Longitudinal dimensionality of adolescent psychopathology: Testing the differentiation hypothesis

Sonya K. Sterba[1], **William Copeland**[2], **Helen Link Egger**[2], **E. Jane Costello**[2], **Alaattin Erkanli**[2], and **Adrian Angold**[2]

[1]Department of Psychology, University of North Carolina at Chapel Hill

[2]Center for Developmental Epidemiology, Department of Psychiatry, Duke University

Abstract

Background—The differentiation hypothesis posits that the underlying liability distribution for psychopathology is of low dimensionality in young children, inflating diagnostic comorbidity rates, but increases in dimensionality with age. This hypothesis not been adequately tested with longitudinal psychiatric symptom data.

Methods—Confirmatory factor analyses of DSM-IV symptoms from seven common Axis I syndromes—major depression (MDD), generalized anxiety (GAD), separation anxiety (SAD), social anxiety (SOC), attention deficit hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiant disorder (ODD) were conducted longitudinally, from ages 9–16, in the general-population Great Smoky Mountains Study sample.

Results—An eight syndrome model fit well at all ages: CD, ODD, SAD, SOC, multidimensional ADHD (hyperactivity, impulsivity, and inattention) and unidimensional MDD/GAD. A high degree of measurement invariance was found for all syndromes except for MDD/GAD. MDD and GAD syndromes slightly diverged at age 14–16, when they also began to explain more symptom variance. Additionally, correlations between some emotional and disruptive syndromes showed slight differentiation.

Conclusions—Marked developmental differentiation of psychopathology, as implied by the orthogenetic principle, is not a prominent cause of preadolescent and adolescent psychiatric comorbidity.

Many competing explanations for child and adolescent psychiatric comorbidity (e.g. Caron & Rutter, 1991), have been empirically investigated (e.g. Angold, Costello, & Erkanli, 1999; Krueger & Markon, 2006), but one persisting explanation has not. This explanation contends on developmental-theoretical grounds that psychiatric disorders become differentiated from one another with advancing age, such that at younger ages the underlying liability distribution for psychopathology is of lower dimensionality (Lahey et al., 2004; Lilienfeld, Waldman & Israel, 1994; Patterson, 1993). Imposing age-invariant diagnostic cutpoints upon an underlying distribution of lower dimensionality induces artifactual psychiatric comorbidity. As articulated by Lilienfeld et al. (1994) this *differentiation hypothesis* states that:

> "children with comorbid syndromes may be at a stage in which the different developmental processes underlying these syndromes have yet to achieve full differentiation. A failure to appreciate the implications of the orthogenetic principle

Correspondence: Sonya Sterba, Psychology Department, UNC-CH, CB#3270, Chapel Hill, NC 27599-3270. ssterba@email.unc.edu..

may partially explain the particularly high rates of comorbidity among many childhood disorders" (p. 77).

Although the dimensionality of multiple common Diagnostic and Statistical Manual (DSM-IV) syndromes has been tested in cross-sectional, general population samples by factor analyzing DSM symptoms (see Table 1), this evidence cannot be used to adequately test the differentiation hypothesis for several reasons. First, comparisons of DSM syndrome differentiation have only been made between-children, across-age, not within-child, across age. Second, between-children, across-age comparisons are complicated by the fact that most studies report syndrome correlations only for markedly age-heterogeneous samples (e.g., 3–19). Third, although some studies have reported syndrome correlations for more homogeneous subgroups—preschoolers (2–5) or pre-adolescents and adolescents (11–19)—the latent syndrome correlations between-study, within-agegroup can be as large or larger than those within-study, across-agegroup, and, moreover, latent syndrome correlations are never accompanied by confidence intervals to facilitate across-study comparison.

Nevertheless, piecing together the available evidence (putting aside potential informant biases, based on results of Lahey et al., 2004; 2008) there appears, at first, to be some evidence of changing syndrome dimensionality between preschool-age and preadolescence, particularly among disruptive disorders. That is, unlike in preadolescents and adolescents, Sterba, Angold, and Egger (2007a) found oppositional defiant disorder (ODD) and conduct disorder (CD) to be unidimensional in preschoolers and Bauermeister (1992) found subfactors of attention deficit hyperactivity disorder (ADHD)—hyperactivity (H), impulsivity (IN), inattention (I)—to be unidimensional in preschoolers and Sterba et al. (2007a) found them nearly unidimensional (see Table 1). Additionally, among younger (4–10) but not older (11–17) children Lahey et al. (2004) found ODD and HI unidimensional, but differentiable from CD and IN (correlations only reported for age 4–17; see Table 1 footnote). However, such developmental changes in syndrome dimensionality actually represent only a trivial amount of differentiation; Table 1 shows that, among preadolescent-and/or adolescent-only samples, ODD and CD have been found related at most at r=.91, and HI and IN have been found related at most at r=.85, and HI and ODD have been found related at most r=.87. Even less evidence exists in Table 1 for developmental differentiation among emotional syndromes from preschool to preadolescence. Common emotional syndromes were either consistently differentiable across agegroups (separation anxiety disorder, SAD, and social phobia, SOC), or consistently undifferentiable across agegroups (major depression, MDD, and generalized anxiety disorder, GAD, except in Hartman et al., 2001). In fact, the only syndrome correlations in Table 1 which were consistently higher in preschoolers versus (pre)-adolescent or older samples by a clinically-meaningful margin (e.g. r .100 involved relations of emotional syndromes (mainly MDD, GAD) to disruptive syndromes (H, I, IN, CD, ODD).

However, these summary remarks are preliminary because they (a) do not account for within-study sampling variability in syndrome correlations, (b) use between-child syndrome differences to make inferences about within-child syndrome change, and (c) generalize across studies that vary considerably regarding specificity and comprehensiveness of symptom measurement and regarding accommodation of overlapping symptoms. Still, Table 1 shows that we know even less about whether syndrome differentiation continues across preadolescence to adolescence than we do about syndrome dimensionality change from preschool to preadolescence. No studies compared syndrome dimensionality across preadolescents, early adolescents, and later adolescents. Particularly relevant to DSM-V is whether GAD and MDD differentiate in adolescence (Moffitt et al., 2007; Greenberg, 2008), a time known to be critical for the emergence and organization of depression (Angold, 1988). This study overcomes these limitations in examining the dimensionality of common

Axis I syndromes (MDD, GAD, ODD, CD, ADHD, SAD, SOC) across age 9–16 in a longitudinal general-population probability sample, and provides the first inferential test of the differentiation hypothesis in this setting.

Methods

Participants

Data were drawn from the Great Smoky Mountains Study (GSMS). See Costello et al., (1996) for study details. A representative sample of 4,500 children, aged 9, 11, and 13, were drawn from a finite population of 12,000 in 11 western North Carolina counties using a household equal-probability accelerated-cohort design. Parent-reported behavioral problem screenings were obtained from 95% of this stage 1 sample. At stage 2, all American Indian youth were recruited (n=450), along with all screen-high children and 10% of screen-low children (total *N*=1420; 44% girls, 56% boys). Present analyses were based on data from when children were 9–16 (first eight annual waves of assessment). Sampling weights accounted for unequal probabilities of selection in all analyses. Re-weighted demographics indicated 89.4% of recruited participants were Caucasian, 6.9% were African American, and 3.7% were American Indian. An average response rate of 83% was maintained across waves included in these analyses (range 75–94%). Sampling weights were adjusted for nonresponse at wave 1. Estimation methods employed for binary indicators only accommodated pairwise deletion of missing data under MCAR; (multiple imputation is problematic for sparse binary data; Allison, 2007). Cohort differences were examined in Sterba et al., (2007b), but not found; present analyses do not control for cohort.

Measures

At each wave, the child and primary caregiver (usually mother) were separately interviewed using the Child and Adolescent Psychiatric Assessment (CAPA; Angold, et al., 1995). The CAPA is an interviewer-based interview which uses structured questioning to gather onset, intensity, frequency and duration information on symptoms described in an extensive glossary, across a 3-month reference period. Computerized algorithms determined whether symptoms meet endorsement criteria operationalized in the DSM-IV. Child and parent reports were combined using the standard `or' rule (Costello et al., 1996)—except in the case of ADHD, where standard practice dictated sole reliance on parent report (Angold et al., 1995). CAPA symptom intra-class correlations ranged from .50 (ODD) to .88 (MDD) (Angold et al., 1995).

Statistical Analysis

Modeling framework—Traditional methods for testing factor structure stability over time—longitudinal factor analysis—cannot simultaneously handle 7 factors (and 66 relatively-sparse binary items) at each of 8 measurement occasions (56 factors total). Instead, we estimated our multi-syndrome model at three condensed age blocks: ages 9–10, 11–13, and 14–16. (Alternative age groupings were tried but did not materially alter results; Sterba et al., 2007b). The number of observations per agegroup were: *N*=936 for age 9–10, *N*=2588 for age 11–13, *N*=3150 for age 14–16. Thus, within each agegroup's factor model, we have up to three observations nested within-child; this dependency is accounted for by adjusting standard error and chi square computations using TYPE=COMPLEX in Mplus 5.0. Fitting factor models separately for 9–10 year-olds, 11–13 year-olds, and 14–16 year-olds complicated comparison of factor loadings and factor correlations across agegroups. To illustrate our approach, consider the comparison of a single factor loading at age 9–10 versus age 11–13. Given the estimate and standard error of that particular factor loading at age 9–10, we used parametric bootstrapping (10,000 resamples) to generate its Monte Carlo sampling distribution at age 9–10, and similarly used parametric bootstrapping to generate

its Monte Carlo sampling distribution at age 11–13. From these two sampling distributions, we created a sampling distribution of the across-agegroup differences in that loading. The $100(\alpha/2)$th and $100(1 - \alpha/2)$th percentile values from that sorted bootstrap sampling distribution of differences served as the lower and upper bounds of a $100(1 - \alpha)$% confidence interval for the across-agegroup difference in that factor loading, which was used to test the null that the difference between the two loadings is 0 in the population. The same procedure was repeated for all factor loadings, for all three agegroup comparisons (i.e. 9–10 vs. 11–13; 11–13 vs. 14–16; 9–10 vs. 14–16). A similar procedure was used to compare factor correlations across agegroups, with the following caveats. Instead of simply using the estimated *r* and its SE to generate bootstrap resamples, first, Fisher's *r* to *z*′ transformation was first used to transform the estimated *r* to an approximately-normal metric, and, second, confidence bounds of the original correlation were transformed and used to derive its transformed SE. The transformed *r* and transformed SE were then used to generate bootstrap resamples, creating sampling distribution of the transformed *r;* the transformed *r*s were back-transformed to create sampling distributions of *r*s.

Model specification—All models contained all syndromes under investigation (ODD, CD, ADHD, MDD, SAD, GAD, SOC) so as not to incur omitted syndrome bias (Angold et al., 1999) in determining dimensionality. All DSM symptoms were allowed to load on their corresponding latent syndrome factor, with the exception of the following symptoms that were never endorsed: ADHD9 (forgetting), CD4 (human cruelty), CD6 (confrontational stealing), CD14 (runs away), at age 9–10; and ADHD9 (forgetting) at age 11–13. Within age-block, competing CFA models were inferentially compared to test for developmental changes in dimensionality of specific syndromes: MDD and GAD; ODD and CD; H, I, and IN; H and ODD, adjusting for all other disorders. Across age-block, final correlated-syndromes models were compared for evidence of across-time syndrome differentiation, particularly between emotional and disruptive syndromes.

Symptoms shared across syndromes can artificially inflate the magnitude of syndrome covariation (Angold et al., 1999; Lilienfeld et al., 1994). Hence, item-specific residuals of overlapping symptoms were allowed to correlate: (a) irritability (ODD, GAD, MDD) (b) too little/much sleep (GAD, MDD) (c) school refusal/absence (SAD, CD) and (d) lying/blaming (CD, ODD). The fatigue symptom for GAD and MDD was correlated >.95; to prevent collinearity, it was combined into a single, cross-loading indicator. In other cases, a *single* symptom from one DSM syndrome related to a *set* of symptoms from another syndrome. To capture this, that symptom was allowed to cross-load: the concentration symptoms of GAD and MDD cross loaded on Inattention and restless/keyed up symptom of GAD cross loaded Hyperactivity (following Hartman et al., 2001).

Model Estimation—Robust weighted least squares with tetrachoric correlation input and adjustments for nonnormality and nonindependence was used for estimation (WLSMV; M*plus* 5.0, Muthén & Muthén, 1998–2008). As a result of low endorsement rates, several symptoms belonging to the same disorder were parceled (summed) into one indicator to avoid estimation problems. At age 9–10, MDD 2,5,1 (anhedonia, psychomotor agitation/retardation, depressed/irritable), were parceled; ADHD 5,7 (unorganized, loses things) were parceled, and CD 5,7 (animal cruelty, forced sex) were parceled; at age 11–12, CD 4,5,6,7 (animal/human cruelty, confrontational stealing, forced sex) and CD 13,14 (breaks curfew, runs away) were parceled; at age 14–16 only CD 4,5,6,7 were parceled. Sensitivity analyses indicated that the 1–2 parcels/factor did not change dimensionality results.

Model evaluation—For assessing model fit, we used RMSEA (population misfit per degree of freedom; .05 indicates good fit) and CFI (fit relative to a null baseline; .95 indicates good fit) which are relatively insensitive to *N*(Yu, 2002). For model comparisons,

we used Robust $\Delta\chi^2$, which is sensitive to N, and we reran models with Robust-Maximum Likelihood to obtain BIC and sample size adjusted BIC, which penalize for model complexity (lower BIC is better).

Results

Within-age-block: Testing syndrome dimensionality

Dimensionality testing via model comparisons #1–#5 in Table 2 resulted in eight-factor final models (MDD/GAD, SAD, ODD, CD, SOC, Hyperactivity, Impulsivity, Inattention) with good fit at each age-block. For 9–10, RMSEA=.03 and CFI=.94; for 11–13, RMSEA=.02 and CFI=.98; for 14–16, RMSEA=.02 and CFI=.97. In model comparison #1, specifying MDD and GAD as separate factors resulted at age 9–10 and 11–13 in a linear dependence between MDD and GAD (r .00), indicating separate dimensions were not supported (Lahey et al., 2008 used similar procedures). BIC showed worse fit for separate GAD and MDD factors at 9–10, and essentially unchanged fit for separate GAD and MDD at 11–13. At age 14–16, the correlation between MDD and GAD was r=.90, which was statistically differentiable according to χ^2 and BIC. Despite some slight indication of a dimensionality change, from uni-to near-unidimensional, we retained GAD and MDD as unidimensional in final models. In model comparison #2, ODD and CD were found to be statistically distinct at all ages according to χ^2, after adjusting for other disorders, yet separating them sizably improved the BIC only at age 14–16. On balance, most of this mixed evidence supported ODD and CD as separate factors across-age. In model comparison #3, at all agegroups, χ^2 and BIC identified a significant decrement in fit from collapsing a trifactorial (I, H, IN) model for ADHD into a unifactorial model, and model comparison #4 identified a smaller, but significant, decrement from collapsing a trifactorial model to a bifactorial (HI, IN) model. A trifactorial ADHD specification was retained for all age groups. Finally, model comparison #5 indicated that ODD and H were always statistically distinct according to χ^2 and BIC.

Across-age-block: Testing the differentiation hypothesis

Across-age invariance testing involved first testing for configural invariance, then metric invariance, then factor correlation invariance. (Note that symptom thresholds cannot be tested for across-age invariance with binary data.) Table 3 shows that configural invariance (same pattern of significant and non-significant loadings across age) was partially met for factor loadings in final models. All but three symptoms showed positive, significant loadings on their designated DSM syndromes at each agegroup. The three age-variant symptoms were those that were allowed to cross-load on multiple factors to prevent artifactual inflation of factor correlation estimates. The primary loading of MDD8 (concentration) was age-invariant, but its secondary loading was not, whereas, the secondary loadings of GAD1 (restlessness) and GAD3 (concentration) were age-invariant, but their primary loadings were not. Table 3 also shows that the more stringent metric invariance (same loading magnitude across age) was partially met for factor loadings in final models. Most of the across-age loading differences were found for MDD/GAD, where some loadings increased at age 14–16. No MDD/GAD loadings significantly changed magnitude between 9–10 and 11–13. This finding is also reflected in Table 4, where the average proportion of variance in DSM symptoms explained by their designated DSM syndromes remained predominantly stable across-ages for most syndromes, but increases at age 14–16 for MDD/ GAD. Finally, Table 5 shows that most factor correlations were not significantly different across-agegroups, and factor correlations had sizable sampling variability (large 95% CIs). Disruptive syndromes were most highly correlated with each other across-age, as were emotional syndromes—with the exception of MDD/GAD, which sometimes associated more strongly with disruptive syndromes. When factor correlations changed significantly

across-agegroups, it almost always happened in early adolescence (i.e. 9–10 vs. 11–13 or 9–10 vs. 14–16, but not 11–13 vs. 14–16), involved instable, low correlations between emotional and disruptive syndromes, and did not represent a consistent pattern of differentiation. For example, whereas SOC and MDD/GAD became less correlated with CD by age 14–16, SAD became more correlated with I, H, IN and ODD by age 14–16.

Discussion

This study represents the first inferential test of the *differentiation hypothesis* across the transition to adolescence using diagnostic-interview symptom data from multiple syndromes in a general-population sample.

Overall, primarily the same number of dimensions (eight) was identified as well-fitting for all age-groups: H, IN, I, ODD, CD, SAD, SOC, and MDD/GAD. This multi-syndrome longitudinal factor structure displayed a high degree of measurement invariance: nearly the same factor loading pattern and factor loading magnitude was found across-age, except for MDD/GAD. That is, areas of suspected dimensionality change from preschool to preadolescence (dimensionality of ODD and CD, of H, I, and IN, and of H and ODD; Bauermeister, 1992; Lahey et al., 2004; Sterba et al., 2007b) showed stable dimensionality from pre-adolescence to later adolescence (Table 2). But areas of suspected dimensionality stability from preschool to preadolescence (dimensionality of MDD and GAD; Lahey et al., 2004, 2008; Sterba et al., 2007b) showed indications of slight differentiation from pre-adolescence to later adolescence (Table 2). Other indications of this small developmental shift or reorganization of MDD/GAD starting at age 14–16 were increases in magnitude of some MDD and GAD symptom loadings at age 14–16 (Table 3), and increases in proportions of symptom variance accounted for by the MDD/GAD syndrome at age 14–16 (Table 4). Relatedly, some MDD/GAD symptom reorganization between 4–10 vs. 11–17 years was found by Lahey et al., (2004). Yet treating MDD/GAD as unidimensional at age 14–16 still resulted in good model fit.

Moreover, across-age correlations among these eight putative dimensions displayed no consistent pattern of developmental differentiation. For example, whereas some emotional syndromes (SOC) became significantly more distinct from disruptive syndromes, others (SAD) became significantly *less* distinct from disruptive syndromes. These heterotypic correlations could temporarily (for one or two agegroups) be very small ($< r = .10$)—lower than found in prior studies (see Table 1)—but their 95% CIs usually included values found in prior studies. The only pattern conceivably interpretable as developmental differentiation, and in line with prior findings in Table 1, was the consistently decreasing correlations between MDD/GAD and CD, H, I, and IN (but not ODD); however, these trends were not statistically significant. Overall, correlations among disruptive syndromes showed greater stability, and correlations between disruptive and emotional syndromes showed less stability.

Future CFA studies are needed that compare the dimensionality of MDD and GAD in adulthood to MDD and GAD in adolescence to (a) clarify whether MDD and GAD indeed remain at- or near-unidimensional into adulthood, and, (b) clarify whether the GAD construct displays longitudinal coherence, in light of our inconsistent loadings for two GAD symptoms (the same inconsistency as was found in Hartman et al., 2001). An essentially-unidimensional MDD/GAD over time, or an unstable-dimensionality of MDD/GAD over time has important implications for comorbidity models presently being used to inform DSM-V (Watson, 2005). These comorbidity models use threshold DSM diagnoses as indicators of higher-order "core psychopathological constructs." MDD and GAD diagnoses are treated as separate indicators, without first establishing distinctness of their liability

distributions, and higher-order "anxious/misery" or "distress" factors are included to explain their covariation (e.g. Vollebergh et al., 2001). If MDD and GAD are indeed unidimensional, such models are misspecified and higher-order anxious/misery or distress factors simply compensate for this misspecification.

Limitations

Several limitations of this study deserve mention. First, testing competing statistical models is only one way of examining the longitudinal internal validity of DSM nosology. Second, barring estimation difficulties with sparse, binary symptom data, testing multi-syndrome models at all ages simultaneously would have allowed us to quantify decrements in model fit associated with imposing age-invariance constraints. Third, symptom sparseness even under the combined-informant `or' rule prohibited splitting analyses by informant, however, Lahey et al. (2004; 2008) found no important informant (parent versus child) invariance for similar multi-syndrome models. Fourth, across-gender and across-race comparisons of syndrome loading and covariation patterns did not yield marked or consistent differences and so were not presented here.

Conclusions

The kind of marked developmental changes in the structure of psychopathology, from an undifferentiated "mass" to distinct DSM dimensions, as predicted by the orthogenetic principle (Lilienfeld et al., 1994), were not supported. Some mild differentiation may occur early—by preadolescence—among disruptive syndromes, and some mild differentiation may occur during adolescence, if at all, for GAD and MDD and between some emotional and disruptive syndromes.

References

Allison, PD. Multiple imputation of categorical variables under the multivariate normal model. Paper presented at the annual meeting of the American Sociological Association; Montreal, Canada. Aug. 2006

Angold A. Childhood and adolescent depression: I. Epidemiological and aetiological aspects. British Journal of Psychiatry. 1988; 152:601–617. [PubMed: 3048525]

Angold A, Costello EJ. The Child and Adolescent Psychiatric Assessment (CAPA). Journal of the American Academy of Child and Adolescent Psychiatry. 2000; 39:39–48. [PubMed: 10638066]

Angold A, Prendergast M, Cox A, Harrington R, Simonoff E, Rutter M. The Child and Adolescent Psychiatric Assessment (CAPA). Psychological Medicine. 1995; 25:739–753. [PubMed: 7480451]

Angold A, Costello EJ. A test–retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment. Psychological Medicine. 1995; 25:755–762. [PubMed: 7480452]

Angold A, Costello J, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry. 1999; 40:57–87. [PubMed: 10102726]

Burns GL, Boe B, Walsh JA, Sommers-Flanagan R, Teegarden LA. A confirmatory factor analysis of DSM-IV ADHD and ODD symptoms: What is the best model for the organization of these symptoms? Journal of Abnormal Child Psychology. 2001; 29:339–349. [PubMed: 11523839]

Burns GL, Walsh JA, Owens SM, Snell J. Internal validity of Attention Deficit Hyperactivity Disorder, and overt Conduct Disorder symptoms in young children: Implications from teacher ratings for a dimensional approach to symptom validity. Journal of Clinical Child Psychology. 1997a; 26:266–275. [PubMed: 9292384]

Burns G, Walsh J, Patterson D, Holte C, Sommers-Flanagan R, Parker C. Internal validity of the disruptive behavior disorder symptoms: Implications from parent ratings for a dimensional approach to symptom validity. Journal of Abnormal Child Psychology. 1997b; 25:307–319. [PubMed: 9304447]

Caron C, Rutter M. Comorbidity in child psychopathology: concepts, issues and research strategies. Journal of Child Psychology and Psychiatry. 1991; 32:1063–1080. [PubMed: 1787137]

Costello EJ, Angold A, Burns BJ, Stangl DK, Tweed DL, Erkanli A, Worthman CM. The Great Smoky Mountains Study of Youth: Goals, design, methods, and the prevalence of DSM-III-R disorders. Archives of General Psychiatry. 1996; 53:1129–1136. [PubMed: 8956679]

Goldberg D. Towards DSM-V: The relationship between generalized anxiety disorder and major depression episode. Psychological Medicine. in press.

Hartman CA, Mellenbergh G, Boyle MH, Offord DR, Racine Y, McNamee J, et al. DSM-IV internal construct validity: When a taxonomy meets data. Journal of Child Psychology and Psychiatry. 2001; 42:817–836. [PubMed: 11583254]

Krueger RF, Markon KE. Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. Annual Review of Clinical Psychology. 2006; 3:111–133.

Lahey B, Applegate B, Waldman I, Loft J, Hankin B, Rick J. The structure of child and adolescent psychopathology: generating new hypotheses. Journal of Abnormal Psychology. 2004; 113:358–385. [PubMed: 15311983]

Lahey BB, Rathouz PJ, Hulle CV, Urbano RC, Krueger RF, Applegate B, Garriock HA, Chapman DA, Waldman ID. Testing structural models of DSM-IV symptoms of common forms of child and adolescent psychopathology. Journal of Abnormal Child Psychology. 2008; 36:187–206. [PubMed: 17912624]

Lilienfeld S, Waldman I, Israel A. A critical examination of the use of the term and concept of *comorbidity* in psychopathology research. Clinical Psychology: Science and Practice. 1994; 1:71–83.

Moffitt TE, Harrington H, Caspi A, Kim-Cohen J, Goldberg D, Gregory AM, Poulton R. Depression and Generalized Anxiety Disorder: Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. Archives of General Psychiatry. 2007; 64:651–660. [PubMed: 17548747]

Molina BSG, Smith BH, Pelham WE. Factor structure and criterion validity of secondary school teacher ratings of ADHD and ODD. Journal of Abnormal Child Psychology. 2001; 29

Muthén, BO. Mplus Technical Appendices. Múthen & Múthen; Los Angeles, CA: 1998–2004.

Muthén, LK.; Muthén, BO. Mplus User's Guide. Third Edition. Múthen & Múthen; Los Angeles, CA: 1998–2008.

Patterson GR. Orderly change in a stable world: The antisocial trait as a chimera. Journal of Consulting and Clinical Psychology. 1993; 61:911–919. [PubMed: 8113492]

Sterba S, Egger HL, Angold A. Diagnostic specificity and nonspecificity in the dimensions of preschool psychopathology. Journal of Child Psychology and Psychiatry. 2007a; 45:1005–1013. [PubMed: 17915001]

Sterba, S.; Egger, HL.; Angold, A. Parallel dimensions of psychopathology in preschoolers and older children. Poster presented at the 13th annual meeting of the International Society for Research on Child and Adolescent Psychopathology; London, England. 2007b.

Vollebergh W, Iedema J, Bijl R, deGraaf R, Smit F, Ormel J. The structure and stability of common mental disorders. Archives of General Psychiatry. 2001; 58:597–603. [PubMed: 11386990]

Watson D. Rethinking the mood and anxiety disorders: A quantitative-hierarchical model for DSM-V. Journal of Abnormal Psychology. 2005; 114:522–536. [PubMed: 16351375]

Yu, C. Unpublished Dissertation, UCLA. 2002. Evaluation of model fit indices for latent variable models with categorical and continuous outcomes.

Table 1

Factor correlations from published multi-syndrome factor analyses of DSM symptoms in non-referred youth

	Heterogeneous Agegroup							Preschool-Age			Pre-adolescent/Adolescent-Age			
Study	1	9	2	3	4	5	6	7	5	8	9	3	6	10
Ages	4–17	6–11	3–19	2–10	4–15	6–13	3–10	2–5	4–5	2.5–6.5	12–17	11–19	11–16	11–16
Model	EFA	CFA	CFA	CFA	CFA	EFA	CFA	CFA	EFA	CFA	CFA	CFA	CFA	CFA
Informant	P	P	P,T	P	T	T	P	P	T	P	P	P	P	T
Measurement	SDI	SDI	C	C	C	C	C	SDI	C	C	SDI	C	C	C
I with H	1.00	1.00	1.00	1.00	.78	1.00	.85	1.00	1.00		1.00	1.00	.89	1.00
I with IN	.67	.78	.69	.86	.78	.57	.66	.94	1.00		.73	.76	.69	.85
I with ODD	1.0	.82	.70	.72	.80	.56	.72	.80	.56		.76	.72	.82	.87
I with CD	.61	.68	.59	.60	.63			.80			.70	.71		
I with SAD	.45	.42						.48			.50			
I with MDD	.59	.65	.18					.89			.60			
I with GAD	.59	.65	.23					.89			.60			
I with SOC	.52	.28						.41			.33			
H with IN	.67	.78	.69	.86	.78	.57	.68	.94	1.00		.73	.76	.75	.85
H with ODD	1.00†	.82	.70	.72	.80	.56	.66	.80	.56		.76	.72	.78	.87
H with CD	.61	.68	.59	.60	.63			.80			.70	.71		
H with SAD	.45	.42						.48			.50			
H with MDD	.59	.65						.89			.60			
H with GAD	.59	.65						.89			.60			
H with SOC	.52	.28						.41			.33			
IN with ODD	.67	.72	.52	.66	.66	.42	.68	.71	.56		.72	.70	.69	.77
IN with CD	.46	.63	.44	.46	.53			.71			.63	.52		
IN with SAD	.26	.40						.41			.39			
IN with MDD	.61	.70	.52					.86			.63			
IN with GAD	.61	.70	.23					.86			.63			
IN with SOC	.56	.40						.46			.37			
ODD with CD	.61	.89	.80	.62	.81			1.00			.91	.68		
ODD with SAD	.45	.43						.59			.44			

Study	Heterogeneous Agegroup							Preschool-Age			Pre-adolescent/Adolescent-Age			
	1	9	2	3	4	5	6	7	5	8	9	3	6	10
ODD with MDD	.59	.68	.43					.80			.70			
ODD with GAD	.59	.68	.30					.80			.70			
ODD with SOC	.52	.39						.59			.40			
CD with SAD	.22	.35						.59			.43			
CD with MDD	.48	.56						.80			.56			
CD with GAD	.48	.56	.18					.80			.56			
CD with SOC	.33	.27	.33					.59			.32			
SAD with MDD	.33	.73						.72			.68			
SAD with GAD	.33	.73						.72		.87	.68			
SAD with SOC	.47	.57						.65		.57	.57			
MDD with GAD	1.00	1.00	.73					1.00			1.00			
MDD with SOC	.63	.73						.76			.67			
GAD with SOC	.63	.73						.76		.55	.67			

Notes: If two factors are unidimensional, their correlations with other factors are the same (duplicated here).

Studies: 1= Lahey et al., 2004; 2= Hartman et al., 2001; 3= Burns et al, 1997b; 4= Burns et al., 1997a; 5= Bauermeister, 1992; 6= Burns et al., 2001; 7= Sterba et al., 2007a; 8=Spence et al., 2001; 9=Lahey et al., 2008; 10= Molina et al., 2001.

P=parent; T=teacher. SDI= structured diagnostic interview; C=checklist.

[†] only found at age 4–10, Not 11–17, but factor correlations not reported by agegroup.

Table 2

Nested model comparisons for syndrome dimensionality testing

Model Comparison	Less vs. more restrictive[2]	Age 9–10		Age 11–13		Age 14–16	
		$\Delta \chi^2$ (df)[1]	ΔBIC[3]	$\Delta \chi^2$ (df)[1]	ΔBIC[3]	$\Delta \chi^2$ (df)[1]	ΔBIC[3]
#1	(A) vs. Final	--	Δ +9	--	Δ −9	Δ 8.84 (1)[**]	Δ −218
#2	Final vs. (D)	Δ 10.94 (1)[**]	Δ −18	Δ 21.99 (1)[***]	Δ −171	Δ 70.39 (1)[***]	Δ −624
#3	Final vs. (B)	Δ 26.62 (2)[***]	Δ −1213	Δ 77.71 (3)[***]	Δ −3908	Δ 81.26 (3)[***]	Δ −2306
#4	Final vs. (C)	Δ 22.50(1)[***]	Δ −272	Δ 32.71 (1)[***]	Δ −1491	Δ 30.27 (1)[***]	Δ −873
#5	Final vs. (E)	Δ 40.20 (1)[***]	Δ −239	Δ 62.84 (1)[***]	Δ −1163	Δ 100.72 (1)[***]	Δ −2033
	Final model	**109.35 (53)** [***]	**−26469**	**169.88 (79)[****]**	**−147970**	**161.98 (78)** [*]	**−267459**

Notes:

-- could not be estimated.

Model A = MDD + GAD + SAD + SOC + ODD + CD + H + I + IN

Model B = MDD/GAD + SAD + SOC + ODD + CD +ADHD

Model C = MDD/GAD + SAD + SOC + ODD + CD + H/I + IN

Model D = MDD/GAD + SAD + SOC + ODD/CD + H + I + IN

Model E = MDD/GAD + SAD + SOC + ODD/H + CD + I + IN

Final model = MDD/GAD + SAD + SOC + ODD + CD + H + I + IN

[***]
p<.001;

[**]
p<.01;

[*]
p<.05;

[1]Degrees of freedom for robust chi square tests of absolute fit and difference tests are *not* determined directly from the model specification, but estimated (Satterthwaite-type) as described in Muthén (1998–2004; equation 110).

[2]The more restrictive model is supported if the chi square difference does not increase appreciably from the less- to more-restrictive model.

[3]Same pattern obtained with sample size adjusted BIC.

Table 3

Standardized factor loadings from age 9–10, 11–13, and 14–16 models

	Age 9–10		Age 11–13		Age 14–16	
	Estimate	*(S.E.)*	**Estimate**	*(S.E.)*	**Estimate**	*(S.E.)*
Inattention						
Careless_mistakes	0.91	*(0.03)*	0.93	*(0.01)*	0.95	*(0.02)*
Sustaining_attention	0.89	*(0.03)*	0.94	*(0.02)*	0.93	*(0.02)*
Listening	0.97	*(0.02)*	0.92[11/14]	*(0.02)*	0.98	*(0.01)*
Following_through	0.95	*(0.02)*	0.94	*(0.01)*	0.92	*(0.02)*
Organizing	0.76[a9/11]	*(0.06)*	0.95	*(0.04)*	0.85	*(0.06)*
Sustaining_tasks	0.98	*(0.03)*	0.98	*(0.02)*	0.95	*(0.02)*
Loses_things	0.76[a]	*(0.06)*	0.85	*(0.03)*	0.88	*(0.03)*
Easily_distracted	0.95	*(0.02)*	0.94	*(0.02)*	0.94	*(0.02)*
Forgetful					0.99	*(0.03)*
GAD3 concentrating	0.95	*(0.05)*	1.05[11/14]	*(0.03)*	0.86	*(0.03)*
MDD8 concentrating	0.26	*(0.13)*	0.52[11/14]	*(0.06)*	0.15[†]	*(0.10)*
Hyperactivity						
Fidgets	0.87	*(0.04)*	0.88	*(0.02)*	0.90	*(0.03)*
Leaves_seat	0.93	*(0.03)*	0.94	*(0.02)*	0.87	*(0.05)*
Runs/climbs	0.93	*(0.03)*	0.95	*(0.02)*	0.95	*(0.03)*
Quiet_activities	0.96	*(0.02)*	0.99	*(0.01)*	0.96	*(0.03)*
On_the_go	0.95	*(0.04)*	0.99	*(0.01)*	0.96	*(0.02)*
Talks_excessively	0.95	*(0.02)*	0.96	*(0.01)*	0.97	*(0.02)*
GAD1 restlessness	0.79	*(0.07)*	0.64[11/14]	*(0.04)*	0.33[9/14]	*(0.06)*
Impulsivity						
Blurts_answers	0.96	*(0.02)*	0.96	*(0.01)*	0.99	*(0.02)*
Awaiting_turn	0.97	*(0.03)*	0.96[11/14]	*(0.01)*	0.86[9/14]	*(0.04)*
Interrupts	0.94	*(0.02)*	0.99	*(0.02)*	0.99	*(0.03)*
Conduct						
Bullies	0.81	*(0.07)*	0.69	*(0.11)*	0.73	*(0.06)*
Initiates_fights	0.50	*(0.08)*	0.60	*(0.06)*	0.67	*(0.07)*
Used_weapon	0.64	*(0.09)*	0.40[11/14]	*(0.1)*	0.78	*(0.06)*
Fire_setting	0.43	*(0.1)*	0.57	*(0.07)*	0.60	*(0.07)*
Property_destruction	0.57	*(0.11)*	0.76[11/14]	*(0.08)*	0.54	*(0.07)*
Breaks_in	0.87[9/11]	*(0.07)*	0.59	*(0.06)*	0.61[9/14]	*(0.07)*
Lies/cons	0.58[9/11]	*(0.08)*	0.79	*(0.06)*	0.80[9/14]	*(0.05)*
Steals_w/o_confronting	0.78	*(0.06)*	0.79	*(0.05)*	0.69	*(0.05)*
Breaks_curfew	0.41	*(0.16)*	0.32[b]	*(0.09)*	0.49	*(0.09)*

	Age 9–10		Age 11–13		Age 14–16	
	Estimate	*(S.E.)*	**Estimate**	*(S.E.)*	**Estimate**	*(S.E.)*
Runs_away			0.32^{b}[11/14]	*(0.09)*	0.67	*(0.09)*
Truant	0.42	*(0.12)*	0.63	*(0.11)*	0.46	*(0.1)*
CD4/5/6/7 parcel	0.45	*(0.13)*	0.53	*(0.09)*	0.71	*(0.08)*
Oppositional Defiant						
Loses_temper	0.39[9/11]	*(0.08)*	0.63	*(0.05)*	0.70[9/14]	*(0.04)*
Argues	0.60	*(0.08)*	0.64	*(0.05)*	0.73	*(0.04)*
Actively_defies	0.74	*(0.06)*	0.82	*(0.04)*	0.77	*(0.04)*
Deliberately_annoys	0.70	*(0.08)*	0.70	*(0.05)*	0.68	*(0.05)*
Blames_others	0.58	*(0.06)*	0.60	*(0.05)*	0.60	*(0.04)*
Touchy/annoyed	0.43	*(0.09)*	0.42[11/14]	*(0.07)*	0.64[9/14]	*(0.05)*
Angry/resentful	0.46	*(0.07)*	0.61[11/14]	*(0.04)*	0.77[9/14]	*(0.03)*
Spiteful/vindictive	0.52	*(0.08)*	0.64	*(0.05)*	0.63	*(0.05)*
Separation Anxiety						
Anticipatory_distress	0.83	*(0.06)*	0.88	*(0.07)*	0.81	*(0.07)*
Worry_loss	0.82	*(0.07)*	0.65	*(0.07)*	0.61	*(0.08)*
Worry_untoward_event	0.78	*(0.07)*	0.78	*(0.12)*	0.99[9/14]	*(0.07)*
School_refusal	0.85	*(0.1)*	0.67	*(0.07)*	0.68	*(0.08)*
Fearful_alone	0.71	*(0.12)*	0.66[11/14]	*(0.1)*	0.92	*(0.05)*
Sleep_alone	0.48	*(0.09)*	0.66	*(0.08)*	0.68	*(0.1)*
Separation_nightmares	0.81[9/11]	*(0.07)*	0.52	*(0.13)*	0.71	*(0.12)*
Somatic_complaints	0.82	*(0.06)*	0.76	*(0.09)*	0.63	*(0.09)*
Depression/Gen. Anxiety						
Restlessness	$-0.02^{\dagger}$	*(0.1)*	0.16[11/14]	*(0.06)*	0.71[9/14]	*(0.05)*
Concentrating	$-0.06^{\dagger}$	*(0.09)*	-0.22[11/14]	*(0.06)*	$0.11^{\dagger}$	*(0.06)*
Irritability	0.67	*(0.14)*	0.55[11/14]	*(0.09)*	0.77	*(0.05)*
Muscle_tension	0.40	*(0.14)*	0.52[11/14]	*(0.09)*	0.87[9/14]	*(0.04)*
Sleep_disturbance	0.57	*(0.1)*	0.52	*(0.06)*	0.55	*(0.05)*
Depressed_mood	0.51^{c}	*(0.16)*	0.73	*(0.08)*	0.86[9/14]	*(0.04)*
Anhedonia	0.51^{c}	*(0.16)*	0.65	*(0.17)*	0.70	*(0.11)*
Weight_change	0.33	*(0.09)*	0.27	*(0.06)*	0.40	*(0.05)*
Insomnia/hypersomnia	0.49	*(0.11)*	0.42	*(0.1)*	0.64	*(0.05)*
Psychomotor_agit./retard.	0.51^{c}	*(0.16)*	0.73	*(0.12)*	0.92[9/14]	*(0.07)*
Guilt/worthlessness	0.62	*(0.1)*	0.73	*(0.07)*	0.82	*(0.04)*
Think/decide/concentrate	0.42	*(0.14)*	0.30[11/14]	*(0.12)*	0.70	*(0.09)*
Suicidal_ideation	0.45	*(0.09)*	0.41[11/14]	*(0.08)*	0.78[9/14]	*(0.04)*
Fatigue parcel	0.55	*(0.13)*	0.44[11/14]	*(0.09)*	0.70	*(0.05)*
Social Phobia						

	Age 9–10		Age 11–13		Age 14–16	
	Estimate	*(S.E.)*	**Estimate**	*(S.E.)*	**Estimate**	*(S.E.)*
Fear_social/performance	0.75	*(0.14)*	0.76	*(0.13)*	0.99	*(0.09)*
Exposure_anxiety	0.96	*(0.17)*	0.98	*(0.15)*	0.85	*(0.08)*

Notes.

In each case, the parcel loading is reproduced in this table for each of the constituent symptoms. Items that were parceled at all age blocks are labeled in the column on the right. A loading significantly different from age 9–10 vs. 11–13 is denoted [9/11] for alpha=.05 and **[9/11]** for alpha=.01. A loading significantly different from age 11–13 to 14–16 at is denoted [11/14] at alpha=.05 and **[11/14]** for alpha=.01. A loading significantly different from age 9–10 to 14–16 is denoted [9/14] at alpha of .05 and **[9/14]** at alpha=.01.

[a] These two items parceled at age 9–10.

[b] These two items parceled at age 11–13.

[c] These three items parceled at age 9–10.

[†] Loading *not* significantly different than 0 at alpha=.05.

Table 4

Average proportion of variance in DSM symptoms explained by their designated DSM syndrome

	Age 9–10	Age 11–13	Age 14–16
Inattention	0.870	0.844	0.868
Hyperactivity	0.905	0.868	0.876
Impulsivity	0.937	0.914	0.894
MDD/GAD	0.382	0.353	0.587
SAD	0.496	0.593	0.584
CD	0.393	0.388	0.427
ODD	0.408	0.319	0.478
SOC	0.769	0.744	0.852

Table 5

Correlations among latent DSM syndromes in the final age 9–10, 11–13, and 14–16 models.

	Factor Correlations			Age Differences			Correlation 95% CIs		
	Age 9–10	Age 11–13	Age 14–16	9–10 vs. 11–13	11–13 vs. 14–16	9–10 vs. 14–16	Age 9–10	Age 11–13	Age 14–16
I with H	.83*	.84*	.83*				(.71, .90)	(.76, .90)	(.74, .89)
I with IN	.84*	.83*	.90*				(.69, .92)	(.72, .91)	(.81, .93)
I with ODD	.65*	.65*	.63*				(.42, .80)	(.54, .75)	(.50, .73)
I with CD	.66*	.48*	.40*				(.41, .81)	(.32, .63)	(.27, .52)
I with SAD	.10	.28*	.43*			X	(−.10, .29)	(.12, .43)	(.27, .56)
I with MDD/GAD	.49*	.39*	.26*				(.30, .64)	(.22, .53)	(.14, .39)
I with SOC	.27*	.04	.22*				(.01, .49)	(−.14, .21)	(0, .41)
H with IN	.82*	.88*	.87*				(.68, .91)	(.83, .92)	(.82, .91)
H with ODD	.60*	.63*	.52*				(.44, .72)	(.51, .72)	(.41, .61)
H with CD	.50*	.49*	.40*				(.34, .62)	(.34, .62)	(.25, .53)
H with SAD	.11	.38*	.55*	X		X	(−.06, .28)	(.25, .49)	(.34, .70)
H with MDD/GAD	.43*	.43*	.29*				(.22, .60)	(.31, .54)	(.15, .42)
H with SOC	.24*	.20*	.22*				(.02, .44)	(.03, .35)	(.02, .41)
IN with ODD	.61*	.50*	.60*				(.45, .73)	(.39, .60)	(.51, .68)
IN with CD	.60*	.46*	.41*				(.41, .74)	(.31, .58)	(.29, .52)
IN with SAD	.09	.30*	.31*	X		X	(−.07, .25)	(.18, .40)	(.16, .45)
IN with MDD/GAD	.48*	.44*	.30*				(.28, .65)	(.31, .55)	(.18, .41)
IN with SOC	.27*	.11	.12				(.01, .50)	(−.05, .27)	(−.05, .29)
ODD with CD	.82*	.81*	.70*				(.59, .93)	(.70, .88)	(.61, .77)
ODD with SAD	.05	.27*	.35*	X		X	(−.14, .23)	(.14, .39)	(.17, .51)
ODD with MDD/GAD	.54*	.63*	.57*				(.32, .70)	(.49, .74)	(.45, .66)
ODD with SOC	.27	.10	.08				(−.07, .55)	(−.08, .27)	(−.07, .23)
CD with SAD	.41*	.27*	.40*				(.19, .60)	(.12, .41)	(.20, .56)
CD with MDD/GAD	.71*	.52*	.37*			X	(.43, .87)	(.34, .67)	(.25, .47)
CD with SOC	.47*	.13	−.03	X		X	(.24, .65)	(−.03, .29)	(−.24, .18)
SAD with MDD/GAD	.58*	.63*	.68*				(.41, .71)	(.49, .74)	(.47, .81)
SAD with SOC	.23	.38*	.44*				(−.03, .46)	(.19, .54)	(.15, .66)
MDD/GAD with SOC	.41*	.30*	.56*		X		(.06, .67)	(.07, .49)	(.38, .70)