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Diagnostic Transitions from Childhood to Adolescence to Early Adulthood

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Abstract

Background—Quantifying diagnostic transitions across development is needed to estimate the long-term burden of mental illness. This study estimated patterns of diagnostic transitions from childhood to adolescence and from adolescence to early adulthood.

Methods—Patterns of diagnostic transitions were estimated using data from three prospective, longitudinal studies involving close to 20,000 observations of 3,722 participants followed across multiple developmental periods covering ages 9 to 30. Common DSM psychiatric disorders were assessed in childhood (ages 9 to 12; two samples), adolescence (ages 13 to 18; three samples), and early adulthood (ages 19 to age 32; three samples) with structured psychiatric interviews and questionnaires.

Results—Having a disorder at an early period was associated with at least a 3-fold increase in odds for having a disorder at a later period. Homotypic and heterotypic transitions were observed for every disorder category. The strongest evidence of continuity was seen for behavioral disorders (particularly ADHD) with less evidence for emotional disorders such as depression and anxiety. Limited evidence was found in adjusted models for behavioral disorders predicting later emotional disorders. Adult substance disorders were preceded by behavioral disorders, but not anxiety or depression.

Conclusions—Having a disorder in childhood or adolescence is a potent risk factor for a range of psychiatric problems later in development. These findings provide further support for prevention and early life intervention efforts and suggest that treatment at younger ages, while justified in its own right, may also have potential to reduce the risk for disorders later in development.

Keywords

Epidemiology; Longitudinal; Depression; Anxiety; Behavioral disorders; Comorbidity

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Introduction

Historically child and adult mental illnesses have often been treated as separate in policy and service approaches. In recent years, both prospective studies following children into early adulthood and retrospective studies of adults reporting on a history of psychiatric problems have confirmed that 50 to 70% of adults with a psychiatric disorder previously had a disorder in childhood or adolescence (Kim-Cohen et al., 2003, Kessler et al., 2005, Copeland et al., 2011). Information on diagnostic transitions from earlier to later developmental periods can assist in formulating estimates of the burden of mental illness over the lifespan and in estimating the potential cost-benefit of prevention and early intervention efforts for policy decisions and evidence-based service planning.

Numerous studies have examined transitions from one disorder to another or one age group to another, often in selected or clinical samples. However, very few datasets are available worldwide with which to quantify transitions across a wide range of disorders with community-based, representative samples followed prospectively. The purpose of this analysis was to estimate diagnostic transitions from childhood to adolescence and from adolescence to adulthood across such studies.

Prior disorder status is typically the strongest diagnostic predictor of having the same disorder later, whether looking from childhood to adolescence (Burke et al., 2005, Costello et al., 2003, Fergusson et al., 1996b) or from adolescence to young adulthood (Kim-Cohen et al., 2003, Lewinsohn et al., 1999, Copeland et al., 2009b). Although such homotypic patterns are common, several heterotypic patterns of transition have also been described. First, broadly-defined anxiety and depression tend to predict one another from childhood/adolescence to adulthood (anxiety predicting depression: full support (Burke et al., 2005, Costello et al., 2003, Kim-Cohen et al., 2003, Pine et al., 1998); partial support (Copeland et al., 2009b); depression predicting anxiety: full support (Burke et al., 2005, Costello et al., 2003, Kim-Cohen et al., 2003, Pine et al., 1998); partial support (Copeland et al., 2009b)). Second, childhood/adolescent conduct/oppositional problems tend to precede adult anxiety and depression (Burke et al., 2005, Kim-Cohen et al., 2003, Loeber and Keenan, 1994), but not vice versa (Costello et al., 2003, Kim-Cohen et al., 2003) (see (Hofstra et al., 2000) for an exception), with some recent evidence that oppositional defiant disorder may account for this association (Copeland et al., 2009a). One pattern of heterotypic continuity that has received less support in the literature is anxiety or depression preceding conduct/oppositional disorders (Costello et al., 2003, Kim-Cohen et al., 2003, Copeland et al., 2009b) (see (Hofstra et al., 2000) for an exception). Finally, conduct problems assessed in childhood or adolescence often predict later substance problems (Kim-Cohen et al., 2003, Costello et al., 2003, Fergusson et al., 2007), although a portion of this effect may be accounted for by early substance problems (Copeland et al., 2009b).

Optimal studies for characterizing diagnostic transitions must be prospective to avoid the biases and forgetting endemic to retrospective recall (Moffitt et al., 2009, Angold et al., 1996), and population-based to minimize the biases related to case ascertainment. They must also use structured interviews that assess a full range of common psychiatric disorders. Each of the three studies included in this analysis meet these criteria. In addition, the three studies were similar enough in design to apply meta-analytic methods to estimate the risk transitions, as an alternative to a review of the entire body of literature on each pair of transitions or small groups of transitions.

Methods

Description of studies

***The* Christchurch Health and Development Study (CHDS)—**The CHDS is a longitudinal study of a birth cohort of 1,265 children born in the Christchurch urban region of New Zealand in 1977 (Fergusson et al., 1989). This cohort involved 97% of children born from April 15th to August 5th, 1977 and has been studied on 22 occasions to the age of 30. Data were gathered using face-to-face interviews with respondents including parents and birth cohort members, supplemented by data from official records. Signed consent was obtained for all aspects of data collection and the study has been subject to ethical review throughout the history of the research. The present analysis is based on data collected during assessments of the cohort in adolescence (ages 15, 16, and 18) and adulthood (ages 21, 25, 30). The samples assessed at these ages ranged between N= 1,003 and N= 1,025 participants, with these samples representing between 81% and 82% of the surviving cohort at each age.

***The* Great Smoky Mountain Study (GSMS)—**The GSMS is a longitudinal, representative study of children in 11 predominantly-rural counties in southeast United States begun in 1993 (Costello et al., 1996). Three cohorts of children, ages 9, 11, and 13 years, were recruited from a pool of some 20,000 children resulting in 1,420 participants. As compared to the US population, Native American children were overrepresented and African Americans and Latinos are underrepresented. Annual assessments were completed with the child and the primary caregiver until age 16 and then with the participant again at ages 19, 21, and 24/25/26 for a total of 9,904 assessments. Signed informed consent was completed for all aspects of data collection and the study protocol was approved by the Duke Institutional Review Board. An average of 82% of all possible interviews was completed across all waves, ranging from 75% to 94% at individual waves. The present analysis is based on data collected during assessments of the cohort in childhood (ages 9, 10, 11, and 12; N=1,009), adolescence (ages 13, 14, 15 and 16; N=1,297) and adulthood (ages 19, 21, 24/25/26; N=1,273).

***The* Dunedin Multidisciplinary Health and Development Study (DMHDS)—**The DMHDS is a longitudinal study of a birth cohort of 1,037 children born in the Dunedin urban region of New Zealand. This cohort was constituted at 3 years of age when the investigators enrolled 91% of consecutive births from April 1st, 1972 through March 3rd, 1973. Cohort families were primarily white (91%) and represented the full range of socioeconomic status in the general population of New Zealand's South Island. At each assessment, participants (including emigrants living overseas) were brought back to the research center within 60 days of their birthday for a full day of individual data collection. The study protocol was approved by the institutional review boards of the participating universities, and participants gave informed consent. The cohort was followed up at ages 5, 7, 9, 11, 13, 15, 18, 21 26 and age 32 (N=972; 96% of the living cohort members). The present analysis is based on data collected during assessments of the cohort in childhood (age 11; N=774 of N=925), adolescence (ages 13, 15 and 18; N=934) and adulthood (ages 21, 26 and 32; N=934).

Measures

Psychiatric status—All three studies used structured clinical interviews that were supplemented by questionnaires in some cases. Although slightly different diagnostic and assessment tools and assessment periods were used (summarized in table 1), disorder assessment was grounded in the DSM classification system. All the studies have published reports describing their assessment procedures, test-retest reliability and validity of

measures, scoring rules and diagnosis algorithms (CHDS (Fergusson and Horwood, 2001); GSMS (Angold and Costello, 2000); DMHDS (Newman et al., 1996)).

DSM disorder status was based upon meeting criteria for a given disorder (or category of disorders) at any assessment within a particular developmental period. Childhood diagnostic categories included depression (major depressive disorder, dysthymia, and depressive disorder, not otherwise specified), anxiety (separation anxiety disorder (childhood only), generalized anxiety disorder, DSM-III-R overanxious disorder, obsessive compulsive disorder, panic disorder, agoraphobia, simple phobia, social phobia), conduct disorder (CD), attention-deficit/hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD). In adolescence, the additional category of substance use disorders (SUD) was added (alcohol abuse/dependence, cannabis abuse/dependence, other drug abuse/dependence). In adulthood, only depression, anxiety, and SUD categories were included. An “any” disorder category included all of the disorders included in the subcategories. DSM-III-R overanxious disorder was included because it predicts later disorders status (Copeland et al., 2009b). Rare disorders (12-month prevalence < 1.0%) were not included in analyses. Some anxiety disorders were not included for all studies if they were rare within a single study (e.g., GSMS did not include obsessive-compulsive disorder in the anxiety group). Specifics are available from the first author by request.

Analyses

Point estimates (odds ratios and their confidence intervals) for each disorder pair were first produced from each dataset for the two developmental transition periods (childhood to adolescence and adolescence to adult) using logistic regression. Next, the risk estimates for each disorder pair transition were pooled across studies using the random effects model described by Horwood and colleagues (Horwood et al., 2010). For each transition, the ORs of all available studies were first log-transformed, then weighted and combined across the studies to obtain the overall effect size:

$$\beta_i = \ln(\mathrm{OR}_i) \quad (1)$$

$$\hat{\beta} = \frac{\sum_i w_i \beta_i}{\sum_i w_i} \quad (2)$$

where β_i is the natural logarithm of OR of study i, $\hat{\beta}$ is the weighted average of the β_i from all studies, and w_i is the reciprocal of the sum of the within- and between- studies variances (DerSimonian and Laird, 1986):

$$w_i = (\Delta^2 + s_i^2)^{-1} \quad (3)$$

The Δ^2 is the variance between studies estimated using the Cochran’s Q Statistic and s_i^2 is the variance of β_i. The standard error of the estimated effect size $\hat{\beta}$ is

$$\mathrm{SE}(\hat{\beta}) = \left(\sum_i w_i\right)^{-\frac{1}{2}} \quad (4)$$

with the 95% CI of $\hat{\beta}$ given by

$$95\%\ \mathrm{CI}=\exp(\hat{\beta} \pm 1.96\mathrm{SE}) \quad (5)$$

The Cochran's Q was also used to assess the non-homogeneity of the parameters across all studies (Patil, 1975).

Unadjusted ORs are provided to estimate transition patterns as they appear naturally in the populations. Adjusted ORs account for comorbidity between the predictor disorder and other psychiatric disorders at the same time period. Such estimates may help disentangle the independent effect of correlated predictors. Previous studies (including studies involving these samples) provide little support for sex-specific transitions (see (Copeland et al., 2009b)) and thus all results are pooled across males and females. For the childhood to adolescence transitions, childhood diagnostic status was available from GSMS (4 possible observations) and DMHDS (1 observation). Psychiatric diagnostic status was not assessed in CHDS prior to age 15. Adolescent status was assessed at four time points in GSMS and three in DMHDS. For the adolescent to adult transitions, unadjusted ORs were available from all three studies. All studies assessed diagnostic status at least three times in adolescence and at least twice in young adulthood. While a previous GSMS study looked at adolescent to young adult transitions, this analysis included new data from the interview of participants in their mid-20s (Copeland et al., 2009b). Study-specific estimates are available in online supplemental tables 1 and 2.

Results

Childhood to Adolescence Transitions

Table 2 displays the results from transitions from childhood to adolescence in GSMS and DMHDS (table 2). Cochran's Q values are provided to test for estimate heterogeneity. Six of forty-two associations had significant Q values in either the adjusted or unadjusted results (e.g., Conduct-Depression, Depression-Anxiety, Conduct-Conduct, ADHD-Conduct, Conduct –ADHD, and ADHD-SUD). This rate higher than would be expected by chance (likelihood of 6+ significant findings by chance is 1.7%), but the vast majority of estimates did not display heterogeneity.

Thirty-one of forty-two unadjusted transitions were statistically significant providing strong support for the overall prediction from childhood to adolescence (likelihood of 31+ significant findings by chance is effectively zero). The increased odds of having an adolescent disorder if one had a history of a childhood disorder as compared to those with no history of a childhood disorder was 3.71 (95%CI 2.65–5.19) and each individual adolescent disorder was significantly predicted by having had a childhood disorder. Homotypic transitions were supported for almost all disorders (depression was the exception) and all homotypic odds ratios (including depression) were greater than 3.0. Adolescent disorders were also predicted by a heterotypic disorder for all behavioral disorders. Strong evidence was found for anxiety and depression predicting one another. Behavioral disorders (ADHD, Conduct, ODD, and SUD) tended to be preceded by both behavioral and emotional disorders (anxiety and depression).

Evidence of widespread heterotypic prediction was attenuated in adjusted models. No single childhood disorder predicted either adolescent depression or anxiety (although some ORS were greater than 2.0). Conduct disorder was predicted significantly by depression only, but childhood conduct disorder had the largest individual estimate (OR=4.70). ADHD was predicted by ADHD only and ODD was predicted by ODD and ADHD. Substance problems in adolescence were predicted by childhood depression and conduct disorder.

Adolescence to Early Adulthood Transitions

Table 3 displays the results from unadjusted transitions from adolescence to adulthood across three studies employing DSM-based diagnoses aggregated across multiple adolescent and young adult assessments. Six of twenty-eight transitions had significant Q values at $p < 0.05$ (Depression-Anxiety, Depression-SUD, Depression-Any, Anxiety-Any, Conduct-SUD, and ODD-Anxiety). This rate is consistent with real differences across studies (likelihood of 8+ significant findings by chance is 0.23%). Only one of these associations differed in terms of statistical significance across the three samples (Depression-SUD). This suggests that estimate heterogeneity is primarily limited to the strength of the association rather than whether there is an association.

Twenty-five of twenty-eight unadjusted transitions were statistically significant (likelihood of 25+ significant findings by chance is effectively zero). Similar to the earlier transition, the odds of having an adult disorder was over 3-fold higher in those that had an adolescent disorder and the likelihood of having individual adult disorders was increased in those with a history of an adolescent disorder. All homotypic transitions were statistically significant and greater than 3.0. All adult disorders were also predicted by at least three other adolescent disorders. Anxiety and depression cross-predicted one another, but both disorders were also predicted by adolescent behavioral disorders. SUDs were only preceded by behavioral disorders.

In adjusted models, predictors of young adult emotional disorders were attenuated. There continued to be evidence of cross-prediction between depression and anxiety, and early substance problems predicted later depression. Substance problems were predicted by behavioral disorders and early substance problems.

Discussion

This combined analysis of multiple “gold standard” longitudinal, epidemiologic samples strongly supports the continuity of psychiatric illness across developmental transitions. Having a disorder at an early period was associated with at least a 3-fold increase in odds for having a disorder at a later period. Both homotypic and heterotypic transitions are common from childhood to adolescence and from adolescence to early adulthood in bivariate models. This finding was the same looking forward and looking back: All disorders predicted multiple disorder categories *later* in development and all disorders were predicted by at least 3 disorder categories at the *prior* developmental period. After accounting for comorbidities, more specific homotypic patterns of prediction were supported for many disorders. These findings generally support conclusions from prior studies (see (Copeland et al., 2009b, Kim-Cohen et al., 2003)), but a few specific findings are noteworthy and diverge from prior findings.

First, although homotypic transitions were common, not all were of similar magnitude. The largest homotypic effects in adjusted models were seen for behavioral disorders from childhood to adolescence and substance disorders from adolescence to adulthood (ORs > 3.0). Even amongst behavioral disorders, however, the continuity of ADHD was striking (OR=17.4), suggesting that the developmental course of ADHD may be more stable than other common disorders. Homotypic transitions for emotional disorders such as depression and anxiety, although still generally displaying significant associations, tended to be more modest (OR < 3.0). This suggests a gradient of continuity and chronicity for common psychiatric disorders.

This study provided mixed support for the patterns of heterotypic associations summarized above. First, cross-prediction between anxiety and depression was strongly supported from

adolescence to adulthood which is the transition that is best studied. On the other hand, there was less support from *childhood to adolescence* and most such associations were attenuated in adjusted models. In fact, few disorders at all – homotypic or heterotypic - predicted emotional disorders in adolescence. This may not be unexpected for disorders which have their median age of onset in adolescence (Costello et al., 2003).

The literature suggests that behavioral disorders tend to precede adult anxiety and depression, but not vice versa. This finding received little support in the current study where no behavioral disorder predicted anxiety or depression in the adjusted models. At the same time, ODD displayed moderate odds ratios for predicting both later depression and anxiety that were often of the same magnitude as the cross-prediction from the other emotional disorder (see adolescent depression versus adolescent ODD predicting adult anxiety). Neither conduct disorder nor ADHD displayed any evidence of association with later emotional disorders. Together with the finding of only modest cross-prediction between emotional disorders, this suggests that the diagnostic histories of depression and anxiety are less predictable than is observed for other common disorders.

Finally, the transition risks from adolescent emotional disorder to young adult substance disorders were not statistically significant for the adolescent to adult transition. This is not consistent with previous published reports (e.g., (Zimmermann et al., 2003, Swendsen et al., 2010, Grant et al., 2008)). There are a number of considerations in making sense of this apparent contradiction. Our study aggregated separate disorders into single categories for each of anxiety and mood disorders, but the degree of association of various anxiety disorders with substance use may vary. In the National Epidemiologic Survey on Alcohol and Related Conditions (Grant et al., 2008), the anxiety disorders with the strongest association with later substance problems – Panic disorder and PTSD - are both rare in adolescence. In a similar manner, among the mood disorders, bipolar disorder has the strongest association with later substance disorders, but the frequency of this condition is also very low in epidemiologic samples. Even studies supporting this link have commonly found that such associations are modest as compared to prediction from early behavioral disorders (Swendsen et al., 2010). Altogether, this suggests that there may be an association between emotional disorders and later substance disorders, but that it is easier to detect in early adulthood with its increased rates of less common mood and anxiety disorders.

Caveats

All three samples were population-based and were carefully ascertained to insure representativeness and minimize selection biases. All samples assessed psychiatric functioning and concomitant psychosocial impairment with “gold standard” instruments at multiple points across development with limited attrition. Despite these strengths, the following caveats should be kept in mind when interpreting these findings.

The samples are not representative of all racial/ethnicity groups. Indigenous groups from New Zealand and the Unites States are represented as are participants of European ancestry. Individuals of African ancestry are generally underrepresented with respect to their prevalence within the US. However, prevalence values for childhood and adolescent psychiatric disorders derived from these samples are similar to rates from studies in other countries and/or with differing racial/ethnic admixture (Roberts et al., 1998).

This study covers three developmental periods and a 23 year period (age 9 to 32), but psychiatric disorders can be reliably diagnosed earlier in development (Egger and Angold, 2006). In a similar manner, these studies only followed participants into early adulthood but not into middle or older adulthood. This limitation is unavoidable because many of the DSM categories studied were only operationalized in the 1980s. However, cohort studies with

longer follow-up periods (40+ years) relying upon questionnaires assessing general internalizing and externalizing problems have suggested the presence of widespread psychiatric problems continuing into middle or older adulthood (Colman et al., 2007).

The goal of the study was to look at transitions for *common* psychiatric disorder or disorder categories. Rare disorders which are among the most debilitating (e.g., schizophrenia) were not included. This study also excluded subclinical phenomena that have been shown to predict later psychiatric problems (Copeland et al., 2011). The impact of these exclusions is that the estimates of transition risk are conservative and likely *underestimate* true risk. Finally, the low prevalence of some disorders in childhood contributed to wider confidence intervals in the transition estimates. Study-specific prevalences are available in prior publications (Copeland et al., 2009b)(Newman et al., 1996).

Implications

It has long been established that comorbidity is the norm with childhood and adult psychiatric disorders (Angold et al., 1999). This study suggests that transitions from one disorder to another across developmental periods are similarly promiscuous. If this is so, then it implies a common liability that extends across a range of common emotional and behavioral disorders. This common liability is likely related to common causes both in terms of genetic risk (Kendler et al., 2003) and environmental exposures (Copeland et al., 2010, Fergusson et al., 1996a, Copeland et al., 2007). Interventions and prevention programs that address sources of such common liability may have a greater public health impact than those focused on specific disorders. The most effective prevention programs for adult mental illness may involve early intervention to ameliorate childhood and adolescent distress, and increasing access to treatment in childhood and adolescence may be justified in its own right but also for its potential to prevent subsequent disorders.

The potential for effective treatment of psychiatric disorders at early ages to prevent disorders at later ages - the ‘treatment as prevention proposition’ - has been raised for more than a decade (Kandel et al., 1999, Merikangas and Avenevoli, 2000). The work of McGorry and colleagues demonstrates the value of the clinical staging model in which prospective identification of participants at high risk for psychosis allows for simple and safe intervention that are effective in terms of both remission and recovery (McGorry et al., 2009). In the case of common childhood disorders, the potential for this secondary benefit, however, is unknown, primarily because current intervention studies rarely measure secondary outcomes or follow participants across developmental transitions (Glantz et al., 2009). Only intervention research can test the treatment as prevention proposition, but our findings suggest the time is nigh for intervention research to take this step.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Comparison of assessment across three studies

		CHD			DMHDS			GSMS		
	Disorders	**Ages**	**Assessment**	**Period Assessed**	**Ages**	**Assessment**	**Period Assessed**	**Ages**	**Assessment**	**Period Assessed**
Childhood (ages 9–12)	Mood Anxiety Conduct ADHD ODD	n/a	n/a	n/a	11	DISC, RCSA, RCS-B, RBPC, SRED	12 months	9, 10, 11, 12	CAPA	3 months
Adolescence (ages 13–18)	Mood Anxiety Conduct ADHD ODD SUD	15, 16, 18	DISC, DIS SRED, RBPC, RAPI	12 months; STLI	13, 15, 18	DIS, DISC, RBPC	12 months	13, 14, 15, 16	CAPA	3 months
Early Adulthood (Ages 19–30)	Mood Anxiety SUD	21, 25, 30	CIDI	STLI	21, 26, 32	DIS	12 months	19, 21, 24/25/26	YAPA	3 months

ADHD= Attention-deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; SUD = Substance use Disorders; CAPA = Child and Adolescent Psychiatric Assessment; CIDI = Composite International Diagnostic Interview; DIS = Diagnostic Interview Schedule; DISC = Diagnostic Interview Schedule for Children; RAPI Survey = Rutgers Alcohol Problems Index Survey; RBPC = Revised Behavior Problems Checklist; RCS = Rutter Child Scale; SRED = Self-Report Early Delinquency; YAPA= Young Adult Psychiatric Assessment; STLI = since the last interview.

Table 2

Diagnostic transitions from childhood to adolescence from GSMS and DMHDS

	Adolescent dx, Unadjusted and adjusted ORs (95% CI)						
Childhood dx	**Depression**	**Anxiety**	**Conduct**	**ADHD**	**ODD**	**SUD**	**Any dx.**
Depression							
Unadjusted	3.33 (0.81–13.67)	3.56(0.68–18.68)≠	8.43 (2.03–35.01) *	11.47 (4.83–27.21) *	4.25 (1.99–9.08) *	3.61 (1.59–8.17) *	5.67 (2.51–12.78) *
Adjusted	2.22 (0.75 – 6.56)	2.04 (0.45 – 9.30)	3.99 (1.24 – 12.83) *	1.83 (0.60 – 5.55)	1.48 (0.58 – 3.82)	2.65 (1.12 – 6.30) *	2.71 (1.12 – 6.55) *
Anxiety							
Unadjusted	2.20 (1.07–4.54) *	3.18 (1.86–5.44) *	2.19 (1.32–3.63) *	4.99 (2.55–9.78) *	2.32 (1.09–4.97) *	1.21 (0.69–2.11)	2.40 (1.56–3.68) *
Adjusted	1.45 (0.75 – 2.84)	1.85 (0.99 – 3.44)	1.38 (0.44 – 4.39)	2.34 (0.86 – 6.36)	2.01 (0.30 – 13.44)	0.83 (0.45 – 1.54)	1.43 (0.80 – 2.56)
Conduct							
Unadjusted	1.69 (0.65– 4.40)	1.17 (0.47–2.91)	7.45 (2.81–19.75) *	10.09 (5.06–20.14) *	4.35 (2.27–8.34) *	2.56 (1.47–4.44) *	5.55 (2.50–12.32) *
Adjusted	0.91 (0.16 – 5.16)≠	0.58 (0.15 – 2.23)	4.70 (0.65 – 34.21)≠	4.13 (0.70 – 24.33)≠	2.23 (0.84 – 5.87)	1.96 (1.05 – 3.64) *	3.15 (1.53 – 6.52) *
ADHD							
Unadjusted	1.16 (0.43–3.12)	1.21 (0.71–2.07)	2.73 (0.42–17.62)≠	28.42 (15.4–52.42) *	5.23 (2.67–10.22) *	1.27 (0.33–4.89)≠	3.81 (2.23–6.49) *
Adjusted	0.66 (0.23 – 1.89)	0.62 (0.31 – 1.26)	1.04 (0.10 – 11.03)≠	17.40 (6.83 – 44.32) *	3.77 (1.44 – 9.90) *	0.77 (0.17 – 3.37)≠	1.92 (0.92 – 3.98)
ODD							
Unadjusted	2.43 (0.95–6.18)	2.33 (0.85–6.38)	3.68 (2.27–5.97) *	6.42 (3.22–12.80) *	6.16 (3.51–10.83) *	2.40 (1.43–4.04) *	4.36 (2.75–6.93) *
Adjusted	1.87 (0.56 – 6.24)	1.63 (0.35 – 7.52)	1.32 (0.66 – 2.67)	1.12 (0.27 – 4.60)	4.41 (1.93 – 10.05) *	1.67 (0.89 – 3.12)	2.16 (1.15 – 4.06) *
Any disorder							
Unadjusted	1.92 (1.05–3.49) *	2.42 (1.44–4.08) *	4.03 (2.83–5.74) *	14.12 (7.80–25.55) *	3.74 (2.28–6.14) *	1.71 (1.18–2.48) *	3.71 (2.65–5.19) *
Adjusted	--	--	--	--	--	--	--

Studies included: Great Smoky Mountain Study and Dunedin Multidisciplinary Health and Development Study (DSM categories). Adjusted analyses accounted for all childhood comorbidities. ADHD= Attention-deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; SUD = Substance use Disorders.

*Values significant at $p < 0.05$.

≠Significant between-study heterogeneity.

Table 3

Unadjusted diagnostic transitions from adolescence to early adulthood from CHDS, GSMS, and DMHDS

	Adult dx,Unadjusted and adjusted ORs (95% CI)			
Adolescence dx	**Depression**	**Anxiety**	**SUD**	**Any dx.**
Depression				
Unadjusted	**3.40 (2.76–4.19)** *	**3.6 (2.21–5.87)** ‡*	1.49 (0.92–2.40) ‡	**3.25 (1.85–5.69)** ‡*
Adjusted	**2.52 (1.65–3.83)** *	**2.11 (1.58–2.81)** *	1.13 (0.55–2.35) ‡	**2.31 (1.33–3.99)** *
Anxiety				
Unadjusted	**2.56 (1.78–3.68)** *	**3.95 (2.89–5.39)** *	1.05 (0.87–1.28)	**2.55 (1.49–4.35)** ‡*
Adjusted	**1.85 (1.11–3.09)** *	**2.81 (2.18–3.62)** *	0.69 (0.42–1.12)	1.70 (0.94–3.09)
Conduct				
Unadjusted	*1.80 (1.19–2.74)**	**1.90 (1.48–2.43)** *	**4.30 (2.53–7.32)** ‡*	**3.39 (2.42–4.75)** *
Adjusted	1.27 (0.91–1.79)	1.24 (0.90–1.72)	**2.37 (1.42–3.94)** *	**2.02 (1.30–3.15)** *
ADHD				
Unadjusted	1.24 (0.61–2.49)	**2.43 (1.65–3.57)** *	**2.88 (1.95–4.24)** *	**2.82 (1.67–4.76)** *
Adjusted	0.72 (0.36–1.43)	1.43 (0.69–2.98)	**1.99 (1.23–3.23)** *	1.46 (0.81–2.64)
ODD				
Unadjusted	**2.18 (1.24–3.85)** *	**3.18 (1.49–6.76)** ‡*	**2.21 (1.66–2.93)** *	**2.53 (1.62–3.95)** *
Adjusted	1.60 (0.99–2.60)	2.11 (0.94–4.71) ‡*	1.24 (0.86–1.78)	**1.52 (1.01–2.29)** *
SUD				
Unadjusted	**1.94 (1.43–2.62)** *	**1.69 (1.38–2.08)** *	**5.96 (4.30–8.27)** *	**4.39 (3.33–5.79)** *
Adjusted	**1.43 (1.10–1.86)** *	1.15 (0.88–1.51)	**4.53 (3.47–5.92)** *	**2.89 (2.09–3.99)** *
Any dx.				
Unadjusted	**2.68 (2.22–3.24)** *	**2.86 (2.36–3.47)** *	**2.47 (2.09–2.93)** *	**3.39 (2.83–4.06)** *
Adjusted	--	--	--	--

Studies included: Great Smoky Mountain Study, Christchurch Health and Development Study, and Dunedin Multidisciplinary Health and Development Study (DSM categories). Adjusted analyses accounted for all adolescent comorbidities. ADHD= Attention-deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; SUD = Substance use Disorders.

* Values significant at $p < 0.05$.

‡ Significant between-study heterogeneity.