THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
Journal of Child Psychology and Psychiatry 51:8 (2010), pp 871–884 doi:10.1111/j.1469-7610.2010.02234.x

Longitudinal dimensionality of adolescent psychopathology: testing the differentiation hypothesis

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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 as latent syndromes become less correlated. This hypothesis has 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, generalized anxiety, separation anxiety, social anxiety, attention deficient hyperactivity, conduct, and oppositional defiant disorders – were conducted longitudinally, from ages 9 to 16, using the general-population Great Smoky Mountains Study sample. **Results:** An eight-syndrome model fit well at all ages, and in both genders. It included social anxiety, separation anxiety, oppositional defiant, and conduct syndromes, along with a multidimensional attention deficit-hyperactivity syndrome (i.e., inattention, hyperactivity, and impulsivity) and a unidimensional major depression/generalized anxiety syndrome. A high degree of measurement invariance across age was found for all syndromes, except for major depression/generalized anxiety. Major depression and generalized anxiety 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. **Keywords:** Comorbidity, *Diagnostic and Statistical Manual*, factor analysis, longitudinal, dimensionality, development, internal validity, adolescent, Axis I psychopathology.

The concept of *differentiation*, one of four original laws of embryology (Von Baer, 1828), was introduced to developmental psychology by organismic theories of development (e.g., Werner, 1957; Piaget, 1954). These theories postulated that development involves an innately-predisposed structural progression whose organizing principles affect child behavior (see Overton & Horowitz, 1991 for a review). For example, differentiation featured centrally in Werner's (1957) orthogenetic principle, that "whenever there is development it proceeds from an initial state of relative globality and lack of differentiation to a state of increasing differentiation, articulation and hierarchic integration" (p. 126). It likewise appeared in Piaget's (1954) equilibration theory, that "assimilation and accommodation proceed from a state of chaotic undifferentiation to a state of differentiation with correlative coordination" (p. 352).

In 1984, Sroufe and Rutter listed differentiation as one of six developmental propositions with implications for research on psychopathology (pp. 20–23). Differentiation has since become a prominent explanation for childhood and adolescent psychiatric comorbidity (e.g., Knapp & Jensen, 2006; Lahey et al., 2004; Lilienfeld, Waldman, & Israel, 1994; Patterson, 1993). For example, Lilienfeld et al. (1994) state 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 may partially explain the particularly high rates of comorbidity among many childhood disorders" (p. 77). However, although the concept of differentiation has often been linked to childhood/adolescent comorbidity, no specifics have been provided regarding precisely (a) what the differentiation hypothesis entails, (b) how differentiation relates to comorbidity rates, (c) at what ages and in which of the sexes we should see syndrome differentiation, (d) how differentiation hypotheses might be tested, (e) what evidence is already available for/against the differentiation hypotheses, and (f) what particular knowledge gaps remain. We address each of these points in turn.

The differentiation hypothesis. One version of the differentiation hypothesis stipulates that at younger ages, fewer underlying liability distributions (latent dimensions, or latent syndrome factors) are needed

Conflict of interest statement: No conflicts declared.

Published by Blackwell Publishing, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA

to explain covariation among observed psychiatric symptoms, as compared to later ages. This implies that the dimensionality of psychopathology increases over time, perhaps from being completely unidimensional (undifferentiated) in infancy to evidencing as many dimensions as *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) syndromes in later adolescence (fully differentiated). A less extreme version of the differentiation hypothesis contends that the same number of syndromes is identifiable across time, but that they become less highly correlated (i.e., more separate) over time.

Syndrome differentiation and psychiatric comorbidity. Imposing age-invariant diagnostic cut-points upon an underlying liability distribution with fewer dimensions, or more correlated dimensions, necessarily results in higher psychiatric comorbidity rates. A simple simulation illustrates the point. We generated data from two liability distributions (i.e., two latent syndromes), with five symptom indicators each. First, we made the syndromes nearly-unidimensional (factor intercorrelation $r = .90$). We 'diagnosed' children scoring above 90% on a given liability distribution; as a result, 77% of children had comorbid diagnoses. Then, we made the syndromes well differentiated (factor intercorrelation $r = .30$). Now only 24% of children had comorbid diagnoses. This simulation shows that the observation of changing levels of comorbidity among disorders at different ages could be explained simply by differentiation of the underlying syndromes.

Syndrome differentiation, age, and gender. A big problem with application of the differentiation concept to psychopathology lies in a fairly general failure to specify exactly when and in whom differentiation of any syndrome is expected to occur. However, puberty/adolescence is an obvious candidate, because it is associated with very great changes in physiology, cognitive abilities, social milieu, and patterns of psychiatric disorders. These are also substantially different in boys and girls. Indeed, this is a period of sexual differentiation. Additionally, the transition to adolescence also serves as a 'sensitive period' for the hormone-dependent reorganization of neural circuitry relevant to the linkage of anxiety and stress response and the linkage of depression and social behavior (Sisk & Zehr, 2005). This reorganization could affect, for example, the distinctness of anxiety and depression dimensions. Given that sex differences in depression rates emerge during this hormone-dependent reorganization (Angold, Costello, Erkanli, & Worthman, 1999b), it could be the case that gender differences in syndrome differentiation arise then as well. That is, perhaps the increased rates of depression in girls arise from depression emerging as a well-differentiated category in girls, but not boys.

Testing the differentiation hypothesis. Whereas some other competing explanations for child and adolescent psychiatric comorbidity (e.g., Berksonian bias, clinical selection bias, shared/overlapping symptoms) have been empirically investigated (e.g., Angold, Costello, & Erkanli, 1999a; Lilienfeld, 2003) and often found wanting, the differentiation hypothesis has not been tested. We can, however, identify the steps that need to be taken to perform such a test, as follows:

1 For a sample of children followed longitudinally: allow the DSM-IV symptoms to serve as indicators of their respective DSM-IV syndromes and assess whether the same number of syndrome dimensions is statistically preferable across time. This establishes whether syndrome dimensionality is changing over time.
2 If the same number of syndrome dimensions is statistically preferable across time (from step 1), assess whether the relationships between symptoms and syndrome (i.e., magnitude of loadings) are also stable over time. This establishes whether syndromes hold the same substantive interpretation, or meaning, over time.
3 If the number of syndromes (from step 1) and the magnitudes of the loadings (from step 2) are stable over time, assess whether the factor correlations are stable over time. This establishes whether syndromes are becoming more distinct over time, despite not changing in dimensionality or substantive meaning.

Prior evidence relevant to the differentiation hypothesis. Although the dimensionality of multiple common DSM-IV syndromes has been tested in cross-sectional general population samples by factor analyzing DSM symptoms (see Table 1), this evidence does not provide an adequate test of the differentiation hypothesis for several reasons.

First, many studies report syndrome correlations only for markedly age-heterogeneous samples (e.g., 3–19). Second, comparisons of DSM syndrome differentiation have been made only between children across age-group, not within child across age-group. Third, across study variability of latent syndrome correlations for a given age-group can be as large as, or larger than, across age-group variability in latent syndrome correlations within a given study. For example, in Table 1, the correlation between impulsivity and hyperactivity varies more between study 3 and study 6 for the same age-group (from $r = 1.0$ to $r = .85$) than it does across younger vs. older age-groups within the same study ($r = .85$ to $r = .89$ in study 6). Moreover, latent syndrome correlations have been rarely accompanied by confidence intervals or standard errors to facilitate across-study comparisons.

Nevertheless, piecing together the available evidence across studies and across parent and teacher

Table 1 Factor correlations from published multi-syndrome factor analyses of DSM symptoms in non-referred youth

	Heterogeneous Age-group							Preschool-Age			Pre-adolescent/Adolescent-Age					
Study	1	9	2	3	4	5	6	7	5	8	9	9	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	12–17	9–11	11–19	11–16	11–16
Model	EFA	CFA	CFA	CFA	CFA	EFA	CFA	CFA	EFA	CFA	CFA	CFA	CFA	CFA	CFA	CFA
Informant	P	P	P&T	P	T	T	P	P	T	P	P	C	C	P	P	T
Measurement	SDI	SDI	C	C	C	C	C	SDI	C	C	SDI	SDI	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	1.00	.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	.18					.89			.60					
H with GAD	.59	.65	.23					.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					
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	.44	.51			
CD with MDD	.48	.56						.80			.56	.56	.62			
CD with GAD	.48	.56	.18					.80			.56	.56	.62			
CD with SOC	.33	.27	.33					.59			.32	.27	.33			
SAD with MDD	.33	.73						.72			.68	.70	.82			
SAD with GAD	.33	.73						.72		.87	.68	.70	.82			
SAD with SOC	.47	.57						.65		.57	.57	.58	.77			
MDD with GAD	1.00	1.00	.73					1.00			1.00	1.00	1.00			
MDD with SOC	.63	.73						.76			.67	.71	.76			
GAD with SOC	.63	.73						.76		.55	.67	.71	.76			

Notes: If two factors are unidimensional, their correlations with other factors are the same (duplicated here). † = only found at age 4-10, not 11-17, but factor correlations not reported by age-group. 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; C=child; SDI= structured diagnostic interview; C=checklist. ODD=Oppositional Defiant; H=Hyperactivity; CD=Conduct; IN=Inattention; I=Impulsivity; SAD=Separation Anxiety; SOC=Social Phobia; GAD=Generalized Anxiety MDD=Depression.

informants, there appears, at first, to be some evidence of changing syndrome dimensionality between preschool-age and preadolescence, particularly among the disruptive disorders. By preadolescence, oppositional defiant disorder, conduct disorder, inattention, and hyperactivity/impulsivity are distinct dimensions, with hyperactivity and impulsivity sometimes distinguishable as well (Lahey et al., 2008; Burns et al., 1997b, 2001; Molina et al., 2001). However, in preschoolers, oppositional defiant and conduct disorder syndromes have been found to be one dimension (Sterba, Egger, & Angold, 2007a), and hyperactivity, impulsivity, and inattention syndromes have been found to be one dimension – or nearly so (see Table 1; Bauermeister, 1992; Sterba et al., 2007a). Additionally, among younger (4–10) but not older (11–17) children, Lahey et al. (2004) found that oppositional defiant disorder and hyperactivity/impulsivity formed a single dimension that was differentiable from conduct disorder and inattention syndromes (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, oppositional defiant and conduct disorder syndromes have been found to be correlated at up to $r = .91$, hyperactivity/impulsivity and oppositional defiant disorder syndromes have been found to be correlated at up to $r = .87$, and the correlation between hyperactivity/impulsivity and inattention has been reported to be as high as $r = .85$.

Even less evidence exists in Table 1 for developmental differentiation among emotional syndromes from preschool to preadolescence, from parent, teacher, and child informants. Common emotional syndromes were either consistently differentiable across age-groups (separation anxiety disorder and social phobia), or consistently undifferentiable across age-groups (major depression and generalized anxiety disorder, except in Hartman et al., 2001). However, some emotional syndromes (major depression, generalized anxiety, social anxiety) but not others (separation anxiety) were more correlated with disruptive syndromes in preschoolers than preadolescents/adolescents.

Gaps in our understanding of syndrome differentiation. However, these conclusions are decidedly preliminary because, on the basis of these published results, we are unable to (a) account for within-study sampling variability in syndrome correlations, (b) use between-child syndrome differences to make inferences about within-child syndrome change, or (c) generalize across studies that vary considerably with respect to the specificity and comprehensiveness of symptom measurement and the handling of symptoms shared by multiple disorders (e.g., sleep disturbance).

Table 1 also indicates that we know even less about whether syndrome differentiation occurs from preadolescence to adolescence than we do about syndrome dimensionality change from preschool to preadolescence. This knowledge gap exists despite the fact that disorder prevalences change substantially across adolescence and new gender differences in disorder prevalences emerge. No studies have compared syndrome dimensionality in preadolescents, early adolescents, and later adolescents. At a time in which DSM-V workgroups are considering revising the distinction between generalized anxiety disorder and major depression disorder (Goldberg, 2008; Moffitt et al., 2007), this lack of evidence is surprising.

The aim of this study was to use longitudinal data from a representative population sample of children and adolescents to overcome these gaps in examining the dimensionality of common Axis I syndromes across age 9–16, and by gender. This study provides the first inferential test of the differentiation hypothesis as applied to child and adolescent psychopathology.

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. The original age 9 cohort was revisited at age 10, 11, 12, 14, 15, 16, 19, and 21. The original age 11 cohort was revisited at age 12, 13, 14, 15, 16, 19, and 21. The original age 13 cohort was revisited at age 14, 15, 16, 19, and 21. 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). Informed consent was obtained. The GSMS dataset contains longitudinal information on child and parent psychopathology, psychiatric service access and use, and family and community resources. The present analyses were based on data from when the children were 9–16. Sampling weights accounted for the 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 the waves included in these analyses (range 75–94%). The sampling weights were adjusted for nonresponse at wave 1. Estimation methods employed for binary indicators only accommodated pairwise deletion of missing data under Missing Completely at Random assumptions (multiple imputation is problematic for sparse binary data; Allison, 2006). Cohort differences were examined in Sterba, Egger, and Angold (2007b), but

not found, so 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 the criteria operationalized in the DSM-IV. Child and parent reports were combined using the 'or' rule (Costello et al., 1996) – except in the case of ADHD, where, following Angold et al. (1995), we relied solely on parent report. CAPA symptom dimension test–retest intra-class correlations ranged from .50 (oppositional defiant) to .88 (major depression) (Angold et al., 1995).

Statistical analysis

Modeling framework. In the introduction, three sequential steps for testing the differentiation hypothesis were described. Ideally, these model-building steps would be implemented in a longitudinal factor analysis framework. However, a longitudinal factor analysis with 7 factors and 66 relatively-sparse binary items at each of 8 occasions (56 factors, 528 binary items total) is not estimable with current software. Instead, we estimated each 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 age-group were: $N = 936$ for age 9–10, $N = 2588$ for age 11–13, $N = 3150$ for age 14–16. Thus, within each age-group's 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.

Model-building step 1: within age-block dimensionality testing. In model-building step 1, syndrome dimensionality was assessed by comparing the fit of alternate confirmatory factor analysis (CFA) models at each age-block to identify the preferred number of dimensions per age-block. Alternative CFA models always allowed DSM syndromes to correlate and always allowed DSM symptoms to load on their respective DSM syndrome – with the exception of three unendorsed symptoms at age 9–10 (conduct disorder's human cruelty, confrontational stealing, and runs away symptoms), and one non-administered symptom at age 9–13 (attention deficit/hyperactivity disorder's forgetting symptom). Alternative CFA models were chosen to reflect possible patterns of differentiation identified from Table 1's cross-sectional studies. We began with the least restrictive model, which had the greatest hypothesized number of dimensions: major depression, generalized anxiety, separation anxiety, social anxiety, oppositional defiant, conduct, hyperactivity, impulsivity, and inattention syndromes. We then tested, through a series of five model comparisons, whether the decrements in model fit associated with rendering certain syndromes unidimensional (first, major depression + generalized anxiety; then hyperactivity + impulsivity; then hyperactivity + impulsivity + inattention; then oppositional defiant + conduct; then oppositional defiant + hyperactivity) were statistically significant, in the context of the other syndromes. We assessed model fit with RMSEA (population misfit per degree of freedom; ≤.05 well-fitting) and CFI (fit relative to a null baseline; ≥.95 well-fitting) 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 the Bayesian Information Criterion (BIC) and sample-size-adjusted BIC, which penalizes for model complexity (lower BIC is better).

Model-building steps 2 and 3: across age-block factor loading and factor correlation comparisons. Preliminarily, the best-fitting, age-block-specific models from step 1 were compared to see whether they showed the same number of syndrome dimensions and the same pattern of significant/nonsignificant loadings across age-blocks (i.e., 'configural' invariance). If so, the best-fitting age-block-specific models from step 1 were compared to see whether syndromes maintained the same substantive meaning over time (step 2: factor loading or 'metric' invariance). If so, we then tested whether these syndromes differentiated over time (step 3: factor correlation invariance).

Fitting CFA models separately for 9–10 year-olds, 11–13 year-olds, and 14–16 year-olds complicated comparison of the factor loadings and factor correlations across age-blocks. 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 age-group 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 age-group difference in that factor loading. That confidence interval was used to test the null hypothesis that the difference between the two loadings is 0 in the population. The same procedure was repeated for all factor loadings, for all three age-group 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 age-groups, with the following caveats. Instead of simply using the estimated r and its SE to generate bootstrap resamples, Fisher's r to z' transformation was first used to transform the estimated r to an approximately-normal metric. Second, the 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 distributions of the transformed r. The transformed rs were

then back-transformed to create sampling distributions of *r*s.

Testing for gender differences. Given symptom sparseness and model complexity, convergence and estimation problems prohibited multiple-group modeling by gender, for *each* age-block *separately*. However, since the results presented below showed essentially the same factor structure across age-groups, we collapsed across age-group (i.e., combined all age-groups into one model, again accounting for the clustering of observations within person) to do multiple-group testing by gender. This allowed us to determine whether the final best-fitting factor structure holds in both sexes. Identification methods used here for binary-response multiple-group modeling were described in Millsap and Yun-Tein (2004), which included imposing item threshold invariance in all models and then investigating loading invariance and then factor correlation invariance.

Procedures for handling overlapping symptoms. Symptoms shared across syndromes can artificially inflate the magnitude of syndrome covariation (Angold et al., 1999a). Hence, item-specific residuals of overlapping symptoms were allowed to correlate: (a) irritability (from oppositional defiant disorder, generalized anxiety disorder, and major depression disorder), (b) too little/much sleep (from generalized anxiety disorder and major depression disorder), (c) school refusal/absence (separation anxiety disorder and conduct disorder), and (d) lying/blaming (conduct disorder and oppositional defiant disorder). The fatigue symptom for generalized anxiety disorder and major depression disorder was found to be correlated >.95. So, 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 symptom from generalized anxiety and major depression disorders cross-loaded on the inattention syndrome, and the restless/keyed-up symptom from generalized anxiety disorder cross-loaded on the hyperactivity syndrome (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, 1998–2007). As a result of low endorsement rates, several symptoms belonging to the same disorder were parceled (summed) into one indicator to avoid estimation problems stemming from zero cells in bivariate contingency tables. At age 9–10, these symptoms were anhedonia, psychomotor agitation/retardation, and depressed/irritable mood (from major depression disorder), unorganized and loses things (from attention deficit hyperactivity disorder), and animal cruelty and forced sex (from conduct disorder). At age 11–12, these symptoms were animal/human cruelty, confrontational stealing, and forced sex (from conduct disorder), and breaks curfew and runs away (from conduct disorder). At age 14–16, these symptoms were animal/human cruelty, confrontational stealing, and forced sex (from conduct disorder). Sensitivity analyses based only on the relevant submodels (that had smaller contingency tables) indicated that the 1–3 parcels did not change the dimensionality results. For instance, when we examined just the conduct disorder and oppositional defiant disorder symptoms we could compare the results of using *animal cruelty* and *forced sex* as separate indicators vs. parceling them together.

Results

Model-building step 1: within age-block dimensionality testing

Dimensionality testing via model comparisons #1–#5 in Table 2 resulted in eight-factor final models (major depression/generalized anxiety, separation anxiety, oppositional defiant, conduct, social anxiety, hyperactivity, impulsivity, and inattention syndromes) 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 major depression and generalized anxiety as separate factors resulted at age 9–10 and 11–13 in a linear dependence between major depression and generalized anxiety ($r \geq 1.00$), indicating dimensional inseparability (Lahey et al., 2008 used similar procedures). The BIC showed worse fit for separate generalized anxiety and major depression factors at age 9–10, and essentially unchanged fit for separate generalized anxiety and major depression at age 11–13. At age 14–16, the correlation between major depression and generalized anxiety was $r = .90$, which was statistically differentiable according to χ^2 and BIC. Despite some slight indication of a dimensionality change, from unidimensionality to near-unidimensional, we retained generalized anxiety and major depression as unidimensional in our final models. In model comparison #2, oppositional defiant and conduct syndromes 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 oppositional defiant and conduct disorders as separate factors across age. In model comparison #3, at all age-groups, χ^2 and BIC identified a significant decrement in fit from collapsing a trifactorial (inattention, hyperactivity, impulsivity) model for attention deficit hyperactivity disorder into a unifactorial model. Model comparison #4 identified a smaller, but significant, decrement from collapsing a trifactorial into a bifactorial (hyperactivity/impulsivity, inattention) model. A trifactorial attention deficit hyperactivity disorder specification was retained for all age groups. Finally, model comparison #5 indicated that oppositional defiant and hyperactivity

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: *** $p < .001$; ** $p < .01$; * $p < .05$; – could not be estimated. [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. ODD = oppositional defiant disorder; CD = conduct disorder; H = hyperactivity; IN = inattention; I = impulsivity; SAD = separation anxiety disorder; SOC = social phobia; MDD = major depression disorder; GAD = generalized anxiety disorder.
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

syndromes were always statistically distinct according to χ^2 and BIC.

Model-building Steps 2 and 3: across age-block factor loading and factor correlation comparisons

Table 3 shows that configural invariance (the same pattern of significant loadings across age-groups) was partially met for factor loadings in the final models. All but three symptoms showed positive, significant loadings on their designated DSM syndromes at each age-group. The three age-variant symptoms were those that were allowed to cross-load on multiple factors to prevent artifactual inflation of their factor correlation estimates. The primary loading of the major depression disorder's concentration symptom was age-invariant, but its secondary loading was not. The secondary loadings of generalized anxiety disorder's restlessness symptom and concentration symptom were age-invariant, but their primary loadings were not. Table 3 also shows that the more stringent metric invariance (same loading magnitude across age-groups) was only partially met for the factor loadings in the final models. The fewest across-age primary loading differences were found for social anxiety (0%) and hyperactivity (0%). The most primary across-age loading differences were found for conduct disorder, where 17% of loadings changed at age 11–13 and 42% of loadings changed at age 14–16, and for major depression/generalized anxiety, where 0% of loadings changed at age 11–13 but 64% of loadings changed at age 14–16. But, whereas the significant changes in conduct disorder symptom loadings were inconsistent (either increasing then decreasing or decreasing then increasing), all the significant changes in major depression/generalized anxiety loadings were increases. The latter finding is 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 increased at age 14–16 for major depression/generalized anxiety.

Finally, Table 5 shows that most factor correlations were not significantly different across age-groups, and that the factor correlations had sizable sampling variability (large 95% CIs). Disruptive syndromes were most highly correlated with each other across age, as were emotional syndromes. An exception was major depression/generalized anxiety, which sometimes associated more strongly with disruptive syndromes. When factor correlations changed significantly across age-groups, 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 unstable, low correlations between emotional and disruptive syndromes, and did not represent a consistent pattern of differentiation. For example, social anxiety and major depression/generalized anxiety became less correlated with conduct disorder syndrome by age 14–16. Yet, separation anxiety became more correlated with inattention, hyperactivity, impulsivity and oppositional defiant disorder syndromes by age 14–16.

Testing for gender differences

In order to estimate multiple-group models by gender, we had to collapse across age-groups. This was feasible given that we found approximately the same factor structure across age-groups. A global test of invariance of all factor loadings across gender was rejected, $\Delta\chi^2$ (42.407, 24) $p = .01$; however, absolute fit (RMSEA = .017) and relative fit (CFI = .979)

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	.91		*(.03)*	.93		*(.01)*	.95		*(.02)*
Sustaining_attention	.89		*(.03)*	.94		*(.02)*	.93		*(.02)*
Listening	.97		*(.02)*	.92	**11/14**	*(.02)*	.98		*(.01)*
Following_through	.95		*(.02)*	.94		*(.01)*	.92		*(.02)*
Organizing	.76[a]	**9/11**	*(.06)*	.95		*(.04)*	.85		*(.06)*
Sustaining_tasks	.98		*(.03)*	.98		*(.02)*	.95		*(.02)*
Loses_things	.76[a]		*(.06)*	.85		*(.03)*	.88		*(.03)*
Easily_distracted	.95		*(.02)*	.94		*(.02)*	.94		*(.02)*
Forgetful							.99		*(.03)*
GAD concentrating	.95		*(.05)*	1.05	**11/14**	*(.03)*	.86		*(.03)*
MDD concentrating	.26		*(.13)*	.52	**11/14**	*(.06)*	.15†		*(.10)*
Hyperactivity									
Fidgets	.87		*(.04)*	.88		*(.02)*	.90		*(.03)*
Leaves_seat	.93		*(.03)*	.94		*(.02)*	.87		*(.05)*
Runs/climbs	.93		*(.03)*	.95		*(.02)*	.95		*(.03)*
Quiet_activities	.96		*(.02)*	.99		*(.01)*	.96		*(.03)*
On_the_go	.95		*(.04)*	.99		*(.01)*	.96		*(.02)*
Talks_excessively	.95		*(.02)*	.96		*(.01)*	.97		*(.02)*
GAD restlessness	.79		*(.07)*	.64	**11/14**	*(.04)*	.33	**9/14**	*(.06)*
Impulsivity									
Blurts_answers	.96		*(.02)*	.96		*(.01)*	.99		*(.02)*
Awaiting_turn	.97		*(.03)*	.96	11/14	*(.01)*	.86	9/14	*(.04)*
Interrupts	.94		*(.02)*	.99		*(.02)*	.99		*(.03)*
Conduct									
Bullies	.81		*(.07)*	.69		*(.11)*	.73		*(.06)*
Initiates_fights	.50		*(.08)*	.60		*(.06)*	.67		*(.07)*
Used_weapon	.64		*(.09)*	.40	**11/14**	*(.1)*	.78		*(.06)*
Fire_setting	.43		*(.1)*	.57		*(.07)*	.60		*(.07)*
Property_destruction	.57		*(.11)*	.76	11/14	*(.08)*	.54		*(.07)*
Breaks_in	.87	9/11	*(.07)*	.59		*(.06)*	.61	**9/14**	*(.07)*
Lies/cons	.58	9/11	*(.08)*	.79		*(.06)*	.80	9/14	*(.05)*
Steals_w/o_confronting	.78		*(.06)*	.79		*(.05)*	.69		*(.05)*
Breaks_curfew	.41		*(.16)*	.32[b]		*(.09)*	.49		*(.09)*
Runs_away				.32[b]	**11/14**	*(.09)*	.67		*(.09)*
Truant	.42		*(.12)*	.63		*(.11)*	.46		*(.1)*
CD cruel/steal/sex parcel	.45		*(.13)*	.53		*(.09)*	.71		*(.08)*
Oppositional defiant									
Loses_temper	.39	9/11	*(.08)*	.63		*(.05)*	.70	**9/14**	*(.04)*
Argues	.60		*(.08)*	.64		*(.05)*	.73		*(.04)*
Actively_defies	.74		*(.06)*	.82		*(.04)*	.77		*(.04)*
Deliberately_annoys	.70		*(.08)*	.70		*(.05)*	.68		*(.05)*
Blames_others	.58		*(.06)*	.60		*(.05)*	.60		*(.04)*
Touchy/annoyed	.43		*(.09)*	.42	11/14	*(.07)*	.64	9/14	*(.05)*
Angry/resentful	.46		*(.07)*	.61	**11/14**	*(.04)*	.77	**9/14**	*(.03)*
Spiteful/vindictive	.52		*(.08)*	.64		*(.05)*	.63		*(.05)*
Separation anxiety									
Anticipatory_distress	.83		*(.06)*	.88		*(.07)*	.81		*(.07)*
Worry_loss	.82		*(.07)*	.65		*(.07)*	.61		*(.08)*
Worry_untoward_event	.78		*(.07)*	.78		*(.12)*	.99	9/14	*(.07)*
School_refusal	.85		*(.1)*	.67		*(.07)*	.68		*(.08)*
Fearful_alone	.71		*(.12)*	.66	11/14	*(.1)*	.92		*(.05)*
Sleep_alone	.48		*(.09)*	.66		*(.08)*	.68		*(.1)*
Separation_nightmares	.81	9/11	*(.07)*	.52		*(.13)*	.71		*(.12)*
Somatic_complaints	.82		*(.06)*	.76		*(.09)*	.63		*(.09)*
Depression/Generalized anxiety									
Restlessness	−.02†		*(.1)*	.16	**11/14**	*(.06)*	.71	**9/14**	*(.05)*
Concentrating	−.06†		*(.09)*	−.22	**11/14**	*(.06)*	.11†		*(.06)*
Irritability	.67		*(.14)*	.55	11/14	*(.09)*	.77		*(.05)*
Muscle_tension	.40		*(.14)*	.52	**11/14**	*(.09)*	.87	**9/14**	*(.04)*
Sleep_disturbance	.57		*(.1)*	.52		*(.06)*	.55		*(.05)*
Depressed_mood	.51[c]		*(.16)*	.73		*(.08)*	.86	9/14	*(.04)*
Anhedonia	.51[c]		*(.16)*	.65		*(.17)*	.70		*(.11)*
Weight_change	.33		*(.09)*	.27		*(.06)*	.40		*(.05)*
Insomnia/hypersomnia	.49		*(.11)*	.42		*(.1)*	.64		*(0.5)*

Table 3 (Continued).

	Age 9–10		Age 11–13			Age 14–16		
	Estimate	*(S.E.)*	Estimate		*(S.E.)*	Estimate		*(S.E.)*
Psychomotor_agit./retard.	.51[c]	*(.16)*	.73		*(.12)*	.92	9/14	*(.07)*
Guilt/worthlessness	.62	*(.1)*	.73		*(.07)*	.82		*(.04)*
Think/decide/concentrate	.42	*(.14)*	.30	**11/14**	*(.12)*	.70		*(.09)*
Suicidal_ideation	.45	*(.09)*	.41	**11/14**	*(.08)*	.78	**9/14**	*(.04)*
Fatigue parcel	.55	*(.13)*	.44	11/14	*(.09)*	.70		*(.05)*
Social phobia								
Fear_social/performance	.75	*(.14)*	.76		*(.13)*	.99		*(.09)*
Exposure_anxiety	.96	*(.17)*	.98		*(.15)*	.85		*(.08)*

Notes: [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. *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 $^{\mathbf{9/11}}$ for alpha = .01. A loading significantly different from age 11–13 to 14–16 at is denoted $^{11/14}$ at alpha = .05 and $^{\mathbf{11/14}}$ for alpha = .01. A loading significantly different from age 9–10 to 14–16 is denoted $^{9/14}$ at alpha of .05 and $^{\mathbf{9/14}}$ at alpha = .01. † = Loading *not* significantly different than 0 at alpha = .05. GAD = generalized anxiety disorder; MDD = major depression disorder; CD = conduct disorder.

remained good in the all-invariant-loading model. We followed up with a series of separate, loading-by-loading differential item functioning (DIF) tests to identify which and how many gender-variant loadings were contributing to finding a global gender loading difference. We found that 12 of the total 65 symptom or parcel loadings and cross-loadings were responsible for this global loading gender difference. Specifically, conduct disorder's fire-setting and stealing symptoms, oppositional defiant disorder's defying, spiteful, and annoying symptoms, and generalized anxiety disorder's irritability and sleep disturbance symptoms were more strongly related to their latent syndrome among boys. However, generalized anxiety disorder's restlessness, difficulty concentrating, and fatigue symptoms were more strongly related to their latent syndrome among girls. Thus, no attention deficit/hyperactivity disorder, separation anxiety disorder or social anxiety disorder symptoms displayed across-gender loading-invariance. Generalized anxiety disorder symptoms showed the most loading variation across gender. However, if we are more conservative and adjust for alpha inflation to retain a family-wise nominal Type I error rate of .05 for DIF-testing, this renders only conduct disorder's fire-setting symptom as having by-gender variability. Moreover, this amount of partial loading invariance across gender turned out to be of little consequence for testing the differentiation hypothesis across gender. Syndrome correlations were found not statistically different across gender, regardless of whether we imposed $\Delta\chi^2$ (14.87, 9), p = .095 or removed $\Delta\chi^2$ (16.15, 10), p = .096 across-gender equality constraints on the loadings for fire-setting, stealing, defying, annoying, spiteful, irritability, sleep disturbance, restlessness, and concentrating symptoms.

Table 4 Average proportion of variance in *Diagnostic and Statistical Manual* (DSM) symptoms explained by their designated DSM syndrome

	Age 9–10	Age 11–13	Age 14–16
Inattention	.870	.844	.868
Hyperactivity	.905	.868	.876
Impulsivity	.937	.914	.894
Major depression/ generalized anxiety	.382	.353	.587
Separation anxiety	.496	.593	.584
Conduct	.393	.388	.427
Oppositional defiant	.408	.319	.478
Social phobia	.769	.744	.852

Discussion

This study represents the first inferential test of the *differentiation hypothesis* across the transition to adolescence using diagnostic-interview symptom data relating to multiple syndromes in a general population sample. Overall, we found that the same number of syndrome dimensions (eight) with the same pattern of fixed and free symptom loadings fit well for all age-groups: hyperactivity, inattention, impulsivity, oppositional defiant, conduct disorder, separation anxiety, social anxiety, and major depression/generalized anxiety syndromes. Hence, areas of suspected dimensionality change from preschool to preadolescence (dimensionality of oppositional defiant disorder and conduct disorder; dimensionality of hyperactivity, impulsivity, and inattention; dimensionality of hyperactivity and oppositional defiant disorder; Bauermeister, 1992; Lahey et al., 2004; Sterba et al., 2007b) actually showed stable dimensionality from preadolescence to later adolescence (Table 2). But an area of suspected dimensionality stability from preschool to preadolescence (dimensionality of major depression and generalized anxiety syndromes; Lahey et al.,

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)

Notes: ODD = oppositional defiant disorder; CD = conduct disorder; H = hyperactivity; IN = inattention; I = impulsivity; SAD = separation anxiety disorder; SOC = social phobia; MDD = major depression disorder; GAD = generalized anxiety disorder.

2004, 2008; Sterba et al., 2007b in Table 1) showed indications of some differentiation from preadolescence to later adolescence (Table 2), with syndrome correlations falling from $r = 1.0$ to $r = .90$. Other indications of this developmental shift or reorganization of major depression/generalized anxiety starting at age 14–16 was that 64% of their loadings varied significantly across age-block, and these displayed a consistent pattern of change – increases at age 14–16 (Table 3). This translated into increases in the proportions of symptom variance accounted for by the major depression/generalized anxiety syndrome at age 14–16 (Table 4). In contrast, an average of only 25% of primary symptom loadings for the other seven syndromes (range 0–42%) varied significantly across age-block, and these displayed no consistent pattern of change. Some symptom reorganization for major depression/generalized anxiety disorder between 4–10 vs. 11–17 years was also found by Lahey et al. (2004). Yet treating major depression/generalized anxiety as unidimensional at age 14–16 still resulted in good model fit.

Moreover, across-age correlations among these eight putative dimensions displayed few significant differences, and showed no consistent pattern of developmental differentiation (Table 5). For example, whereas one emotional syndrome (social anxiety) became significantly more distinct from some disruptive syndromes (conduct disorder, but not oppositional defiant disorder, hyperactivity, impulsivity, or inattention), another (separation anxiety) became significantly *less* distinct from disruptive syndromes (oppositional defiant disorder, hyperactivity, impulsivity, inattention, but not conduct disorder). These heterotypic correlations could temporarily (for one or two age-groups) be very small ($< r = .10$) – lower than has been found in prior studies (see Table 1). But their 95% CIs usually included values found in prior studies. The only consistent pattern conceivably interpretable as developmental differentiation, and in line with prior findings in Table 1, was the decreasing correlations between major depression/generalized anxiety and conduct, hyperactivity, impulsivity, and inattention syndromes (but not oppositional defiant syndrome). However, these trends were not statistically significant. Overall, the correlations among disruptive syndromes showed the greatest stability, and correlations between disruptive and emotional syndromes showed the least stability.

Regarding gender differences, whereas we were unable to test for gender differences in longitudinal syndrome differentiation per se, we had grounds for collapsing across age-blocks in order to assess

gender-invariance of syndrome factor structure. By doing so, we found that about one-fifth of the factor loadings varied by gender. Similarly, Lahey et al. (2008) found that one-third of their factor loadings for child-reported psychopathology varied by gender. However, maintaining a nominal alpha for these loading-by-loading significance tests resulted in only one remaining highly variant loading (fire-setting). Likewise, Lahey et al. (2008) found only two large and variant loadings (breaking and entering; running away). Constraining versus freeing the 12 gender-variant loadings did not change the syndrome correlations, which were not significantly different by gender. We conclude, therefore, that considerable gender differences in the relationships among syndromes were absent. Despite the fact that hormone-dependent neural circuitry relevant to anxiety and depression reorganizes at adolescence (Sisk & Zehr, 2005) and gender differences in rates of depression and some anxiety disorders emerge at adolescence, there is no evidence that either phenomenon corresponds with meaningful changes in the relationships among generalized anxiety, separation anxiety, social anxiety, and major depression in boys or girls.

Conclusions

All in all, this work, coupled with our previous work on the dimensionality of preschool symptomatology, indicates that the kind of marked developmental change in the structure of psychopathology – from a relatively undifferentiated 'mass' to distinct DSM dimensions – as predicted by the orthogenetic principle (Werner, 1957; Lilienfeld et al., 1994), does not appear to occur. In particular, there is no evidence of clinically-meaningful syndrome differentiation. A little statistically-detectable, but hardly very striking, differentiation may occur early – by preadolescence – among disruptive syndromes (Table 1; Bauermeister, 1992; Lahey et al., 2004; Sterba et al., 2007b). Some further differentiation may occur during adolescence, for generalized anxiety and major depression, and between some emotional and disruptive syndromes (Table 2). But the striking pattern is one of consistency rather than change. Despite the fact that adolescence is accompanied by substantial changes in the rates of a range of psychiatric symptoms and disorders, it appears that the *structural organization* of those symptoms and disorders is highly stable.

Additional research extending dimensionality results into young adulthood would be useful to clarify precisely (a) whether major depression and generalized anxiety indeed remain at- or near-unidimensional into adulthood, and (b) whether the generalized anxiety construct displays longitudinal coherence, in the light of our inconsistent loadings for two generalized anxiety disorder symptoms (also found in Hartman et al., 2001). Nevertheless, given our findings, and those of others who have looked at these questions in childhood and adolescence, we would be surprised to find substantial reorganization of the symptom structure of generalized anxiety and major depression syndromes in adulthood – particularly given their very close association in adults (e.g., Moffitt et al., 2007).

Limitations

Several limitations of this study deserve mention. First, testing competing statistical models is only one way of examining the longitudinal internal validity of the DSM nosology. Second, testing our final, multi-syndrome model at all eight ages simultaneously (rather than separately at three condensed age-blocks) would have allowed us to quantify decrements in model fit associated with imposing age-invariance constraints. However, it was not even possible to fit our final, multi-syndrome model at *two* ages simultaneously. For two ages, there were 16 factors and 120 sparse categorical indicators and this exceeds the capacity of current estimation routines for categorical data, despite our large sample (see Jöreskog, 2002). Third, symptom sparseness, which was marked even under the combined-informant 'or' rule, prohibited our splitting analyses by informant, because that would have resulted in even greater sparseness. However, Lahey et al. (2008) found no important informant (parent versus child) differences for similar multi-syndrome CFA models (see Table 1). Nonetheless, potential moderation of longitudinal syndrome differentiation by informant status is an important avenue for future research. Fourth, by-race comparisons of syndrome loading and covariation patterns did not yield meaningful or consistent differences, and so were not presented here. Fifth, we tested only a small subset of the possible models that could have generated these data – the subset based upon DSM-IV classification. This subset could, theoretically, be wrong. On the other hand, exploratory factor analytic studies (e.g., Lahey et al., 2004; Bauermeister, 1992) have revealed dimensions that are quite reminiscent of the DSM-IV. These studies suggest that our subset of models are serious candidates for the 'true' factor structure of childhood and adolescent psychopathology.

Implications for comorbidity models featuring higher order constructs

An essentially-unidimensional major depression/generalized anxiety syndrome over time, or an unstable-dimensionality of major depression/generalized anxiety syndrome over time, has important implications for comorbidity models that use threshold DSM diagnoses as indicators of higher-order 'core psychopathological constructs' (e.g.,

Watson, 2005). In this approach, observed major depression and generalized anxiety diagnostic scores are treated as separate indicators, and a latent higher-order factor (alternately labeled 'anxious/misery' by Vollebergh et al., 2001 or 'distress' by Slade & Watson, 2006) is included to explain their covariation. If major depression and generalized anxiety syndromes are indeed unidimensional, such models are misspecified and higher-order anxious/misery or distress factors compensate for this misspecification. Further empirical exploration of this issue is outside the scope of the present paper, but is included in an online appendix.

Implications for etiology of successive comorbidity

These results suggest some etiological implications regarding what has been termed longitudinal, successive comorbidity, from one disorder to another (controlling for concurrent comorbidity at any given timepoint; Angold et al., 1999a). Prior research has identified several patterns of longitudinal, successive comorbidity from typically childhood-onset syndromes to typically adolescent-onset syndromes (e.g., separation anxiety to depression, Silberg et al., 2001; oppositional defiant disorder to conduct disorder, Loeber et al., 1993, 1997) that may involve shared environment and/or genetic influences (see Costello, Mustillo, Erkanli, Keeler, & Angold, 2003 for other examples). One potential explanation for such patterns is a developmental or maturational shift at the syndrome level, where a single underlying liability exists at an early age (e.g., oppositional defiant/conduct disorder) and multiple liability distributions exist at later ages. Because of this maturational shift or 'pathoplasticity' (Lonigan, Phillips, & Hooe, 2003; Patterson, 1993), some children with high scores on the underlying liability could manifest one syndrome at one age and another at a later age (heterotypic continuity). Yet other children could manifest with the same syndrome across age (homotypic continuity). Another potential explanation for such patterns is that the dimensionality of psychiatric syndromes stays largely intact across development, but some children progress through a pathway or sequence of disorders, for a variety of reasons (including one syndrome directly causing another, or a mutual risk factor triggering each in turn, e.g., Neale & Kendler, 1995). For the syndromes considered here, the second general explanation seems more plausible than the first.

Implications for developmentally-modified psychiatric nosology

Knapp and Jensen (2006) commented in *Toward a new diagnostic system for child psychopathology: Moving beyond the DSM* that "the phenomenon of comorbidity, then, may reflect an underlying global psychopathological factor that assumes a more specific symptomatic form with increasing differentiation at successive stages of development" (p. 165). If this were true, alternate nosological criteria across age would be warranted based on imposing as little as one diagnostic cutpoint on a unidimensional psychopathological syndrome at young ages, and imposing more diagnostic cutpoints on an increasingly multidimensional liability distribution with increasing age. If this were true, childhood/adolescent comorbidity could also be a simple by-product of diagnostic cutpoints being placed on a unidimensional, rather than multidimensional, underlying liability distribution. However, our empirical investigation of this often-stated but little-tested *differentiation hypothesis* provided no convincing evidence that prominent differentiation occurs in the commonest forms of psychopathology from childhood to adolescence. Combining our work with previous cross-sectional studies, we summarize by saying that there is no evidence that a general process of psychopathological differentiation is responsible for high rates of childhood/adolescent comorbidity and no evidence that an entirely new diagnostic system for child psychopathology is warranted based on presumed developmental changes in syndrome differentiation.

Supplementary material

The following supplementary material is available for this article:

Appendix (Pdf document)

This material is available as part of the online article from:

http://www.blackwell-synergy.com/doi/abs/10.1111/j.1469-7610.2010.01458.x

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Acknowledgements

This work was supported by a NIMH F31 MH080494 award for Sterba, a NIH K23-MH080230 award for Copeland, a NIMH R01 MH081025 award for Egger, a NIDA R01 DA011301 award for Costello and Erkanli and a NIMH R01 MH0704888 award for Angold.

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Key points

- A common explanation of high psychiatric comorbidity in childhood/adolescence is that underlying psychopathological syndromes are not yet fully differentiated, but should differentiate across development.
- This explanation has not been tested for common Axis I syndromes using longitudinal data on *Diagnostic and Statistical Manual* (DSM-IV) psychiatric symptoms. Results showed little evidence of statistically significant differentiation in common Axis I syndromes across ages 9–16, except between depression and generalized anxiety.
- With the possible exception of depression and generalized anxiety, this study provides evidence of the stable underlying structure of DSM-IV syndromes across the transition to adolescence. A general process of psychopathological differentiation is not likely to be responsible for high rates of comorbidity in this timeframe.

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Manuscript accepted 6 January 2010