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Which Childhood and Adolescent Psychiatric Disorders predict which Young Adult Disorders?

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Abstract

Context—Most adults with a psychiatric disorder first met diagnostic criteria during childhood and/or adolescence, yet specific homotypic and heterotypic patterns of prediction have not been firmly established.

Objective—To establish which childhood and adolescent psychiatric disorders predict particular young adult disorders when accounting for comorbidities, disaggregating similar disorders, and examining childhood and adolescent predictors separately.

Design/Setting/Patients—Eleven waves of data from the prospective population-based Great Smoky Mountains Study (*N* = 1,420) were used, covering children in the community ages 9−16, 19, and 21 years old.

Outcome—Common psychiatric disorders were assessed in childhood (ages 9 to 12) and adolescence (ages 13 to 16) with the Child and Adolescent Psychiatric Assessment, and in young adulthood (ages 19 and 21) with the Young Adult Psychiatric Assessment.

Results—Adolescent depression significantly predicted young adult depression in the bivariate analysis, but this effect was entirely accounted for by comorbidity of adolescent depression with adolescent oppositional defiant disorder, anxiety and substance disorders in adjusted analyses. Generalized anxiety and depression cross-predicted each other, and oppositional defiant disorder (but not conduct disorder) predicted later anxiety disorders and depression. Evidence of homotypic prediction was supported for substance use disorders, antisocial personality disorder (from conduct disorder) and anxiety disorders, although this effect was primary accounted for by DSM-III-R overanxious disorder.

Conclusions—Stringent tests of homotypic and heterotypic prediction patterns suggest a more developmentally and diagnostically nuanced picture in comparison with the previous literature. The putative link between adolescent and young adult depression was not supported. Oppositional defiant disorder was singular in being part of the developmental history of a wide range of young adult disorders.

Introduction

More than three quarters of young adults with psychiatric disorders first had a diagnosis between the ages of 11 and 18 1(see also 2-8), indicating that we must consider childhood and

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adolescent mental illness as a key risk factor for later psychiatric problems. However, there remain important questions about diagnostic prediction from childhood and adolescence to adulthood. Here we present new evidence about which childhood and adolescent disorders reliably precede later disorders.

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. Homotypic prediction has been identified in most studies predicting from childhood to late adolescence [9-13] and from childhood and adolescence to young adulthood [1, 2, 5, 14-19]. Indeed, prior disorder status is typically the strongest predictor of having that disorder later.

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. Although typically less common than homotypic prediction, two 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 [1, 3, 10, 11, 20]; partial support [9, 13, 16, 19]; depression predicting anxiety: full support [1, 3, 10, 11, 20]; partial support [13, 16]). Second, childhood/adolescent conduct/oppositional problems tend to precede adult anxiety and depression [1, 10, 16, 21-23], but not vice versa [1, 11, 13] (see [17] for an exception).

Taken together, homotypic prediction appears to be common across a range of disorders, whereas heterotypic prediction is limited to a few specific pathways, but a number of substantive and methodological issues complicate the interpretation of this work. First, many studies collapse childhood and adolescent disorders to predict adult disorders, despite the fact that important changes in the prevalence of some disorders, such as depression and conduct disorder, occur between childhood and adolescence [24-26]. Such changes could indicate that disorders at different ages result from different etiologic pathways (see, for instance, [27-30]), and so combining across childhood and adolescence could obscure important differences between childhood and adolescent prediction. It is also the case that little attention has been paid to the fact that these developmental pathways may be different for males and females. For example, the prevalence of depression changes at puberty for girls but not for boys[31].

Second, most studies collapse multiple, potentially heterogeneous, disorders into more general diagnostic groupings. For example, all anxiety disorders tend to be combined into one category, which could mask differences in prediction between individual anxiety disorders. Furthermore, oppositional defiant disorder and conduct disorder are often combined, despite the fact that they are distinct in factor analytic studies [32, 33], in risk factors studies[34] and when tested as predicting later problems[35]. Indeed, oppositional defiant disorder may be more likely to be linked with emotional disorders than conduct disorder [10].

Finally, studies of diagnostic predictors of later disorders have typically focused on pairwise associations; one earlier disorder predicting one later disorder. Yet, disorders tend to co-occur, and when comorbidity is not taken into account, pairwise associations may simply represent indirect effects rather than direct associations [36, 37]. For example, bivariate analyses may suggest that childhood anxiety disorders predict adolescent depression, but this association could be accounted for by comorbidity between childhood anxiety and depression.

Here, we use Great Smoky Mountain Study (GSMS) data from middle childhood through young adulthood and stringent criteria to examine a broader range of patterns of homotypic and heterotypic prediction from middle childhood and adolescence to young adulthood.

Methods

Sample and Procedures

The Great Smoky Mountains Study (GSMS) is a longitudinal study of the development of psychiatric disorder and need for mental health services in rural and urban youth. 14-19 A representative sample of three cohorts of children, age 9, 11, and 13 at intake, was recruited from 11 counties in western North Carolina. Potential participants were selected from the population of some 20,000 children using a household equal probability, accelerated cohort design.20 The accelerated cohorts design means that over several years of data collection each cohort reaches a given age in a different year, thus controlling for cohort effects.21 Youth with behavior problems were over sampled. A screening questionnaire was administered to a parent (usually the mother) of the first stage sample (N=3,896). The questionnaire consisted mainly of the externalizing (behavioral) problems scale of the Child Behavior Checklist [22], and was administered by telephone or in person. All children scoring above a predetermined cutpoint (the top 25% of the total scores), plus a 1 - in - 10 random sample of the rest (i.e., the remaining 75% of the total scores), were recruited for detailed interviews. Ninety-five percent of families contacted completed the telephone screen.

About 8% of the area residents and the sample are African American, and fewer than 1% are Hispanic. American Indians make up only about 3% of the population of the study area, which is overwhelmingly White, but were oversampled from school records to constitute 25% of the study sample. This was done by using the same screening procedure but recruiting everyone irrespective of screen score. Of the 456 Indian children identified, screens were obtained on 96%, and 81% (N=350) participated in the study. All subjects were given a weight inversely proportional to their probability of selection, so that the results presented are representative of the population from which the sample was drawn. Of all subjects recruited, 80% (N=1420) agreed to participate.

Table 1 presents the study design and participation rates at each wave. Data were collected on one cohort at ages 9 and 10, 2 cohorts at ages 11, 12, and 13, and all 3 cohorts at ages 14, 15, 16, 19 and 21. This paper presents data on 8806 parent-child pairs of interviews carried out across the age range 9 through 21. Participants were interviewed as closely as possible to their birthday each year. Funding constraints prevented our interviewing the youngest cohort from January 1997 through June 1998.

Interviews were completed with the child and their primary caregiver at their home or a convenient location until age 16 and with the young adult only thereafter. Before the interviews began, interviewees signed informed consent forms approved by the Duke Institutional Review Board. Across waves, an average of 82% of all possible interviews were completed, ranging from 75% to 94% at individual waves.

Measures

Psychiatric disorders were assessed using 1) the *Child and Adolescent Psychiatric Assessment* (CAPA)38-40 until age 16, and 2) the upward extension of the CAPA, the *Young Adult Psychiatric Assessment* (YAPA) at ages 19 and 21[40]. Scoring programs for the CAPA and YAPA, written in SAS 41, combined information about the date of onset, duration, and intensity of each symptom to create diagnoses according to the DSM-IV.29 With the exception of attention-deficit/hyperactivity disorder, for which only parental reports were counted, a symptom was counted as present if it was reported by either the parent or the child until age 16 or by the young adult at ages 19 and 21, as is standard clinical practice. Two-week test-retest reliability of CAPA diagnoses in children aged 10 to 18 years is comparable to that of other highly structured interviews (*K*s for individual disorders range from .56 to 1.0)[39]. To

minimize recall bias, the timeframe of both interviews for determining the presence of most psychiatric symptoms is the preceding 3 months. A previous publication suggested that there was little evidence of symptom attenuation (lower reported symptom levels in subsequent data waves), cohort differences, or differential dropout in this sample[11].

In the current study, disorder status was aggregated across childhood (i.e., ages 9 to 12), adolescence (i.e., ages 13 to 16) and young adulthood (i.e., ages 19 and 21). Childhood and adolescent diagnostic groupings included depression (including major depressive disorder, dysthymia, and depressive disorder, not otherwise specified), separation anxiety disorder in childhood, generalized anxiety disorders, conduct disorder (CD), attention-deficit/ hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD). DSM-III-R overanxious disorder (OAD) was also included because we had previously found that it predicted several adolescent disorders when diagnosed in childhood[9]. Disorders with a prevalence of less than 1% in a given developmental period were not included in analyses (e.g., separation anxiety in adolescence, social phobia, PTSD). Substance disorders (including those meeting abuse or dependence criteria) were only sufficiently common for inclusion beginning in adolescence. Young adult diagnostic groups include depression (same as childhood and adolescence groups), generalized anxiety, panic disorder without agoraphobia, agoraphobia without panic, and antisocial personality disorder (ASPD).

Analyses

Weighted logistic regression models were estimated using generalized estimating equations (GEE) implemented by SAS PROC GENMOD. Sampling weights were inversely proportional to selection probability. Robust variance (sandwich type) estimates were used to adjust the standard errors of the parameter estimates for the stratified design effects. Therefore, the resulting parameters are representative of the population from which the sample was drawn.

Ideally, all diagnoses from all developmental periods would be combined into a single path-type multivariate analysis. Because such complex models did not converge despite our reasonable sample size, we examined psychiatric status across two developmental periods at a time: from adolescence to young adulthood and from childhood to young adulthood. Homotypic and heterotypic patterns were determined by predicting each later disorder (e.g., depression in young adulthood) from each earlier disorder in a series of three models. In the bivariate, unadjusted model, a prior disorder was the single predictor of the later diagnosis. The unadjusted odds ratios resulting from Models 1 are reported in Tables 2 and 3. In the sex differences model, Model 2, the prior disorder, sex and the sex X prior disorder interaction were included (full results are available by request from the first author). In the final comorbidity or adjusted model, the prior disorder that corresponded to the outcome variable, and all other prior disorders were included. For example, childhood depression and all other childhood disorders were entered to predict young adult depression in the childhood-young adulthood model. The adjusted odds ratios resulting from the final model are reported in Tables 2 and 3. Inclusion of comorbid disorders provides a stringent test of homotypic and heterotypic prediction patterns. Where evidence of a sex by disorder interaction was detected, separate results for adjusted models were provided for males and females. Analyses involving childhood were based upon two GSMS cohorts (N=1008; <age 13 at intake) and those involving adolescence were based on all three GSMS cohorts, N = 1420.

As with any longitudinal study, not all assessments were completed at each data wave. Such missingness may affect prediction estimates if individuals with missing observations were more or less likely to have a psychiatric disorder than individuals with complete observations. To test for such effects, each individual's total number of missed assessments adjusted for the total number of expected assessments was predicted by the individual's psychiatric status at their first assessment (because all subjects have at least one assessment). Initial rates of

psychiatric disorder did not predict the likelihood of missing assessments (z= 1.5, p= .23) suggesting no effect of differential dropout. Therefore, observations missing within a given developmental period were excluded from analyses involving that development period. Because subjects were interviewed multiple times within each developmental period, subjects could miss one interview and still be included in analyses for that period.

Results

Homotypic Predictors

Tables 2 and 3 display the results from unadjusted and adjusted models for each pair of developmental groups (adolescence to young adulthood, childhood to young adulthood). Separate adjusted results are provided for males and females where a significant sex by disorder interaction was detected. Prediction between anxiety disorders, even if not the same disorder, is discussed as homotypic.

Adolescence to young adulthood—(See Table 2) In unadjusted models, homotypic prediction was found for antisocial personality disorder (from adolescent conduct disorder), depression, and substance disorders. Both generalized anxiety and panic disorders were predicted from overanxious disorder. The apparent association of adolescent depression with young adult depression was completely attenuated in adjusted models, whereas the homotypic prediction to substance, antisocial personality, generalized anxiety and panic disorders was undiminished. Overanxious disorder predicted later generalized anxiety and panic disorders more strongly for males than females.

Because the attenuation of homotypic prediction of depression in the comorbidity adjusted model was unexpected, possible informant effects were tested by running the adjusted models separately by parent and self-reports. The results were the same regardless of informant (parent-report OR= 0.6, 95% CI= 0.2, 2.7; self-report OR= 1.0, 95% CI= 0.4, 3.1). To clarify whether homotypic prediction for conduct disorder predicting antisocial personality disorder was an artifact of the diagnostic criterion for ASPD requiring prior evidence of CD before age 15, the adjusted model was rerun using an ASPD diagnosis in which subjects were not required to have displayed prior evidence of CD before age 15. Again, CD alone predicted ASPD (OR= 5.2, 95% CI= 1.4, 19.1).

Childhood to young adulthood—(see Table 3) As in the adolescence-young adulthood models, the link between conduct disorder and antisocial personality disorder was also found after adjusting for comorbidity. This link remained if the alternative form of the ASPD diagnosis (requiring no prior CD symptoms) was used (OR= 3.2, 95% CI= 1.0, 9.6).

There was evidence of prediction between various anxiety disorders in unadjusted models, although only three associations were significant after adjustment - overanxious disorder predicted panic disorder, separation anxiety disorder predicted agoraphobia without panic, and generalized anxiety disorder predicted agoraphobia without panic.

Heterotypic Predictors

Adolescence to young adulthood—In adjusted models, heterotypic patterns were found for depression and all anxiety disorders. Specifically, generalized anxiety and overanxious disorder predicted depression (in males for the depression-OAD link). Adolescent depression also predicted agoraphobia without panic. Adolescent oppositional defiant disorder predicted later generalized anxiety disorder, panic disorder without agoraphobia (in males only), and depression. Finally adolescent substance disorders predicted later depression.

Childhood to young adulthood—Compared to adolescent-young adulthood models, limited support for heterotypic prediction emerged in the childhood to young adulthood adjusted models: childhood oppositional defiant disorder predicted young adult depression, and childhood depression predicted panic disorder without agoraphobia and generalized anxiety.

Discussion

A review of previous studies suggested the following conclusions with respect to the continuity of disorders from childhood and adolescence to young adulthood: 1) Homotypic prediction is the norm from childhood/adolescence to young adulthood; 2) generalized anxiety and depression cross-predict; and 3) childhood/adolescent combined disruptive disorders (ODD and/or CD treated as a single entity) predict adult anxiety and depressive disorders in addition to antisocial personality disorder. This study provided developmentally differentiated and stringent tests of these patterns by 1) separating childhood from adolescent diagnostic predictors, 2) disaggregating specific anxiety and disruptive disorders, and 3) adjusting for comorbid conditions. Our results indicate that prediction patterns are actually more developmentally and diagnostically nuanced than the previous literature suggests.

In summary, although homotypic patterns were common, the path from adolescent to young adult depression was entirely accounted for by other comorbidities. Of childhood and adolescent anxiety disorders, DSM-III-R overanxious disorder was most likely to predict later young adult anxiety disorders. Generalized anxiety and depression cross-predicted, although these effects were not uniform across childhood and adolescence. Finally, a single behavioral disorder, adolescent ODD, preceded anxiety and depressive disorders.

Caveats

Before considering these findings in more detail, the following methodological considerations should be kept in mind. First, the GSMS participants lived in a rural area, and the study oversampled Native American children, with very few African Americans (8%) and no Latinos or Asian Americans. Thus, the sample is not representative of the U.S. population. However, comparison of the GSMS to other studies indicates that there are similar rates of cumulative childhood disorders in representative samples from other counties, other regions of the United States, and samples involving higher levels of Hispanic and African American youth[42, 43]. While the subjects were followed up to 12 years (age 9 to 21), cases will have been missed because subjects may have met criteria for disorders prior to our study, between assessments, or after their last assessment.

Second, some associations of moderate to large magnitude were nonsignificant because of the limited number of youth with the particular disorder. However, studies of homotypic and heterotypic predictors from child to adult psychiatric disorder that deal with a range of disorders are rare because they depend on large, longitudinal, community-based samples carefully characterized over many years. We are unaware of any currently existing studies with greater power to address these questions. Finally, our research aim was to determine which childhood and adolescent disorders reliably precede young adult disorders. This did not allow us to examine the chronological order of disorders *within* a developmental period. For the current analysis, the order of the disorders within a developmental period, however, has no effect on the strength of the association with the young adult outcome.

A terminological note is also in order. We have referred here to homotypic and heterotypic “prediction” rather than “continuity,” despite the latter term's common usage. We believe that typical measurement schedules in prospective studies cannot adequately capture (dis) continuity, because observations of disorders tend to be intermittent (rather than continuous).

In fact, observations/interviews are often interspersed with gaps in measurement, and therefore cannot truly capture (dis)continuity.

Homotypic Prediction

Although homotypic patterns were identified (e.g., CD to ASPD and substance-related disorders), homotypic patterns were less common than previously reported by other studies when accounting for comorbidity between disorders. There was no evidence of homotypic prediction for depression and homotypic prediction to young adult anxiety disorders was primarily accounted for by DSM-III-R OAD, rather than by DSM-IV GAD.

In preparation for DSM-IV, Klein and colleagues[44] reviewed taxonomic issues related to the DSM-III-R anxiety disorders. Overanxious disorder was the focus of particular attention because it included a group of heterogeneous worries (e.g., about the future, academic performance, self-consciousness) and was highly comorbidity with other anxiety disorders (particularly GAD). Despite these concerns, it was recommended that it be retained as a childhood anxiety disorder, but with modified criteria to reduce overlap with other disorders. Instead, it was eliminated with the rationale that these children would likely receive a diagnosis of DSM-IV GAD. In a prior study, our group compared the relative predictive of validity of childhood OAD as compared to DSM-IV GAD in predicting adolescent disorders. OAD not only predicted later anxiety disorders but also predicted adolescent depression and conduct disorder. In contrast, DSM-IV GAD only predicted later conduct disorder. In this study predicting young adult disorder status, OAD again predicted both anxiety disorders and depression. This is in line with findings from the New York Child Longitudinal Study in which OAD predicted young adult depression, social phobia, and generalized anxiety[3]. Together, these findings suggest that the DSM-IV GAD criteria are insufficient for assessing the full range of "generalized anxiety" in children and adolescents and fail to identify anxious children at risk for a range of later disorders. It seems that Klein and her colleagues were right to suggest that OAD should have been retained in the DSM-IV. We recommend its rehabilitation in the DSM-V.

The example of depression illustrates the importance of separating childhood from adolescent predictors and controlling for comorbidities: The significant bivariate prediction from adolescent to young adult depression (OR=3.3) was entirely accounted for by comorbidity of adolescent depression with adolescent ODD, GAD, OAD and substance disorders (OR for depression reduced to 0.8), whereas there was no direct prediction from childhood depression to young adult depression even in the bivariate models. This suggests that the apparent association between adolescent depression and young adult depression is epiphenomenal, resulting from the direct associations between comorbid adolescent disorders and later depression.

This may appear to be a clear departure from the consensus of previous research[45], but actually it is not. Many studies looking at the adolescence-young adult depression link have used highly selected or clinical samples and/or failed to account for common comorbid disorders[46-50]. While these studies can demonstrate that adolescent depression precedes young adult depression, they are insufficient, on their own, to provide evidence of direct prediction. Such evidence can only come from community samples that assess for a range of disorders, in addition to depression, at multiple time points in both adolescence and young adulthood.

To date, three such studies have been published. The first, a community sample of adolescents followed into young adulthood by Lewinsohn and colleagues[5 p.61], concludes that their results "clearly illustrate a strong pattern of continuity for depression." Their initial analyses demonstrate higher risk of later depression for a group of adolescents with MDD as compared to groups with either no depression or a nonaffective disorder. In this comparison, however,

adolescent MDD cases were allowed to have other Axis I nonaffective disorders (and 51.0% did). A more stringent test compared risk for depression between a group with "pure" adolescent depression and a group with comorbid depression and found no difference. This might seem to suggest that even after controlling for comorbidity there is a link from adolescent to young adult depression, but rates of young adult depression are not provided for either of these two groups. It is entirely plausible then that the "pure" MDD group could both only be marginally associated (or not associated) with later MDD and, at the same time, not be statistically different from the comorbid MDD group. Without knowledge of the rates of young adult depression in these two groups, one cannot draw any conclusions about the role of comorbidity in the adolescent-young adult MDD link.

The second study tested this link in a birth cohort of 1265 children and concluded that there was a "direct and specific" link from adolescent depression to later depression[51]. The study design provides a rather stringent test for the outcomes of adolescent depression by accounting for the effects of anxiety disorders, early cigarette smoking, conduct disorders, alcohol abuse, and a range of other putative risk factors. At the same time, the negative outcomes (including depression) are assessed for ages 16 to 21 and thus overlap both with late adolescence and young adulthood. If there were a rather punctuated shift in depression between adolescence and young adulthood, it would not be detected by this design. But is this likely? In fact, such a striking shift occurs in depression a few years earlier in the pubertal transition from childhood to adolescence[52], so this possibility cannot be rejected *a priori*. While the pubertal shift is associated with significant biochemical changes, the shift to young adulthood and the associated transition to independent living may be similarly substantial in the social domain.

The final study by Pine and colleagues found that the best-fitting multivariable prediction model of young adult depression did *not* include adolescent depression after accounting for comorbidities[3]. As with our findings, there was evidence of significant prediction from adolescent to adult depression in bivariate analyses. This significant effect was primarily attenuated by inclusion of CD in the best-fitting adjusted model; ODD was not included in their analysis (Pine, D., personal communication).

We suggest, therefore, that the early conclusions about the link between adolescent and young adult depression may have been premature. This putative link may be attenuated by comorbid adolescent disorders, particularly anxiety and behavioral disorders. It may also be the case that there is a rather punctuated shift in the natural course of depression around age 17 or 18. This hypothesized shift is consistent with an emerging literature that suggests heterogeneity in childhood/adolescent and adult depressions with respect to biological correlates and psychosocial predictors[30, 53].

Heterotypic Findings

Each heterotypic pattern identified from previous research was extended by the current study. An emerging body of literature has suggested that generalized anxiety not only reliably precedes depression[3, 54-56], but vice versa3. By disaggregating childhood and adolescent diagnoses, the current study found that this pattern was developmentally nuanced: only childhood depression predicted young adult GAD and only adolescent GAD predicted later depression. In addition, adolescent OAD was a stronger predictor of later depression in males than GAD. Furthermore, the cross-prediction was stronger than homotypic prediction for these two disorders (a finding also previously reported by Pine and colleagues3).

Together with evidence that GAD and depression co-occur more often with one another than with other disorders20, 57 and have shared genetic etiology[58, 59], this lends support to the notion of grouping these disorders more closely than is currently reflected in the DSM-IV. Cross-prediction (or sequential comorbidity) is not, however, very strong evidence of

diagnostic unity. Childhood and adolescent GAD and MDD predicted to *different* adult disorders and young adult GAD and MDD were predicted by *different* childhood and adolescent disorders. Our findings, together with those of other longitudinal epidemiologic samples[3, 20], suggest that GAD and MDD, while closely related, are distinct both in terms of in their natural courses and developmental histories.

A recent study by Kim-Cohen and colleagues' found that CD/ODD "was a part of the developmental history of every adult disorder" (p. 713, 1). Because CD and ODD were combined in that study, it was unclear, for any given outcome disorder, whether it was preceded by ODD, CD or both. We found that young adult depression and anxiety disorders were preceded by adolescent ODD, but not CD. This finding is at odds with the traditional 'failure model' 22, 60 which suggests that depression results from the social and educational failures that often follow conduct disorder. As with homotypic patterns of depression, the bivariate link between adolescent CD and young adult depression in our study was entirely accounted for by comorbid disorders (here, adolescent GAD, ODD, and substance disorders). If it is indeed ODD, rather than CD, that predicts later depression then this might suggest an amended 'failure model' which emphasizes the social and emotional consequences of irritability and interpersonal difficultness rather than the legal and social sequelae of delinquency and overt aggression.

As part of the research agenda for DSM-V, questions have been raised about the diagnostic and predictive validity of ODD after accounting for comorbid disorders (e.g., ADHD, CD)[61]. Our findings suggest that ODD is a singular disorder in being part of the developmental history of many young adult affective and anxiety disorders. No other childhood or adolescent disorder demonstrated such pleiotropic effects. In DSM-IV, ODD is ruled out if criteria for CD are met. In ICD-10, ODD is a mere subtype of CD. Our data suggest that this subordination of ODD may be misguided. One accepted measure of the utility of a psychiatric diagnosis is the extent to which it predicts future psychiatric functioning 62, 63. On this measure, ODD may be in a class by itself.

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References

1. Kim-Cohen J, Caspi A, Moffitt T, Harrington H, Milne B, Poulton R. Prior juvenile diagnoses in adults with mental disorder: Developmental follow-back of a prospective-longitudinal cohort. Archives of General Psychiatry 2003;60:709–717. [PubMed: 12860775]
2. Ferdinand RF, Verhulst FC, Wiznitzer M. Continuity and change of self-reported problem behaviors from adolescence into young adulthood. Journal of the American Academy of Child and Adolescent Psychiatry 1995;34(5):680–690. [PubMed: 7775363]
3. Pine DS, Cohen P, Gurley D, Brook J, Ma Y. The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. Archives of General Psychiatry 1998;55:56–64. [PubMed: 9435761]
4. Brook JS, Cohen P, Brook DW. Longitudinal study of co-occurring psychiatric disorders and substance use. Journal of the American Academy of Child and Adolescent Psychiatry 1998;37:322–330. [PubMed: 9519638]
5. Lewinsohn PM, Rohde P, Klein DN, Seeley JR. Natural course of adolescent major depressive disorder: I Continuity into young adulthood. Journal of the American Academy of Child and Adolescent Psychiatry 1999;38(1):56–63. [PubMed: 9893417]

6. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. Archives of General Psychiatry June 1;2005 62(6):593–602. 2005. [PubMed: 15939837]
7. Christie KA, Burke JD, Regier DA, Rae DS, Boyd JH, Locke BZ. Epidemiologic evidence for early onset of mental disorders and higher risk of drug abuse in young adults. American Journal of Psychiatry 1988;145:971–975. [PubMed: 3394882]
8. Burke KC, Burke JD Jr, Rae DS, Regier DA. Comparing age at onset of major depression and other psychiatric disorders by birth cohorts in five US community populations. Archives of General Psychiatry 1991;48(9):789–795. [PubMed: 1929768]
9. Bittner A, Egger HL, Costello EJ, Foley D, Angold A. What do childhood anxiety disorders predict? Journal of Child Psychology & Psychiatry 2007;48(12):1174–1183. [PubMed: 18093022]
10. Burke JD, Loeber R, Lahey BB, Rathouz P. Developmental transitions among affective and behavioral disorders in adolescent boys. Journal of Child Psychology and Psychiatry Nov.;2005 46(11):1200–1210. [PubMed: 16238667]
11. Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. Prevalence and development of psychiatric disorders in childhood and adolescence. Archives of General Psychiatry 2003;60:837–844. [PubMed: 12912767]
12. Fergusson DM, Lynskey MT, Horwood LJ. Factors associated with continuity and change in disruptive behavior patterns between childhood and adolescence. Journal of Abnormal Child Psychology 1996;24:533–553. [PubMed: 8956083]
13. Orvaschel H, Lewinsohn PM, Seeley JR. Continuity of psychopathology in a community sample of adolescents. Journal of the American Academy of Child and Adolescent Psychiatry 1995;34(11):1525–1535. [PubMed: 8543521]
14. Goodwin RD, Fergusson DM, Horwood L. Early anxious/withdrawn behaviours predict later internalising disorders. Journal of Child Psychology and Psychiatry 2004;45:874–883. [PubMed: 15056317]
15. Ferdinand RF, Verhulst FC. Psychopathology from adolescence into young adulthood: An 8-year follow-up study. American Journal of Psychiatry 1995;152(11):1586–1594. [PubMed: 7485620]
16. Hofstra, MB.; van der Ende, J.; Verhulst, FC. Child and adolescent problems predict DSM-IV disorders in adulthood: A 14-year follow-up of a Dutch epidemiological sample. 2002. p. 182-189.41
17. Hofstra, MB.; van der Ende, J.; Verhulst, FC. Continuity and change of psychopathology from childhood into adulthood: A 14-year follow-up study. 2000. p. 850-858.39
18. Newman DL, Moffitt TE, Silva PA, Avshalom C, Magdol L. Psychiatric disorder in a birth cohort of young adults: Prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. Journal of Consulting and Clinical Psychology 1996;64(3):552–562. [PubMed: 8698949]
19. Sourander AMD, Multimaki PMD, Nikolakaros GMD, et al. Childhood Predictors of Psychiatric Disorders Among Boys: A Prospective Community-Based Follow-up Study From Age 8 Years to Early Adulthood. Journal of the American Academy of Child & Adolescent Psychiatry 2005;44(8):756–767. [PubMed: 16034277]
20. Moffitt TE, Harrington H, Caspi A, et al. Depression and Generalized Anxiety Disorder: Cumulative and Sequential Comorbidity in a Birth Cohort Followed Prospectively to Age 32 Years. Arch Gen Psychiatry June 1;2007 64(6):651–660. 2007. [PubMed: 17548747]
21. Zoccolillo M. Co-occurrence of conduct disorder and its adult outcomes with depressive and anxiety disorders: A review. Journal of the American Academy of Child and Adolescent Psychiatry 1992;31:547–556. [PubMed: 1592790]
22. Capaldi DM. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: II. A 2-year follow-up at grade 8. Development and Psychopathology 1992;4(1):125–144.
23. Loeber R, Keenan K. Interaction between conduct disorder and its comorbid conditions: Effects of age and gender. Clinical Psychology Review 1994;14:497–523.
24. Rutter M. Adolescence as a transition period: Continuities and discontinuities in conduct disorder. Journal of Adolescent Health 1992;13:451–460. [PubMed: 1390809]
25. Angold A, Costello EJ, Worthman CM. Puberty and depression: The roles of age, pubertal status, and pubertal timing. Psychological Medicine 1998;28:51–61. [PubMed: 9483683]

26. Costello, EJ.; Egger, HL.; Angold, A. The developmental epidemiology of anxiety disorders: Phenomenology, prevalence, and comorbidity.. In: Swedo, S.; Pine, D., editors. Child and Adolescent Psychiatric Clinics of North America. Vol. 14. Elsevier; New York: 2005. p. 631-648.
27. Rice F, Harold GT, Thappar A. Assessing the effects of age, sex, and shared environment on the genetic aetiology of depression in childhood and adolscence. Journal of Child Psychology 2002;43:1039–1051.
28. Silberg J, Pickles A, Rutter M, et al. The influence of genetic factors and life stress on depression among adolescent girls. Archives of General Psychiatry 1999;56:225–232. [PubMed: 10078499]
29. Eley T, Stevenson J. Exploring the covariation between anxiety and depression symptoms: A genetic analysis of the effects of the age and sex. Journal of Child Psychiatry 1999;40:1273–1282.
30. Kaufman J, Martin A, King R, Charney D. Are child-, adolescent-, and adult-onset depression one and the same disorder? Biological Psychiatry 2001;49:980–1001. [PubMed: 11430841]
31. Angold A, Worthman CW. Puberty onset of gender differences in rates of depression: A developmental, epidemiologic and neuroendocrine perspective. Journal of Affective Disorders 1993;29:145–158. [PubMed: 8300975]
32. Lahey BB, Rathouz PJ, Hulle C, et al. Testing Structural Models of DSM-IV Symptoms of Common Forms of Child and Adolescent Psychopathology. Journal of Abnormal Child Psychology 2008;36 (2):187–206. [PubMed: 17912624]
33. Lahey BB, Applegate B, Waldman ID, Loft JD, Hankin BL, Rick J. The Structure of Child and Adolescent Psychopathology: Generating New Hypotheses. Journal of Abnormal Psychology Aug; 2004 113(3):358–385. 2004. [PubMed: 15311983]
34. Shanahan L, Copeland W, Costello EJ, Angold A. Specificity of putative psychosocial risk factors for psychiatric disorders in children and adolescents. Journal of Child Psychology & Psychiatry 2008;49(1):34–42. [PubMed: 18181879]
35. Rowe R, Maughan B, Pickles A, Costello EJ, Angold A. The relationship between DSM-IV oppositional defiant disorder and conduct disorder: Findings from the Great Smoky Mountains Study. Journal of Child Psychology and Psychiatry 2002;43:365–373. [PubMed: 11944878]
36. Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry 1999;40:57–87. [PubMed: 10102726]
37. Ford T, Goodman R, Meltzer H. The British child and adolescent mental health survey 1999: The prevalence of DSM-IV disorders. Journal of the American Academy Child and Adolescent Psychiatry 2003;42:1203–1211.
38. Angold A, Costello E. The Child and Adolescent Psychiatric Assessment (CAPA). Journal of the American Academy of Child and Adolescent Psychiatry 2000;39:39–48. [PubMed: 10638066]
39. Angold A, Costello EJ. A test-retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment (CAPA-C). Psychological Medicine 1995;25:755–762. [PubMed: 7480452]
40. Angold, A.; Cox, A.; Prendergast, M., et al. The Young Adult Psychiatric Assessment (YAPA). Duke University Medical Center; Durham, NC: 1999.
41. *SAS/STAT® Software: Version 9* [computer program]. Version.. SAS Institute, Inc.; Cary, NC: 2004.
42. Jaffee S, Harrington H, Cohen P, Moffitt TE. Cumulative prevalence of psychiatric disorder in youth. Journal of the American Academy Child and Adolescent Psychiatry 2005;44(5):406–407.
43. Roberts R, Attkisson C, Rosenblatt A. Prevalence of psychopathology among children and adolescents. American Journal of Psychiatry 1998;155:715–725. [PubMed: 9619142]
44. Klein, RG.; Tancer, NK.; Werry, JS. Anxiety disorders of childhood or adolescence.. In: Widiger, TA.; Frances, AJ.; Pincus, HA.; Ross, R.; First, MB.; Davis, W., editors. DSM-IV Sourcebook. Vol. 3. American Psychiatric Association; Washington, DC: 1997. p. 221-239.
45. Kessler R, Avenevoli S, Merikangas K. Mood disorders in children and adolescents: An epidemiologic perspective. Biological Psychiatry 2001;49:1002–1014. [PubMed: 11430842]
46. Garber J, Kriss MR, Koch M, Lindholm L. Recurrent depression in adolescents: A follow-up study. Journal of the American Academy of Child and Adolescent Psychiatry 1988;27:49–54. [PubMed: 3343206]
47. Harrington R, Fudge H, Rutter M, Pickles A, Hill J. Adult outcomes of childhood and adolescent depression. Archives of General Psychiatry 1990;47:465–473. [PubMed: 2184797]

48. Rao U, Ryan ND, Birmaher B, et al. Unipolar depression in adolescents: Clinical outcome in adulthood. Journal of the American Academy of Child and Adolescent Psychiatry 1995;34(5):566–578. [PubMed: 7775352]
49. Rao U, Hammen C, Daley S. Continuity of depression during the transition to adulthood: A 5-year longitudinal study of young women. Journal of the American Academy of Child and Adolescent Psychiatry 1999;38:908–915. [PubMed: 10405510]
50. Weissman MM, Wolk S, Goldstein RB, et al. Depressed adolescents grown up. JAMA 1999;281:1707–1713. [PubMed: 10328070]
51. Fergusson DM, Woodward LJ. Mental health, educational, and social role outcomes of adolescents with depression. Archives of General Psychiatry 2002;59:225–231. [PubMed: 11879160]
52. Angold A, Erkanli A, Silberg J, Eaves L, Costello E. Depression scale scores in 8-17-year-olds: Effects of age and gender. Journal of Child Psychology and Psychiatry 2002;43:1052–1063. [PubMed: 12455926]
53. Jaffee SR, Moffitt TE, Caspi A, Fombonne E, Poulton R, Martin J. Differences in early childhood risk factors for juvenile-onset and adult-onset depression. Archives of General Psychiatry 2002;59:215–222. [PubMed: 11879158]
54. Breslau N, Schultz L, Peterson E. Sex differences in depression: A role for preexisting anxiety. Journal of Psychiatric Research 1995;58:1–12.
55. Parker G, Wilhelm K, Mitchell P, Austin M-P, Roussos J, Gladstone G. The influence of anxiety as a risk to early onset major depression. Journal of Affective Disorders 1999;52(1-3):11–17. [PubMed: 10357013]
56. Lewinsohn PM, Rohde P, Seeley JR, Klein DN, Gotlib IH. Natural course of adolescent major depressive disorder in a community sample: Predictors of recurrence in young adults. American Journal of Psychiatry 2000;157:1584–1591. [PubMed: 11007711]
57. Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. Archive of General Psychiatry June 1;2005 62(6):617–627. 2005.
58. Kendler KS, Gardner CO, Gatz M, Pedersen NL. The sources of co-morbidity between major depression and generalized anxiety disorder in a Swedish national twin sample. Psychol Med Mar; 2007 37(3):453–462. [PubMed: 17121688]
59. Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ. Major depression and generalized anxiety disorder: Same genes, (partly) different environments? Archives of General Psychiatry 1992;49:716–722. [PubMed: 1514877]
60. Capaldi DM, Stoolmiller M. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: III. Prediction to young-adult adjustment. Development and Psychopathology 1999;11:59–84. [PubMed: 10208356]
61. Moffitt T, Arseneault L, Jaffee S, et al. Research Review: DSM-V conduct disorder: research needs for an evidence base. Journal of Child Psychology and Psychiatry 2008;49(1):3–33. [PubMed: 18181878]
62. Meehl P. Towards an integrated theory of schizotaxia, schizotypy, and schizophrenia. Journal of Personality Disorders 1990;(4):1–99.
63. Kendell R, Jablensky A. Distinguishing between the validity and utility of psychiatric diagnoses. American Journal of Psychiatry 2003;160(1):4–12. [PubMed: 12505793]

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Table 1

Great Smoky Mountains Study: Data collection by cohort

Cohort	Age	1993	94	95	96	97	98	99	00	01	02	03	04	05
A N=508	**9**	A1												
	10		A2											
B N=497	**11**	B1		A3										
	12		B2		A4									
C N=415	**13**	C1		B3										
	14		C1		B4		A5							
	15			C3		B5		A6						
	16				C4		B6		A7					
	19							C5		B7		A8		
	21									C6		B8		A9
Participation %		*94*	*91*	*87*	*78*	*80*	*81*	*74*	*81*	*81*		*80*		*76*

Light shading indicates the childhood observations, medium shading indicates the adolescent observations, and dark shading indicates young adulthood.

Table 2

Young adult diagnoses predicted from adolescent diagnoses: Percent prevalence, odds ratio, (95% CI)

	Young Adult Disorders											
	GAD		**PAN**		**AGOR**		**DEP**		**ASPD**		**SUB**	
	3.1% (1.3, 4.8)		**5.0% (2.8, 7.1)**		**4.5% (2.4, 6.6)**		**5.2% (3.1, 7.2)**		**1.9% (0.6, 3.2)**		**18.0% (14.2, 21.9)**	
Adolescent dx	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present
OAD	2.3%	17.6%	3.2%	40.7%	4.3%	8.6%	4.9%	11.0%	1.9%	1.6%	18.3%	13.3%
Unadjusted	**8.9 (2.1, 37.2)**		**20.9 (7.0, 62.5)**		2.1 (0.5, 9.9)		2.4 (0.6, 10.6)		0.8 (0.2, 4.2)		0.7 (0.2, 2.4)	
Adjusted†	F: 6.0 (0.4, 88.5)		**F: 13.2 (2.8, 61.1)**		1.3 (0.3, 6.1)		F: 0.1 (0.1, 0.7)		0.6 (0.1, 3.2)		0.4 (0.1, 1.4)	
	M: 51.7 (1.8, 149.6) *		**M: 77.0 (8.5, 701.8)** *				**M: 37.1 (3.6, 387.7)** *					
GAD	3.0%	4.2%	4.8%	11.3%	4.4%	5.4%	4.7%	26.5%	1.9%	1.5%	18.2%	11.1%
Unadjusted	1.4 (0.4, 5.6)		2.5 (0.9, 7.3)		1.2 (0.3, 4.5)		**7.4 (1.9, 28.5)** *		1.0 (0.2, 5.2)		0.6 (0.2, 1.5)	
Adjusted†	**F: 0.1 (0.1, 0.7)**		0.3 (0.1, 1.8)		0.4 (0.1, 2.9)		**4.6 (1.0, 21.9)**		0.6 (0.1, 4.4)		0.4 (0.1, 1.5)	
	M: 1.6 (0.2, 16.2)											
DEP	2.4%	11.8%	3.9%	18.2%	4.0%	10.9%	4.5%	13.7%	2.1%	0.0%	17.6%	24.3%
Unadjusted	**5.5 (1.4, 21.6)**		**5.4 (1.9, 15.9)** *		3.0 (0.8, 10.7)		**3.3 (1.1, 10.5)**		--		1.5 (0.7, 3.5)	
Adjusted†	F: 0.5 (0.1, 3.0)		1.0 (0.2, 4.4)		**4.3 (1.3, 13.9)**		0.9 (0.2, 4.5)		--		1.7 (0.6, 4.7)	
	M: 1.6 (0.4, 7.0)											
ADHD	3.1%	0.0%	5.0%	4.2%	4.5%	4.2%	5.2%	8.5%	1.9%	0.0%	18.0%	22.0%
Unadjusted	--		0.9 (0.1, 6.6)		1.0 (0.1, 7.4)		1.7 (0.4, 7.7)		--		1.3 (0.5, 3.4)	
Adjusted†	--		0.5 (0.1, 6.9)		0.9 (0.1, 9.5)		0.8 (0.1, 5.1)		--		1.1 (0.3, 3.4)	
CD	2.8%	7.2%	4.6%	10.6%	4.3%	6.6%	4.3%	18.2%	1.5%	7.7%	17.6%	24.7%
Unadjusted	2.7 (0.5, 15.2)		2.5 (0.6, 10.9)		1.6 (0.3, 9.6)		**4.9 (1.5, 16.4)** *		**5.4 (1.1, 27.0)**		1.5 (0.7, 3.4)	
Adjusted†	0.8 (0.1, 10.0)		1.1 (0.2, 4.8)		1.8 (0.5, 7.0)		2.1 (0.7, 6.9)		**5.6 (1.0, 29.2)**		1.1 (0.4, 3.1)	
ODD	2.0%	15.9%	4.1%	16.0%	4.3%	6.3%	4.2%	17.4%	1.8%	3.2%	17.7%	22.8%
Unadjusted	**9.2 (2.7, 31.4)** *		**4.5 (1.5, 13.5)** *		1.5 (0.3, 7.0)		**4.8 (1.8, 13.4)** *		1.8 (0.6, 5.6)		1.4 (0.7, 2.9)	
Adjusted†	**6.3 (2.0, 19.8)** *		F: 2.0 (0.3, 13.5)		0.8 (0.2, 2.7)		**2.8 (1.0, 8.3)**		1.1 (0.3, 4.6)		1.2 (0.5, 3.0)	
			M: 5.3 (1.6, 17.5) *									

	Young Adult Disorders											
	GAD		PAN		AGOR		DEP		ASPD		SUB	
	3.1% (1.3, 4.8)		5.0% (2.8, 7.1)		4.5% (2.4, 6.6)		5.2% (3.1, 7.2)		1.9% (0.6, 3.2)		18.0% (14.2, 21.9)	
Substance	2.6%	7.5%	4.4%	9.9%	4.8%	1.1%	4.0%	15.5%	1.8%	2.7%	16.2%	34.6%
Unadjusted	3.1 (0.8, 12.2)		2.4 (0.7, 7.7)		**0.2 (0.1, 0.8)**		**4.4 (1.6, 12.0)** *		1.5 (0.5, 4.4)		**2.8 (1.4, 5.4)** *	
Adjusted†	1.5 (0.3, 8.7)		1.2 (0.3, 4.9)		**0.1 (0.0, 0.7)**		**3.1 (1.0, 9.6)**		0.9 (0.3, 3.3)		**2.7 (1.3, 5.5)** *	

Analyses are based on N = 1149 cases. OAD=Overanxious Disorder; GAD=Generalized Anxiety Disorder; ADHD=Attention-deficit/hyperactivity disorder; ODD=Oppositional defiant disorder; ASPD=Antisocial personality disorder CD=Conduct disorder; DEP=Depression; PAN=Panic disorder without agoraphobia; AGOR= Agoraphobia without panic; SUB=substance-related disorder. The following sex by disorder interactions were found: OAD predicting GAD, z=2.3, p=.02; OAD predicting AGOR, z=2.2, p=.03; OAD predicting DEP, z=3.3, p=.001; GAD predicting GAD, z=2.3, p=.02; DEP predicting GAD, z=2.0, p=.05; ODD predicting PAN, z=2.5, p=0.01. In these cases, results from adjusted models are presented separately for males and female.

† Adjusted for other disorders from ages 13 to 16. Odds ratios in bold are significant at the p<.05 level.

* = p<.01. – indicates no cases of the two disorders overlap.

Table 3

Young adult diagnoses predicted from childhood diagnoses Percent prevalence, odds ratio, (95% CI)

	Young Adult Disorders											
	GAD		**PAN**		**AGOR**		**DEP**		**ASPD**		**SUB**	
	2.6% (0.7, 4.4)		**5.7% (3.0, 8.3)**		**5.0% (2.4, 7.5)**		**5.1% (2.8, 7.4)**		**1.5% (0.2, 2.8)**		**17.9% (13.3, 22.4)**	
Childhood dx	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present	Prior dx Absent	Prior dx Present
OAD	2.6%	3.0%	5.3%	29.2%	4.9%	6.6%	5.2%	3.2%	1.4%	3.2%	17.8%	19.8%
Unadjusted	1.2 (0.1, 10.8)		**7.4 (1.2, 46.5)**		1.4 (0.3, 7.3)		0.6 (0.1, 5.3)		2.3 (0.2, 23.4)		1.1 (0.3, 3.9)	
Adjusted†	0.6 (0.1, 5.3)		**5.3 (1.33, 22.1)**		1.7 (0.3, 9.7)		0.6 (0.1, 5.9)		1.9 (0.2, 15.5)		0.8 (0.2, 2.6)	
SAD	2.4%	7.6%	5.6%	5.8%	4.4%	17.7%	4.8%	11.4%	1.5%	1.4%	12.5%	16.1%
Unadjusted	**3.4 (1.0, 11.5)**		1.0 (0.3, 3.3)		**4.7 (1.0, 21.7)**		**2.5 (1.0, 6.6)**		1.0 (0.1, 8.9)		2.2 (0.7, 6.8)	
Adjusted†	2.6 (0.8, 8.6)		1.1 (0.4, 3.4)		**3.0 (1.1, 8.0)**		2.0 (0.7, 5.4)		0.8 (0.1, 6.3)		1.7 (0.4, 7.0)	
GAD	2.4%	11.6%	5.7%	1.0%	4.5%	30.3%	5.0%	12.2%	1.5%	0.0%	12.4%	18.1%
Unadjusted	**5.3 (1.2, 24.4)**		0.2 (0.0, 1.5)		**9.3 (1.2, 70.4)** *		2.6 (0.7, 10.7)		--		3.1 (0.6, 16.8)	
Adjusted†	2.4 (0.6, 10.2)		**0.1 (0.0, 0.8)**		**10.7 (1.8, 64.5)** *		2.0 (0.4, 9.7)		--		1.8 (0.3, 10.1)	
DEP	2.4%	8.2%	5.3%	18.9%	4.9%	4.1%	5.1%	4.4%	1.5%	0.0%	12.8%	6.0%
Unadjusted	**3.7 (1.0, 13.7)**		4.2 (0.8, 22.0)		0.8 (0.2, 4.0)		0.9 (0.2, 3.6)		--		2.4 (0.7, 8.3)	
Adjusted†	**2.7 (1.0, 7.5)**		**4.3 (1.2, 15.5)**		0.2 (0.0, 2.1)		0.5 (0.1, 2.8)		--		1.8 (0.5, 5.7)	
ADHD	2.6%	2.6%	5.3%	12.9%	4.9%	5.2%	5.1%	5.2%	1.5%	1.3%	12.4%	18.1%
Unadjusted	1.0 (0.2, 5.3)		2.6 (0.4, 17.6)		1.1 (0.3, 3.7)		1.0 (0.3, 3.4)		0.8 (0.1, 8.0)		1.4 (0.4, 4.8)	
Adjusted†	0.7 (0.2, 3.4)		3.3 (0.3, 27.4)		1.3 (0.4, 4.5)		0.9 (0.3, 2.6)		0.6 (0.1, 4.9)		1.0 (0.2, 4.5)	
CD	2.5%	2.9%	5.9%	1.3%	5.0%	3.0%	5.1%	5.3%	1.2%	5.2%	11.7%	28.8%
Unadjusted	1.1 (0.3, 4.6)		0.2 (0.0, 1.2)		0.9 (0.3, 2.7)		1.0 (0.4, 2.9)		**4.3 (1.0, 18.6)**		**2.5 (1.0, 6.3)**	
Adjusted†	0.9 (0.3, 2.5)		0.1 (0.0, 0.7)		0.4 (0.1, 1.7)		0.7 (0.2, 1.9)		**5.2 (1.1, 23.8)**		2.3 (0.8, 6.1)	
ODD	2.5%	4.1%	5.7%	3.9%	5.0%	4.4%	4.8%	10.1%	1.5%	1.5%	12.1%	21.7%
Unadjusted	1.7 (0.5, 5.9)		0.7 (0.2, 2.1)		0.9 (0.3, 2.7)		**2.2 (1.0, 5.3)**		1.4 (0.3, 7.9)		1.8 (0.8, 4.2)	
Adjusted†	1.2 (0.5, 2.7)		0.6 (0.2, 2.0)		0.7 (0.2, 2.5)		**2.4 (1.0, 5.6)**		0.6 (0.1, 3.2)		1.0 (0.4, 2.6)	

Analyses are based on N = 838 cases. OAD=Overanxious Disorder; GAD=Generalized Anxiety Disorder; ADHD= Attention-deficit/hyperactivity disorder; ODD=Oppositional defiant disorder; ASPD=Antisocial personality disorder CD=Conduct disorder; DEP=Depression; PAN=Panic disorder without agoraphobia; AGOR= Agoraphobia without panic; SUB=substance-related disorder. No significant sex by disorder interactions were found when predicting young adult disorders from childhood disorders.

† Adjusted for other disorders from ages 9 to 12. Odds ratios in bold are significant at the p<.05 level.

* = p<.01.