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Puberty and depression: the roles of age, pubertal status and pubertal timing

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ABSTRACT

Background. Previous work has indicated that the 2:1 female:male sex ratio in unipolar depressive disorders does not emerge until some time between ages 10 and 15.

Methods. Data from four annual waves of data collection from the Great Smoky Mountains Study (GSMS) involving children aged nine to 16 were employed.

Results. Pubertal status better predicted the emergence of the expected sex ratio than did age. Only after the transition to mid-puberty (Tanner Stage III and above) were girls more likely than boys to be depressed. The timing of this transition had no effect on depression rates. Before Tanner Stage III, boys had higher rates of depression than girls, and the prevalence of depression appeared to fall in boys at an earlier pubertal stage than that at which it began to rise in girls. In addition, recent transition to Tanner Stage III or higher had a transient effect in reducing the prevalence of depression in boys.

Conclusions. The period of emergence of increased risk for depression in adolescent girls appears to be a relatively sharply demarcated developmental transition occurring in mid-puberty. Previously reported effects of the timing of puberty (which have tended to be transient) appeared less important in increase of risk for depression than pubertal status.

INTRODUCTION

Studies of adults suggest that the gender difference in rates of depression emerges during adolescence

Studies of adults from several counties have emphatically documented that women have 1·5 to 3 times more current and lifetime unipolar depression than men (Weissman & Klerman, 1977; Bebbington *et al.* 1981; Canino *et al.* 1987; Lee *et al.* 1987; Bland *et al.* 1988*a*, *b*; Cheng, 1989, Hwu *et al.* 1989; Wells *et al.* 1989; Wittchen *et al.* 1992; Kessler *et al.* 1993, 1994; Weissman *et al.* 1993, 1996; Blazer *et al.* 1994). Using retrospective data, some of these have pointed to adolescence as the time when this gender difference first appears. For example, the Epidemiological Catchment Area (ECA) studies (Burke *et al.* 1990) suggested that unipolar depression onset rates were equal in males and females until age 15–19, while the National Comorbidity Survey (NCS) (Kessler *et al.* 1993), provided evidence for the emergence of an onset differential by age 10–14.

Uncertainty about the timing of effects on depression

The child and adolescent epidemiological literature generally agrees that rates of depression are similar in prepubertal boys and girls, and that rates of depressive disorders begin to rise in girls at some time between childhood and age 15 (Rutter *et al.* 1976; Anderson *et al.* 1987; Cohen & Brooks, 1987; Kashani *et al.* 1987, 1989; Bird *et al.* 1988; McGee & Williams, 1988; Guyer *et al.* 1989; Velez *et al.* 1989; Fleming & Offord 1990; McGee *et al.* 1990; Nolen-Hoeksema *et al.* 1991; Angold & Rutter, 1992; Reinherz *et al.* 1993; Lewinsohn *et al.* 1995). But this age range covers a lot of developmental ground. Two

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longitudinal community studies suggested that female excess did not emerge until after age 13; the Dunedin Longitudinal Study (Anderson *et al.* 1987; McGee *et al.* 1992); and the New York Study (Cohen & Brooks, 1987; Velez *et al.* 1989; Cohen *et al.* 1993*a*,*b*). On the other hand, Angold & Rutter's (1992) study of a large clinical population found that the preponderance of referred girls with depressive disorders began to emerge at around the age of 10.

The timing of the change in sex ratios has important implications for theories about the relationship between depression and puberty. For example, if such a change actually begins by age 10, it may be that adrenarche (which is in progress by middle childhood) is more relevant than puberty (Brooks-Gunn & Warren, 1992; Eccles & Buchanan, 1992). It is also possible that these 'biological' changes are irrelevant, and that some other correlates of age or pubertal development are responsible for the apparent effects of puberty. For example, one large clinical study (Angold & Rutter, 1992), found that age continued to predict the presence of depression over and above effects of pubertal status. Only one of the studies considered so far involved any measures of pubertal status *per se*, rather than using age as a marker of development status. On the other hand, none of the work using one or more measures of pubertal status which we review next, has employed diagnostic measures of depression.

Morphological stage and pubertal timing

Previous studies of menarche or morphological development (secondary sex characteristics – usually measured by Tanner stages (Tanner, 1962)) have suggested that levels of these pubertal status markers *per se* may not be significantly related to mood or other disturbances (Olweus *et al.* 1988; Susman *et al.* 1987*b*; Brooks-Gunn & Warren, 1989; Paikoff *et al.* 1991; Angold & Rutter, 1992; Ge *et al.* 1996), but that their timing may be. Early puberty has been associated with problem behaviours in girls, but with good adjustment in boys (see Stattin & Magnusson (1990) for a review). Stattin & Magnusson (1990) argued from their longitudinal study that the negative effects of early development in girls were generated by the impact of early maturity on girls' social lives. However, these effects had largely disappeared by the time the girls were in mid-adolescence, whereas the female excess of depressive disorders continues throughout adulthood (Weissman & Klerman, 1977). In the Carolina Longitudinal Study, Cairns & Cairns (1994) did not find any affect of early puberty on behavioural deviance.

Hormonal studies of puberty and psychopathology

Studies with direct hormonal measures lend further weight to the idea that puberty is 'bad' for girls, but 'good' for boys as far as depression is concerned, but these studies have mainly (though not exclusively) focused on hormone concentrations (the analytical equivalent of morphological or menarcheal status), rather than on the timing of changes in hormonal concentrations. The NIMH study of puberty and psychopathology (Nottelmann *et al.* 1987*a*,*b*; Susman *et al.* 1987*a*,*b*) found negative associations between testosterone:oestradiol ratio, sex hormone binding globulin, androstenedione concentration and negative emotional tone in boys. These workers also reported an association of early maturation (measured by oestradiol and testosterone:oestradiol ratio) with reduced negative emotional tone in boys, but more negative emotional tone in girls. Adrenal androgen concentrations correlated with negative emotional tone in boys, but not girls, while the opposite was the case for FSH (Susman *et al.* 1985). Overall, hormone concentrations were more strongly associated with affective behaviour in boys than in girls (Nottelmann *et al.* 1990). Brooks-Gunn & Warren (1989) found that negative affect increased in 10–14 year-old girls during rapid oestrogen rise, but saw no effect of the age at which increased oestradiol secretion occurred. This work has been extended to include a 1-year follow-up of 72 girls from the original sample (Paikoff *et al.* 1991). The authors found a significant linear effect of oestradiol level at time 1 on self-reported symptoms of depression 1 year later, using the Center for Epidemiologic Studies Depression Scale (CES-D (Radloff, 1977)), but no such effect on the Depressed-Withdrawal Scale of the Youth Self-Report (Achenbach & Edelbrock, 1991) or on the mothers' reports of their daughters' depressive affect (Brooks-Gunn *et al.* 1994).

Problems with the literature to date

These lines of work, taken together, implicate the morphological and physiological developments of puberty in the increasing depression of adolescent girls. On the other hand, it is far from clear which of the many aspects of adrenarcheal and pubertal development are really important. In particular, studies based on morphological status (Tanner stages) or timing of menarche have tended to identify the timing of puberty as being central, while hormonal studies have found more effects in relation to pubertal status. There are several reasons (stated below) for this state of affairs (see (Angold & Worthman, 1993) for a more detailed discussion of these and further points).

1 Few general population studies have explicitly considered the multiple dimensions of puberty and none of the diagnostic general population studies has included adequate measures of pubertal status.

2 The general population studies with good measures of morphological development have not used detailed or diagnostic measures of depression status.

3 The endocrine studies have lacked samples that are representative of the general population, and have been characterized by non-diagnostic (questionnaire) measures of depression and the use of many statistical tests (mostly non-significant) in relatively small samples.

4 Few studies have included an age range beginning before puberty and extending to a point where most subjects could be expected to have begun puberty.

5 Few studies have taken a longitudinal approach, or provide repeated measures of the same subjects across puberty.

6 Retrospective information from adults, the main source of onset information until recently, does not provide the level of precision as to timing, or the information on pubertal status, needed to address the key questions here. It is also subject to the danger of differential recall by currently depressed and non-depressed adults.

The workers involved in previous studies have recognized these problems, and the need for larger scale epidemiological studies of the impact of pubertal development on depression. We present such a study here, addressing the following questions: (1) At what age does the female preponderance of depressions begin?; (2) Is pubertal status more strongly associated with the emergence of the female preponderance than age?; (3) Does pubertal timing have a greater effect than pubertal stage?; (4) Does the amount of pubertal change over time have more effect on depression rates than the level of puberty reached by a certain age?

METHOD

The data came from the Great Smoky Mountains Study (GSMS) of children and adolescents. A detailed account of the study design and instrumentation used can be found in an earlier paper (Costello *et al.* 1996). We present a summary here.

Sampling frame

A representative sample of 4500 9, 11, and 13 year-olds, recruited through the Student Information Management System (SIMS) of the public school systems of 11 counties in western North Carolina, was selected using a household equal probability design. As close as possible to the child's birthday, a screening questionnaire was administered to a parent (usually the mother), by telephone or in person. This consisted of 55 questions from the Child Behavior Checklist about the child's behaviour ('externalizing') problems, together with some basic demographic and service use questions. All children scoring above a predetermined cut-off score of 20 (design to include about 25% of the population) on the behavioural questions, plus a 1-in-10 random sample of those scoring below the cut-off, were recruited for the longitudinal study. Eighty per cent of eligible families agreed to participate in the interviews in at least one wave (1073 of 1346).

Shortly after being screened, eligible children and one of their parents were interviewed. They were re-interviewed 1, 2 and 3 years later. Interviews were conducted between 1992 and 1996. Between 80% and 94% of the sample participated in each wave; 3733 observations on 1073 children are included in the analyses.

Informed consent was obtained from both children and parents. Because sexual development is a sensitive topic, we showed the Tanner stage pictorial assessment instrument (see below) to parents before giving it to the

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children, and specifically asked permission to use it. At each wave, between 6% and 13% of parents refused to have the scale administered to their children. Surprisingly, the refusal rate was twice as high for boys as it was for girls (11·5% *v.* 5·9%; $P = 3 \times 10^{-9}$). Thus, girls were significantly over-represented for analyses involving Tanner staging. However, refusal to complete the Tanner stage assessment was not significantly related to age, pubertal status, or depression diagnosis, so it seems unlikely that this source of missing data was a source of bias in the results. Table 1 shows the numbers of boys and girls interviewed at each wave of data collection and the numbers for whom Tanner stage data were available. The total number of observations of pubertal status over four waves was 3392.

The number of males was always greater than the number of females because the behavioural items in the screen used to select the interviewed sample selected more boys than girls, as expected (see e.g. Achenbach *et al.* 1987). This imbalance was corrected using a weighting system in all the analyses to generate unbiased population estimates. Table 2 shows the age distribution (rounded to the nearest birthday) of the observations by gender. At ages 11 to 14, the numbers are larger because individuals from two age cohorts contributed observations; for instance, 11-year-olds came from wave 1 of the 11-year-old's cohort and wave 3 of the 9-year-old's cohort.

Table 1. *Sample sizes by wave in the Great Smoky Mountains Study (GSMS)*

Wave	Gender	Total *N*	*N* with data on Tanner Staging
1	Female	443	425
	Male	572	527
2	Female	428	417
	Male	541	481
3	Female	412	381
	Male	513	448
4	Female	367	329
	Male	453	384

Table 2. *Age and gender distribution of the observations over four waves*

Age	Girls	Boys
9	153	191
10	155	197
11	290	385
12	275	359
13	269	349
14	256	331
15	142	153
16	111	117
Total	1651	2082

Measures

Psychiatric symptoms and disorders

Children and parents were interviewed using the Child and Adolescent Psychiatric Assessment (CAPA) (Angold *et al.* 1995), which generates Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association 1994) diagnoses and a range of symptom scale scores, measures of functional impairment and family burden. The interview also includes measures of maternal depression, family psychiatric history, the child's physical health and development and mental health service use (for details, see (Costello *et al.* 1996)).

Computer algorithms that combined symptom information from parent and child were used to determine whether a symptom was present. When symptoms were found to be present during the interview, their dates of onset were also collected, thus allowing for determination of whether they met the symptom overlap and duration criteria for the various DSM-IV depression diagnoses. If either parent or child reported a symptom as present in the past 3 months, it was counted toward the relevant CAPA/DSM-IV scale score or diagnosis. This 3 month 'primary period' was selected rather than, say, a 1 year or lifetime period, because shorter recall periods are associated with more accurate recall (see e.g. Angold *et al.* 1996*a*). We considered three depression diagnoses; DSM-IV major depressive episode, dysthymia and depression-not otherwise specified (NOS). The last of these diagnostic categories was made up of individuals who met the DSM-IV experimental criteria for minor depressive disorder (American Psychiatric Association 1994, p. 719).

Pubertal morphology status

Ratings of pubertal morphology status were based on the standard Tanner staging system (Tanner, 1962). Originally developed for clinical use, it involved a physical examination, which is

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impractical in non-clinical settings. Self-ratings performed with the aid of schematic drawings of secondary sexual characteristics (breasts and pubic hair in girls; genitalia and pubic hair in boys) have yielded good correlations with physical examination based on Tanner stages (Morris & Udry, 1980). Each child was provided with sex-appropriate schematic drawings and requested to rate her- or himself on each dimension. Unless otherwise noted, both self-ratings were averaged to yield a single individual score (ranging from I-prepubertal, to V-adult level of development). For some analyses, children were defined as 'immature' (Tanner Stages I or II) or 'mature' (Tanner stages III or higher). Girls were also asked about their menstrual histories, and if post-menarcheal, questions about the date of onset of their menstrual periods.

Analytical strategy

The principal analytical technique was mixed effects hierarchical linear modelling (HLM) (Diggle *et al*. 1994) with the logistic link function (since the outcome variable was the categorical variable of depression diagnosis). A random effect was introduced to account for correlations between the scores of each individual across waves. In addition, sandwich variance corrections (Diggle *et al*. 1994; Pickles *et al*. 1994) were applied to adjust for the effects induced by the sampling stratification based on the use of a screen. The SAS macro GLIMMIX was used to compute the estimates.

RESULTS

1 Age and gender differences in depressive disorders

Fig. 1 shows the 3-month prevalence of depressive disorders by age. The age-specific rate curves were quite different for boys and girls. At or above age 13 girls had consistently higher rates of depression than boys, but this was not the case at earlier ages. A second notable point is that the boys showed a fall in depression rates after the age of 9, while the girls showed an increase after the age of 12. However, in neither case did we observe a smooth change in depression rates with age. Fitting a mixed effects logistic model to these data resulted in a significant effect only of age (OR = 1·07, P = 0·02), reflecting the small overall increase in depression prevalence across this age range.

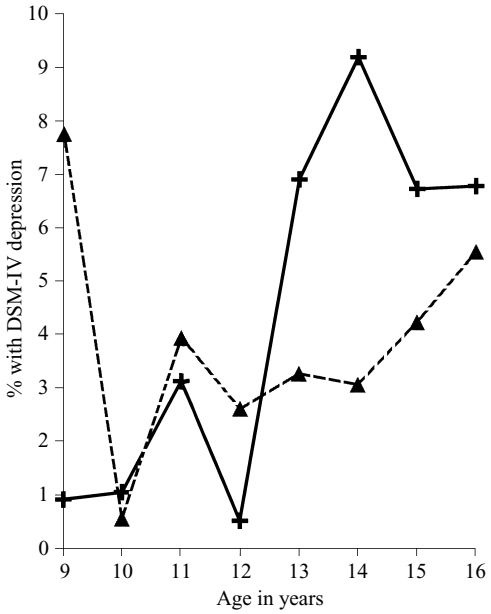

FIG. 1. Age and 3-month prevalence of depression in girls (+——+) and boys (▲---▲).

2 Morphological development and gender differences in depression

Age and morphological development are highly correlated (Pearson $r = 0{\cdot}75$ in this sample). However, if morphological development is really an 'active ingredient' in generating age- and gender-related changes in rates of depression, the morphological development by gender interaction should be a better predictor of depressive disorders than the age by gender interaction. As shown in Fig. 2, this is exactly what we found. Using Tanner stage, rather than age, 'cleaned up' the developmental pattern quite considerably, with girls having consistently higher rates of depressive disorders after Tanner stage III and boys having higher rates before that. The expected significant interaction between developmental level and gender was seen (gender, OR = 7·2, $P = 0{\cdot}02$; morphological development, OR = 1·3, $P = 0{\cdot}002$; gender by morphological development, OR = 0·72 (male

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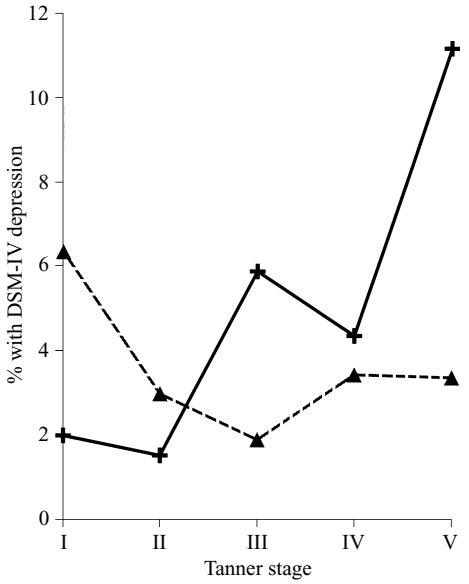

FIG. 2. Pubertal stage and 3-month prevalence of depression in girls (+——+) and boys (▲---▲).

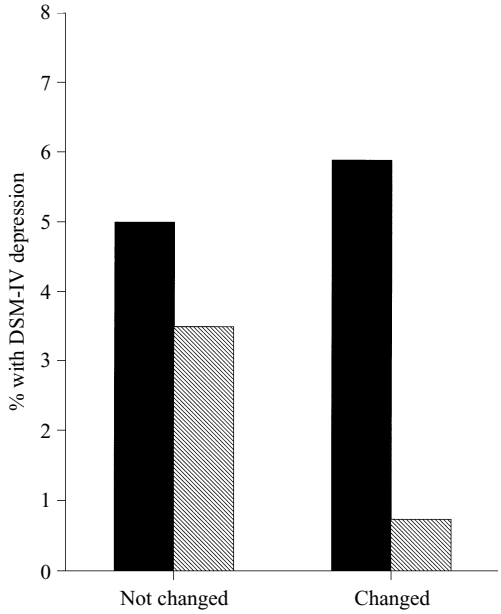

FIG. 3. Change in pubertal status and depression in girls (■) and boys (▨).

coded 1, female coded 0), $P = 0{\cdot}008$). Immature girls were less likely to be depressed than immature boys (OR = 0·46, $P = 0{\cdot}02$), but more mature girls were more likely to be depressed than more mature boys (OR = 2·5, $P = 0{\cdot}04$). More mature girls had a substantially higher rate of depression than immature girls (OR = 3·4, $P = 0{\cdot}005$). In boys, the only notable Tanner stage effect lay in the difference between the rate of depression in those at Tanner stage I and the rest. Dividing the boys in this way resulted in an OR of 0·43 ($P = 0{\cdot}03$). These effects were consistent for each of the three depression diagnoses (major depression, dysthymia, and depression-NOS), and so these and other analyses consider these three as a single group (which approach also ensures sufficient power for the subsequent analyses).

3 Effects of the process of change in pubertal status

The idea that the process of change in pubertal status itself might be important in generating differences in depression rates (Brooks-Gunn & Warren, 1985) implies that among the more mature children, girls who had only recently reached this level of maturity should have higher levels of depression than those who had been at that level of maturity for some time. We tested this hypothesis by dividing observations on the more mature participants ($N = 1614$) into those involving individuals who at the previous observation had been classified as being immature ($N = 384$) *versus* those who had been classified as being more mature ($N = 1230$). In the former group a change in maturity level had occurred in the past year, while in the latter it had not. We then looked at the rates of depression in boys and girls in these two groups (Fig. 3). There was a significant interaction between change in pubertal status and gender ($P = 0{\cdot}009$), but the effect driving the interaction was the low rate of depression in boys who changed from the immature to the more mature group. The effect of change was not significant in girls (OR = 1·25, $P = 0{\cdot}6$), but was highly significant in boys (OR = 0·24, $P = 0{\cdot}0009$).

We examined rate of change in pubertal status over the previous year, defined as number of Tanner stages traversed between waves. In neither boys nor girls was the rate of change found to have an effect on depression rate in the more mature children.

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4 Pubertal status or pubertal timing?

As mentioned in the introduction, several studies have suggested that pubertal timing may be important in generating changes in levels of psychopathology. A true timing effect could have generated the results seen in our data so far, because those with earlier puberty contributed more observations at later stages – in other words, timing of puberty and level of puberty were partially confounded. However, we can unconfound these variables: among more mature children (Tanner stage III and above), the timing hypothesis predicts that the excess of depression in girls compared with boys will only be seen in those who reached Tanner stage III relatively early. There should be no effect of gender in those who reached Tanner stage III at a normative age. Thus, we would expect to find a significant interaction between timing of puberty and gender in more mature children. On the other hand the 'level' hypothesis, which argues that girl's increased risk is associated with maturity *per se*, regardless of the age at which that maturity was reached, indicates that only a main effect of gender should be observed in more mature children.

To investigate these possibilities we divided the children into those who reached Tanner stage III before the age of 12, and those who did not reach Tanner stage III until later (data from 13-year olds at wave 1, who were already more mature, were excluded because we did not know when they reached this level of maturity). The results failed to provide any support for the timing hypothesis. The interaction between early onset and gender was non-significant, and the trend was in the wrong direction (with the early onset group of girls having a lower rate of depression than the normative puberty group), but as we saw above, there was a main effect of gender.

Since one of the major markers of pubertal stage in a number of previous studies of pubertal timing-psychopathology links has been date of menarche, we dichotomized girls into those with menarche before age 12 (the definition of early puberty used by Stattin & Magnusson (1990)), and those with later menarche. Looking at the impact of menarcheal timing on the rate of depression in observations conducted at Tanner stage III and above, we found no evidence for an effect of menarcheal timing (OR = 0·91, P = 0·8). Again, even this minimal trend was in the opposite direction from that predicted by the timing hypothesis.

DISCUSSION

Previous work established that the female predominance observed in numerous adult studies of depression is absent in childhood, but present by age 15–19. However, it has not been determined just where in the adolescent period this change occurs, or which of the many components of the adolescent transition are most strongly related to it. Most previous studies have relied either on adult recall of dates of onset in lifetime diagnostic interviews; or depression scale scores rather than diagnoses; or age or menarche as the only developmental markers. Our purpose was to examine these questions in a relatively large prospective study, with diagnostic assessments and specific measures of pubertal status.

The timing of the emergence of the female preponderance in depression

In keeping with the two other general population studies that provide some kind of age breakdown in early adolescence, the Dunedin Study (Anderson *et al.* 1987; McGee *et al.* 1992) and the New York Study (Cohen & Brooks, 1987; Velez *et al.* 1989; Cohen *et al.* 1993*a*, *b*), we found that the female preponderance of depression did not emerge until age 13. Adult retrospective studies have pointed to some time between 10 and 19 (Burke *et al.* 1990; Kessler *et al.* 1993). If the difference were already present at age 10 (transitions at age 10–11 were also suggested by Angold & Rutter's work on a large clinical sample (Angold & Rutter, 1992) and Weissman *et al.*'s (1987) family study of children at high and low risk of depression), then it would be unlikely that puberty could be invoked as the active factor in the generation of these differences, since at age 10 most girls are still pre-pubertal. By age 13, the great majority of girls are past menarche and into puberty. Timing of the change in depression rates around age 13 makes it unlikely that the increases in adrenal androgens that constitute adrenarche can be seen as having any major responsibility, since those changes occur in later childhood.

Some aspect of puberty itself is important in the appearance of the female preponderance in depression

An important question is whether the biological changes of puberty themselves are implicated in the increasing rates of depression in girls, or whether these might better be explained by other age-related factors. In relation to this issue, we first observed that Tanner stage fitted the depression rate data better than age. The expected interaction between age and sex in predicting depression was not significant, but that involving Tanner stage was, suggesting that Tanner stage was closer to being an 'active ingredient' in the transition to the adult sex ratio than age or some other age-related change.

It also appeared that this transition was a mid-pubertal event (occurring in Tanner stage III), since the rate of depression in girls in the earliest pubertal stage (i.e., Tanner stage II) was no different from that in prepuberty (Tanner stage I). This finding is in accord with retrospective data from the National Comorbidity Survey, which placed the increase in rates of onset of depression in the age range 10–14 (Kessler *et al*. 1993), whereas the ECA studies (also retrospective) had suggested that the female excess of onsets did not begin until ages 15–19 (Burke *et al*. 1990). Similarly, it agrees with findings from the Dunedin and New York child and adolescent epidemiological studies (Anderson *et al*. 1987; Cohen & Brooks, 1987; Velez *et al*. 1989; McGee *et al*. 1992; Cohen *et al*. 1993*a*, *b*). However, it extends these findings in demonstrating that: (*a*) by this stage, statistically significant differences are present rather than just suggestive differences; (*b*) the effect is quite sharply localized in its developmental location at the transition to mid-puberty, regardless of its timing; and (*c*) that the sex ratio reverses, with boys being more likely than girls to be depressed before mid-puberty. The most important component of this reversal was a tripling of the prevalence of depression in girls at mid-puberty.

Pubertal status was more important than age at puberty

Although some writers have emphasized the importance of pubertal 'timing' in the generation of psychopathology, it must be remembered that: (*a*) such timing effects have not, by any means, been consistently observed; (*b*) they have often been based on very limited measures of depression; and (*c*) any observed differences have not been demonstrated to be stable beyond mid-adolescence. This last point is of particular relevance here, because the female preponderance of depression persists through at least middle age. Based on either age at reaching Tanner stage III or menarcheal age in girls, we found no evidence for a timing effect, and such trends as there were, were in the wrong direction.

Pubertal status and change in pubertal status

Another possible explanation for the apparent effects of pubertal status discussed so far was that they might result from a transient effect of changing physical development (Brooks-Gunn & Warren, 1985). The key difference between these interpretations is that the status hypothesis predicts that physical maturity in girls is associated with increased levels of depression whether or not it was achieved recently. The change hypothesis suggests that the psychological effects of hormonal and physical changes are responsible for increased rates of depression, and that once a steady state has been reached, depression should again become less common in girls – in other words we should observe a transient peak in the female prevalence of depression around puberty. First, we looked at whether having made the transition to Tanner stage III or above only during the preceding year, as opposed to earlier, increased the rate of depression in girls, and found that it did not. Secondly, we found that the degree of change in Tanner stage over the preceding year had no significant effect on the rate of depression in those who were at Tanner stage III and above. Thus, in girls, the identified effect on depression rates was reaching Tanner stage III and above – it made no difference when they got there 'how fast' they got there, or once there, 'how fast' they subsequently matured.

In boys, however, a different, and unexpected, picture emerged. The transition to stage III of puberty was associated with a significant reduction in the rate of depression. Those who had reached this stage only during the preceding year had a much lower rate (OR = 0·24) than those who had been at stage III or above for a year or more. Thus, the transition to mid-

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puberty appeared transiently to protect boys from depression. On the other hand, whether they had recently made this transition or not, boys had lower depression rates after Tanner stage III than girls; and the *rate* of change in pubertal status had no effect on the prevalence of depression.

What really happens to rates of depression in boys across puberty?

Depression was about twice as common in boys before puberty (Tanner stage I) than in boys who were experiencing the physical changes of puberty (around 6% *v.* around 3%). This difference was significant ($P = 0{\cdot}03$), but this comparison was only made *post hoc* because the data indicated that this was where any significant comparison would lie. However, in combination with the substantial effect of the transition to stage III just discussed, the possibility that puberty is associated with at least a transient reduction in the prevalence of depression in boys should not be dismissed. No such effects were observed in the Dunedin or New York epidemiological studies, but those studies included few male cases of depression (fewer than 10 at each wave), and did not include measures of pubertal status. Work from the Pittsburgh Youth Study, using a depression questionnaire (Angold *et al.* 1996*b*) identified a substantial fall in depression scale scores in boys between first and sixth grades, with a levelling off after sixth grade. However, a linear reduction in depression scores with age from first to sixth grade is not consistent with an explanation in terms of puberty, though it would fit with the fall in rates of depression observed after the age of 9 in our data. These data are also consistent with work linking higher levels of adrenal and gonadal androgens (androstenedione and testosterone) with lower levels of 'negative affectivity' (Nottelmann *et al.* 1990).

How are these changes generated?

The first point to make here is that the occurrence of changes in girls in mid-puberty rules out an explanation in terms of adrenarche, though the presence of adrenarcheal effects remains open in boys. Secondly, the absence of a timing effect contradicts Stattin & Magnusson's interpretation of effects on psychopathology in terms of early maturing girls become associated with older males with conduct problems. However, since these effects had largely dissipated by age 15 in their study (Stattin & Magnusson, 1990), and were not reproduced in Cairns & Cairns' (1994) work, they were never a good candidate for explaining the long-enduring phenomenon of women having a higher rate of depression than men.

While this study does not indicate what mechanism is actually at work in generating the change in relative rates of depression in males and females (which could be anything from cognitive changes, the social impact of puberty, or changes in levels of life stress to direct effects of hormones on the brain (see Nolen-Hoeksema & Girgus, 1994; Leadbeater *et al.* (1995) for extensive discussions of many possibilities), it does suggest that further work needs to be focused on identifying enduring changes in risk or protective factors that occur in relation to the transition to mid-puberty, rather than at other times. It also suggests that age is a poor marker for identifying such changes, and that we need more studies that include direct measures of the components of puberty itself.

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