosis from questionnaire data usually involves identifying a pre-defined percentage of the sample as 'cases', so population prevalence rates are pre-defined rather than empirically estimated. A meta-analysis of over 300 studies using one popular depression questionnaire found no cohort effect (Twenge & Nolen-Hoeksema, 2002). The comparison of the three British birth cohorts (Collishaw et al., 2004) found a significant increase in conduct symptoms, but only a non-significant trend in emotional symptoms.

Conclusions

There is good reason to be concerned about the rate of depression in children and adolescents. However, 30 years of research suggests that, for as far back as we have reliable assessments, a similar proportion of children have been depressed, albeit largely unrecognized by clinicians (Costello, 1986; Costello & Edelbrock, 1985; Costello et al., 1988c; Dulcan et al., 1990; Horwitz, Leaf, Leventhal, Forsyth, & Speechley, 1992). If more depressed children are being identified, or are receiving antidepressant medication, this is more likely to be the result of increased sensitivity to a long-standing problem than of an 'epidemic'.

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