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Developmental Trajectories of Co-Occurring Depressive, Eating, Antisocial, and Substance Abuse Problems in Female Adolescents

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Growth trajectories of co-occurring symptomatology were examined in a community sample of 493 female adolescents who were followed annually from early to late adolescence. On average, depression, eating disorder, and substance abuse symptoms increased over time, whereas antisocial behavior decreased. Increases in each symptom domain were associated with relative increases in all other domains. Initial depressive and antisocial behavior symptoms predicted future increases in the other; substance abuse and antisocial behavior symptoms also showed prospective reciprocal relations. Initial depression predicted increases in eating disorder and substance abuse symptoms. Initial eating disorder symptoms predicted increases in substance abuse problems. Finally, the results suggest that the developmental covariation between depressive and eating disorder symptoms and between antisocial behavior and substance abuse symptoms was accounted for by distinct but related 2nd-order growth parameters.

Keywords: comorbidity, developmental trajectories, female psychopathology

Recent epidemiological investigations of gender-related aspects of psychopathology have helped to elucidate a number of worrisome aspects about female psychopathology during the 2nd decade of life. For female adolescents, adolescence is characterized by increased levels of symptomatology in a number of domains, particularly depression, eating pathology, and substance use symptoms (Langerbucher & Chung, 1995; Lewinsohn, Pettit, Joiner, & Seeley, 2003; Stice, Killen, Hayward, & Taylor, 1998); externalizing problems are also common among teenage girls (Broidy et al., 2003). Further, adolescence is marked by high rates of co-occurring psychopathology for girls (Kessler et al., 1994). Indeed, rates of female comorbidity typically surpass the rates of single disorders (Angold, Costello, & Erkanli, 1999; Kessler et al., 1994) and tend to increase with age (Tolan & Henry, 1996).

Although progress has been made in understanding psychiatric comorbidity (Angold et al., 1999; Caron & Rutter, 1991), significant gaps in knowledge remain regarding the developmental course of co-occurring domains of psychopathology. Relatively little is known about the temporal relations among symptom domains or about individual differences in mental health trajectories comprising co-occurring problems. In addition, few studies have prospectively examined more than two co-occurring conditions, particularly those that span syndromal domains, which seem important given evidence that the presence of a third syndrome might significantly alter developmental processes (Harrington, Rutter, & Frombonne, 1996).

The overarching goal of this study was to examine the developmental trajectories of the symptoms of psychiatric disorders that often co-occur over a 5-year span of adolescence. Specifically, we investigated the course and covariation among four domains of self-reported symptomatology in a community sample of female adolescents: depressive, eating disorder, antisocial, and substance abuse symptoms. The aims were to (a) characterize girls' symptom trajectories during adolescence, (b) examine the temporal associations among co-occurring syndromes, and (c) determine the number of higher order factors needed to adequately describe the relations among the first-order growth factors. To address these aims, we used latent growth curve models (LGMs). Given that our sample of girls ranged from 13 to 15 years of age at the first of four annual assessments, we used an age-based analytic approach (Mehta & West, 2000) in which we fit growth curves in each symptom domain that spanned a 5-year period of adolescence from age 13 to 18.

Single and Co-Occurring Syndromes Over Time

Depression

Depression is the most prevalent psychiatric problem for female adolescents, with nearly 20% experiencing clinically significant

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depressive symptomatology during the teenage years (Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn et al., 2003). In terms of the developmental course, studies suggest that a significant proportion of female adolescents will show increasing levels of depressive symptoms over time (Garber, Keiley, & Martin, 2002; Lewinsohn et al., 2003). Although there is some variation in the symptoms of depression that occur over time (Angst, Merikangas, & Preisig, 1997), severity levels have been shown to increase during adolescence and early adulthood (Lewinsohn et al., 2003).

In terms of comorbidity, girls with depression frequently experience elevated levels of anxiety, conduct, substance abuse, and eating disorder symptomatology (Angold et al., 1999; Kessler et al., 2001; Rohde, Lewinsohn, & Seeley, 1991; Stice, Presnell, & Bearman, 2001). Research investigating the temporal relations between symptoms of depression and co-occurring problems suggests that depressive symptoms tend to follow the onset of other mood-related problems (Avenevoli, Stolar, Li, Dierker, & Ries Merikangas, 2001) as well as conduct problems (Rohde et al., 1991; Silberg, Rutter, D'Onofrio, & Eaves, 2003). These findings have led to speculation that depressive comorbidity is either a nonspecific reflection of general psychopathology or a reaction to the failures in adaptation created by earlier behavior problems (Capaldi & Stoolmiller, 1999). However, there is also evidence that elevated depressive symptoms predict future onset of other disturbances, including eating pathology (Stice, Burton, & Shaw, 2004; Stice, Presnell, & Spangler, 2002). Because prospective comorbidity research is sparse, it is not clear how the associations between depressive symptoms and other symptom areas change over time or how the reciprocal influences among co-occurring symptom domains become established.

Eating Problems

Eating pathology is another common psychiatric problem faced primarily by girls (Lewinsohn, Striegel-Moore, & Seeley, 2000; Wilson, Becker, & Heffernan, 2003), and middle to late adolescence represents the period of greatest risk for onset of these problems (Lewinsohn et al., 2000; Stice, Killen, et al., 1998). The trajectories of eating problems appear highly variable and are defined by considerable instability (Stice et al., 2002). Many female adolescents will recover after initial elevations in eating disorder symptomatology, whereas others will manifest patterns of relapse and recovery (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). Much remains to be understood about the prospective nature of eating pathology, particularly eating disorder symptoms that co-occur with other symptom domains.

Eating problems show high rates of co-occurrence with substance abuse (Dansky, Brewerton, & Kilpatrick, 2000; Strober, Freeman, Bower, & Rigali, 1996) and depressive symptoms (Lewinsohn et al., 2000; Stice et al., 2001). However, understanding the processes that give rise to these comorbidities as well as clarity about their temporal interplay remains tentative. For example, eating disorder and depressive symptomatology appear to covary over time (Stice et al., 2001); however, the origin and the course of this covariation are unclear. As with any pair of syndromes, eating disorder and depressive symptomatology may covary as a function of shared risk, a direct causal association (or reciprocal causality), or a common underlying syndrome of psychopathology (e.g., similar cognitive biases; Fergusson, Lynskey, & Horwood, 1996; Zoccolillo, 1992).

Antisocial Behavior

Although preadolescent conduct problems occur more frequently in boys than in girls (Moffitt & Caspi, 2001), the prevalence of conduct problems is similar in male and female adolescents (Hinshaw & Lee, 2003; Moffitt, Caspi, Harrington, & Milne, 2002). The developmental trajectories of girls' conduct problems have received little attention. One exception found that girls (and boys) showed declining levels of externalizing symptoms during adolescence (Bongers, Koot, van der Ende, & Verhulst, 2003). However, there is evidence that girls' (and boys') conduct problems tend to increase over time when they co-occur with depression (Beyers & Loeber, 2003; Silberg et al., 2003). Indeed, the developmental course of antisocial behavior across adolescence appears to be significantly intertwined with co-occurring forms of psychopathology, particularly depression and substance abuse symptoms (Angold et al., 1999; Loeber, Stouthamer-Loeber, & White, 1999). Among girls, however, little is known about the temporal relations among antisocial behavior and co-occurring symptomatology (Hinshaw & Lee, 2003).

Substance Abuse Problems

Substance abuse also typically emerges during adolescence (O'Malley, Johnston, Bachman, & Schulenberg, 2000). Although there are some gender differences in the substances used (e.g., male adolescents use and abuse alcohol more than do female adolescents, whereas female adolescents favor stimulants; Chassin, Ritter, Trim, & King, 2003), epidemiological studies suggest that substance abuse symptoms increase steadily across adolescence in female adolescents (Johnson, Cohen, Kotler, Kasen, & Brook, 2002).

Longitudinal studies indicate that the developmental trajectory of substance abuse symptoms depends largely on the age of onset. Early onset of substance abuse (e.g., before age 15) is associated with a stable and escalating course of abuse for girls (Chassin, Pitts, & Prost, 2002; Nagin & Tremblay, 2001). Investigations of substance abuse trajectories that consider other forms of psychopathology find that female substance abuse development is interwoven with antisocial, depressive, and eating disorder symptomatology (Angold et al., 1999). Prospective studies indicate that substance abuse symptoms typically follow onset of other disturbances, especially antisocial symptoms (Brook, Cohen, & Brook, 1998). Rohde, Lewinsohn, and Seeley (1996), however, found that alcohol abuse preceded future depression in female adolescents. In general, a lack of prospective work on female adolescents leaves open the question of temporal sequencing with certain substance abuse comorbidities.

Overview of Study

The goal of this study was to examine the codevelopment of depression, eating disorder, antisocial, and substance abuse symptoms in a community sample of female adolescents who varied in age from 13 to 15 years at the first of four annual assessments. We used LGMs to address these questions, as it can provide univariate and multivariate estimates of stability and time-related change (T. E. Duncan, Duncan, Strycker, Li, & Alpert, 1999; Willett & Sayer, 1994). Our use of an age-based analytic approach (Mehta &

West, 2000) enabled us to generate growth curves in each symptom domain that spanned a 5-year period of adolescence from age 13 to 18. We focused on female adolescents because these data were drawn from a longitudinal study of the risk factors for eating pathology, which predominantly affects girls. We felt this was an appropriate sample because girls often experience symptomatic increases in depression (Hankin et al., 1998), substance abuse (Langerbucher & Chung, 1995), and eating problems (Stice, Barrera, & Chassin, 1998) during adolescence. Antisocial behavior may also be more common in female adolescents than was previously thought (Cote, Zoccolillo, Tremblay, Nagin, & Vitaro, 2001).

Our first objective was to characterize the developmental trajectory of each domain as well as to evaluate the amount of sample- and individual-level variability in the initial levels and rates of change in each symptom domain. Although there have been longitudinal investigations of the trajectories of psychopathology in samples of male adolescents, little is known about the trajectories of female adolescents' symptomatology. Of interest was whether girls' symptom trajectories changed in a linear (constant) or nonlinear fashion across a 5-year span of adolescence.

Our second objective was to examine the temporal associations among symptom dimensions to better understand whether initial elevations in one symptom domain predicted future increases in other symptom domains. The use of a multivariate LGM (T. E. Duncan et al., 1999) that examined the associations among growth parameters enabled us to test processes that might influence the expression of symptom co-occurrence over time. Initial levels and growth of two syndromes may be related with neither predicting future changes in the other (unrelated coexistence), initial levels of one syndrome may predict future changes in the other (unidirectional effect), or initial levels of both syndromes may predict future changes in the other (reciprocal effects; Caron & Rutter, 1991). It is important to investigate such alternative explanations of symptom co-occurrence because each has distinct etiological, prevention, and treatment implications. For example, if substance abuse is a risk factor for depression, but not vice versa, prevention programs would need to target the former to effectively reduce risk for both conditions.

Our third objective was to determine the number of second-order factors needed to account for the variance in and covariation among first-order intercept and growth factors. The general notion of a second-order factor model is one familiar to researchers using oblique confirmatory factor analysis, in which the question arises as to the source of the obliqueness among first-order factors (Hancock, Kuo, & Lawrence, 2001). One explanation of comorbidity is that it constitutes an undifferentiated accumulation of distress (Krueger, 1999; Lilienfeld, 2003). If a single higher order growth factor could adequately characterize the temporal covariation among girls' depressive, eating disorder, antisocial, and substance abuse symptoms, this might be interpreted as support for a core or common factor explanation of symptom co-occurrence. Evidence of a core factor might also suggest that strong diagnostic lines separating common syndromes might be developmentally premature during adolescence. Alternatively, a core factor might suggest the presence of common risk, such as shared genetic risk (Silberg et al., 2003) or a core type of temperamental vulnerability (Krueger, Caspi, Moffitt, & Silva, 1998).

Studies with adults suggest that more than one higher order factor is needed to account for the covariation among common domains of psychopathology (Krueger, 1999; Vollebergh et al., 2001). By extension, more than one set of higher order growth factors may be needed to represent patterns of codevelopment among depression, eating disorder, antisocial, and substance use symptomatology. Our a priori expectation was that two higher order factors would be necessary, one to account for the codevelopment of depressive and eating disorder symptomatology and another to account for the codevelopment of antisocial and substance abuse symptoms. Theories of affect disturbance and mood regulation often link depressive symptomatology and eating pathology (Stice et al., 2004), whereas models of behavioral disinhibition and poor impulse control often link antisocial behavior and substance abuse problems (Pennington, 2002). Support for a two-factor higher order model might help to elucidate similarities and differences among symptom domains. An improved understanding of the latent structure underlying co-occurring symptom domains might eventually help identify shared versus specific risk factors (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998; Silberg et al., 2003).

Method

Participants

Participants were 496 female adolescents who were assessed annually over a 5-year period (Time 1–Time 5 [T1–T5]). Because antisocial behavior was not assessed at T1, only data from T2, T3, T4, and T5 were used in the present report. This study had a low attrition rate and little missing data. Of the original 496 female adolescents who started the study at T1, 493 had usable data at T2–T5, and of these, the amount of missing data ranged from 1.4% to 9.7%, depending on the variable.[1] The time between assessments was approximately 1 year ($M = 376$ days, $SD = 6$ days), with 80% of the sample completing each assessment within ± 15 days of their prior assessment and the remaining girls within ± 30 days of their prior assessment.

At T2, there was age heterogeneity in the sample as girls ranged in age from 12 to 15 years ($M = 14.48$, $SD = 0.67$). To conduct an age-based analysis (following procedures outlined by Mehta & West, 2000), we first rounded participants' age to the nearest whole year. Five girls were within 2 months of 15½ years; given the instability associated with estimating trajectory means for just five 16-year-olds, the age of these girls was rounded to 15. This resulted in a sample comprising 123, 243, and 127 thirteen-, fourteen-, and fifteen-year-olds, respectively, at T2. Participants were from four public (82%) and four private (18%) schools in a metropolitan area of the southwestern United States. The sample included 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Latinas, 1% Native Americans, and 4% who specified "other" or "mixed" racial heritage, which was representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islanders, 8% African Americans, 65% Caucasians, 21% Hispanics, 4% "other" or "mixed"). Average educational attainment of parents (29% high school graduate or

[1] Mplus draws on the theory of Little and Rubin (1987; expectation/maximization algorithm) to allow for the inclusion of respondents whose data appear to be missing at random. Through the use of maximum-likelihood estimation, the actual data are sorted into missing and nonmissing patterns. Mplus then estimates a covariance matrix from the available raw data and a second coverage matrix in which missing data are held constant to correspond with the maximum-likelihood estimation of that portion of the nonmissing model (Muthén & Muthén, 2001). Thus, while not imputing new data, Mplus estimates latent models by using the maximum-likelihood estimation of the coverage matrix.

less, 23% some college, 33% college graduate, 15% graduate degree) was also similar to census data for comparably aged adults (34% high school graduate or less, 25% some college, 26% college graduate, 15% graduate degree).

Procedure

The study was described to parents and participants as an investigation of adolescent mental and physical health. An active parental consent procedure was used to recruit participants, in which an informed-consent letter and a stamped self-addressed return envelope were sent to parents of eligible girls (a second mailing was sent to nonresponders after 2 weeks). Adolescent assent was secured immediately before data collection took place. This procedure resulted in an average participation rate of 56%. Although lower than we hoped for, this participation rate was similar to that of other school-recruited samples that used active consent procedures and involved structured interviews (e.g., 61%; Lewinsohn, Hops, Roberts, & Seeley, 1993). Furthermore, two pieces of evidence suggest that our sample was representative. First, as just described, the ethnic composition and parental education of the sample were comparable to the ethnic composition of the schools from which we sampled. Second, the 1-year prevalence rates of major depression (4.2%), bulimia nervosa (0.4%), and substance abuse (8.9%; Stice et al., 2001) were similar to the prevalence rates from other epidemiological studies (Lewinsohn et al., 1993).

Girls completed a questionnaire, participated in a structured interview, and had their weight and height measured by female research assistants at all assessments. Female assessors with a bachelor's, master's, or doctoral degree in psychology conducted all interviews. Assessors attended 24 hr of training, in which they learned interview skills, reviewed diagnostic criteria for relevant disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; *DSM–IV*; American Psychiatric Association, 1994), observed simulated interviews, and role-played interviews. Assessors had to demonstrate an interrater agreement for diagnoses ($\kappa > .80$) with experts using tape-recorded interviews before collecting data. Interviewers were recorded periodically throughout the study to ensure that assessors continued to demonstrate acceptable interrater agreement ($\kappa > .80$). Assessments took place during regular school hours or immediately after school on the school campus or at participants' houses. Girls received a gift certificate or cash for completing each assessment.

Measures

Depressive symptoms. An adapted version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich, 1982), a structured psychiatric interview, was used to assess diagnostic criteria for *DSM–IV* major depression. Severity ratings for each symptom were averaged to form a continuous depressive symptom composite ($\alpha = .85$ at T2), ranging from 1 (*never*) to 4 (*always*). The K-SADS generally has good test–retest reliability ($\kappa = .63$–1.00), interrater reliability ($\kappa = .73$–1.00), and internal consistency ($\alpha = .68$–$.84$) and discriminates between depressed and nondepressed individuals (Lewinsohn et al., 1993). The K-SADS depression diagnoses have shown excellent interrater agreement ($\kappa = 1.00$) and 1-week test–retest reliability ($\kappa = 1.00$) in the present study (Stice et al., 2004).

Eating pathology. The Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured psychiatric interview, was used to assess diagnostic criteria for *DSM–IV* bulimia nervosa, anorexia nervosa, and binge eating disorder. Diagnostic items were averaged to form an overall eating symptom composite at each time point ($\alpha = .96$ at T2), ranging from 0 (*never*) to 2 (*always*). The EDE generally has good internal consistency ($\alpha = .76$–$.90$) and interrater reliability ($\kappa = .70$–$.99$) and discriminates between individuals with eating disorders and controls (Fairburn & Cooper, 1993; Williamson, Anderson, Jackman, & Jackson, 1995). The EDE eating disorder diagnoses have shown excellent interrater agreement ($\kappa = .88$) and 1-week test–retest reliability ($\kappa = 1.00$) in the present study (Stice et al., 2004).

Antisocial behavior. Girls' antisocial behavior symptoms were assessed with 13 items from the Externalizing Syndrome of the Child Behavior Checklist (CBCL), 5 of which came from the CBCL's Delinquency subscale and 8 of which came from the CBCL's Aggression subscale (Achenbach & Edelbrock, 1983). In the present study, the response scale was expanded to a 5-point format, ranging from 1 (*never*) to 5 (*always*), to increase variance. Severity ratings for all 13 symptoms were averaged to form a continuously measured antisocial behavior composite ($\alpha = .91$ at T2). This symptom composite evidenced internal consistency ($\alpha = .88$), 1-year test–retest reliability ($r = .62$), and predictive validity in a prior study (Stice, Barrera, & Chassin, 1998).

Substance abuse. Items adapted from Stice, Barrera, & Chassin (1998) assessed *DSM–IV* substance abuse symptoms over the past year. These items were specifically developed to assess substance abuse in adolescents. Items were averaged to create a substance abuse symptom composite at each time point ($\alpha = .86$ at T2), ranging from 0 (*never*) to 2 (*twice or more*). Prior work with this measure indicated that these items possess adequate internal consistency ($\alpha = .85$) and convergent validity (Stice, Barrera, Chassin, 1998; Stice et al., 2001). Pilot testing ($N = 62$) revealed a 1-month test–retest coefficient of .78 for the substance abuse symptom composite. More generally, self-reports of substance abuse appear to be the most valid measure of substance abuse (Winters, Stinchfield, Henly, & Schwartz, 1991).

Statistical Analyses

We used Mplus (Version 2.13, Muthén & Muthén, 2001) to fit LGMs using maximum-likelihood estimation procedures. For our purposes, Mplus was an appropriate option as it allowed for the estimation of growth parameters in the presence of age heterogeneity by using the missing-data approach in which the outcome at each distinct age in the data is read in as a separate variable, and each individual is assumed to have missing data at ages for which she did not provide data (Mehta & West, 2000; Muthén & Muthén, 2001). A common LGM is then applied using these individual growth curves. Following procedures outlined by Mehta and West (2000), we estimated the growth parameters in our LGMs with age scaled to age 13, the youngest value at our first wave of data.

We took an empirical approach to testing the error variance in each LGM. Because a model with homoscedastic variance (invariant across time points) is nested within a model with heteroscedastic variance (varying across time points), we compared the fit of each to see which was more tenable. Our initial expectation was that different error variances would be needed because of age-related differences in measurement error as well as the possible influences of actual age-specific events on girls' reporting, which may have a greater impact at some ages than others. Additionally, we tested to see whether allowing for covarying errors, specifically autocorrelations among adjacent observed indicators, would be necessary. If such effects were significant, they might be said to represent the effects of symptomatology at one point in time on the next, independent of the developmental effects represented by the latent growth factors.

We assessed model fit with multiple criteria as outlined by Hu and Bentler (1999): the comparative fit index (CFI $> .95$), the Tucker-Lewis index (TLI $> .95$), and the root-mean-square error of approximation (RMSEA $< .06$) and its 90% confidence interval (CI). Although reported, nonsignificant chi-square values would be unlikely given the sample size and are thus of little value in evaluating model fit (T. E. Duncan et al., 1999). To compare the fit of competing higher order models, we examined improvements in the chi-square coefficients by using a nested chi-square test (Stoolmiller, 1998), Akaike's (1987) information criterion, and Bozdogan's (1987) consistent version of this statistic, all of which are parsimony-based indices intended for model comparison, not fit evaluation.

To test the form of growth in each symptom domain, we initially hypothesized that there would be significant linear change from age 13 to 18. To identify the linear growth model, the slope loadings were fixed at 0 (age 13), 1 (age 14), 2 (age 15), 3 (age 16), 4 (age 17), and 5 (age 18) to reflect a constant rate of change between each age. However, individuals may not change in a linear fashion alone, and change (accelerations or decelerations) in each symptom domain may occur at different rates at different points of development (Anderson, 1993). Thus, to test the adequacy of the linear hypothesis, we also tested unspecified models within each symptom domain (Anderson, 1993; S. C. Duncan & Duncan, 1996; McArdle & Anderson, 1990). These unspecified models allowed us to evaluate nonlinearities by freely estimating portions of each developmental trajectory (see McArdle & Hamagami, 1991).

Once we could reasonably characterize the shape of each developmental trajectory—be it linear or nonlinear—we examined the associations among symptom domains. To do so, we tested an associative LGM in which multivariate relationships between symptom growth parameters were evaluated. A reparameterization of this model enabled us to test prospective associations among symptom domains. To achieve the latter, the slope factor for each symptom domain was regressed on its own and the three other intercept factors.

Finally, we estimated competing higher order LGMs to examine the degree to which the relations among the primary, first-order growth factors could be described by one or more higher order latent growth constructs. The higher order model follows a structure that is similar to the first-order associative LGM; however, the covariances among the first-order factors are thought to be explained by the higher order factors (S. C. Duncan & Duncan, 1996). It is important to note that even if the higher order model is able to account for all of the covariation among the first-order factors, the goodness-of-fit indices cannot improve over those of the corresponding first-order model. Nonetheless, if the fit indices for the second-order model approach those of the corresponding first-order model, then the hierarchical model can be considered appropriate (S. C. Duncan & Duncan, 1996; Marsh, 1985).

Our first higher order LGM reflected the hypothesis that a single pair of growth factors (intercept and slope estimates) would best account for the developmental associations among all four symptom domains. A second competing model reflected our a priori expectation that separate pairs of growth parameters would be needed to model the variance in and covariation among the first-order growth factors of each symptom domain. In particular, we expected that separate internalizing and externalizing developmental growth factors would best account for the codevelopment of depression and eating pathology on the one hand and antisocial behavior and substance abuse on the other hand.

Table 1
Descriptive Statistics and Intercorrelations for Female Adolescents' Depressive, Eating Disorder, Antisocial Behavior, and Substance Abuse Symptoms

Symptom and age	*M*	*SD*	Mean
Depression			
Age 13	1.30	0.30	
Age 14	1.35	0.35	
Age 15	1.39	0.40	
Age 16	1.40	0.41	
Age 17	1.41	0.42	
Age 18	1.41	0.44	
Stability			.49
Intercorrelation			.33
Eating disorder			
Age 13	0.41	0.37	
Age 14	0.55	0.41	
Age 15	0.59	0.37	
Age 16	0.61	0.36	
Age 17	0.63	0.32	
Age 18	0.64	0.41	
Stability			.51
Intercorrelation			.29
Antisocial behavior			
Age 13	1.62	0.54	
Age 14	1.64	0.54	
Age 15	1.63	0.56	
Age 16	1.61	0.53	
Age 17	1.56	0.48	
Age 18	1.50	0.44	
Stability			.58
Intercorrelation			.34
Substance abuse			
Age 13	0.07	0.22	
Age 14	0.07	0.25	
Age 15	0.13	0.28	
Age 16	0.14	0.30	
Age 17	0.12	0.29	
Age 18	0.12	0.28	
Stability			.50
Intercorrelation			.28

Note. Correlations greater than .15 are significant at $p < .01$. Stability mean = average (Fisher *r*-to-*z* to *r* transformation) of correlations between symptom scores that are adjacent in years. Intercorrelation mean = average (Fisher *r*-to-*z* to *r* transformation) intercorrelation between domains.

Results

Preliminary Analyses

The means and standard deviations for each symptom domain are presented in Table 1 for all of the ages covered in the age-based analysis. Girls' depression, eating disorder symptoms, and substance abuse scores increased on average, whereas girls' antisocial behavior decreased on average. Although the observed mean changes from age 13 to 18 for girls' depressive (Cohen's $d = .29$), eating disorder (Cohen's $d = .59$), antisocial (Cohen's $d = -.24$), and substance abuse (Cohen's $d = .24$) symptoms represent small to medium effect sizes (Cohen, 1988), information about sample averages is likely to mask evidence of meaningful individual variability in terms of initial levels (intercept) and symptom trajectories. The statistical significance of this variability is addressed below in the univariate LGM analyses.[2]

Table 1 also presents mean stability data as well as an estimate of the average level of intercorrelation among symptom domains. Girls' reports within each symptom domain demonstrated moderate but significant levels of 1-year stability. The coefficients in Table 1 also show that there were significant levels of association

[2] We also examined the extent to which mean-level changes masked clinically meaningful change. For these analyses we used dichotomous scores that classified girls as either below or above subthreshold or threshold levels of symptomatology. Of the 435 girls who were not clinically depressed at T2, 14% showed onset of subthreshold or threshold major depression by T5. Of the 480 girls without self-reported eating problems at T2, 9% showed onset of subthreshold or threshold eating pathology by T5. Of the 466 nonantisocial girls at T2, 11% showed onset of diagnostically relevant antisocial behavior by T5. Alternatively, despite the downward mean trajectory, 36% of the girls reporting clinical elevations in antisocial problems at T2 were still above threshold by T5. Of the 461 nonsubstance-abusing girls at T2, 8% showed onset of substance use by T5. These data highlight the changing nature of many girls' clinical status, with a meaningful number of girls worsening over time.

between domains, with higher levels in one domain associated with higher levels in each of the other symptom domains.

Univariate Symptom Trajectories

Our first goal was to characterize the developmental trajectory of each symptom domain. Within each domain, we tested a linear growth model first followed by a nonlinear growth model. To identify our linear model, each slope factor was scaled to reflect constant change between each age (0, 1, 2, 3, 4, 5). To test for the possibility of nonlinear change, we relaxed the constraints on linear growth by freeing all but the first and last loadings on the slope factor. For identification purposes, at least two loadings must be fixed when testing nonlinear growth (S. C. Duncan & Duncan, 1996; McArdle & Anderson, 1990; Meredith & Tisak, 1990; Stoolmiller, 1998). When there are enough points in time to freely estimate factor loadings beyond the two required for identification purposes, the slope factor now represents individual differences in both general trend (up and down) and nonlinear shape and, thus, is better labeled as a *slope/shape factor* (S. C. Duncan & Duncan, 1996; Stoolmiller, 1998). Additionally, because the linear model is a nested case of the less restrictive unspecified model, a nested chi-square difference test can be used to compare the relative adequacy of each model's capacity to account for girls' symptom development (T. E. Duncan, Duncan, & Stoolmiller, 1994).

The results of our preliminary error testing procedures indicated that allowing the residual variances in each symptom domain to vary from age to age would yield significantly better fitting models than models in which the residual variance was constrained to be invariant across ages. Additionally, there was only inconsistent evidence of the need to allow for covarying error terms. As such, in the LGM results that follow, models comprised both heteroscedastic error variance and unrelated error terms.

The overall fit indices for depression suggested that the linear model fit the data reasonably well, $\chi^2(13, N = 487) = 32.20$, $p = .001$; CFI = .97; TLI = .97; RMSEA = .04 (90% CI = .03, .07). In contrast, the unspecified model did not fit the data as well, $\chi^2(9, N = 487) = 24.82$, $p = .00$; CFI = .94; TLI = .93; RMSEA = .06 (90% CI = .03, .10). By freeing three slope parameters, the chi-square statistic was reduced by just 7.38 ($df = 4$, $p > .05$), indicating that the unspecified model did not offer a statistically better fit than the linear model.

The linear model for adolescents' eating disorder symptoms provided a good fit for the data, $\chi^2(13, N = 487) = 22.84$, $p = .03$; CFI = .98; TLI = .98; RMSEA = .04 (90% CI = .01, .07). The unspecified model of girls' eating symptomatology also provided a tenable fit, $\chi^2(9, N = 487) = 17.28$, $p = .001$; CFI = .99; TLI = .98; RMSEA = .05 (90% CI = .01, .07). However, the chi-square difference test was not significant (22.84–17.28 = 5.56, $df = 4$, $p > .05$), indicating that the unspecified model did not offer a superior fit.

The linear model for girls' antisocial behavior provided a good fit for the data, $\chi^2(13, N = 485) = 11.60$, $p = .17$; CFI = .99; TLI = .99; RMSEA = .02 (90% CI = .00, .06). However, the unspecified model of girls' antisocial behavior appeared to improve the fit, $\chi^2(9, N = 485) = 2.23$, $p = .52$; CFI = 1.00; TLI = 1.00; RMSEA = .00 (90% CI = .00, .06). Indeed, the unspecified antisocial model represented a significantly better fit over the linear model (11.60–2.23 = 9.37, $df = 4$, $p < .05$).

The linear model for girls' substance abuse symptoms fit the data reasonably well, $\chi^2(13, N = 487) = 15.78$, $p = .05$; CFI = .99; TLI = .98; RMSEA = .04 (90% CI = .01, .07). The unspecified model of girls' substance abuse symptomatology also fit the data well, $\chi^2(9, N = 487) = 5.24$, $p = .26$; CFI = .99; TLI = .99; RMSEA = .02 (90% CI = .00, .07) and represented a significantly better fit over the initial linear model (15.78 – 5.24 = 10.54, $df = 4$, $p < .05$).

In sum, these results indicate that it is reasonable to characterize girls' reports of their depressive and eating disorder symptomatology as growing in a linear fashion across the 5-year period from age 13 to 18. In contrast, it is more appropriate to characterize the development of girls' antisocial and substance abuse symptomatology as nonlinear, with antisocial behavior decreasing over time and substance abuse symptoms increasing over time. For each of our best fitting models, we present intercept and slope means and variances, residual variances, and factor intercorrelations in Table 2.

The intercept means were significantly different from zero in each symptom domain. In absolute terms, however, the intercept means were indicative of mild levels of symptomatology for the sample at age 13. Additionally, the variance around each mean was also significant, indicating that there was substantial variation in girls' initial status in each domain. Girls' depressive, eating disorder, and substance abuse symptoms increased significantly over time, as evidenced by the positive slope means, whereas girls' antisocial behavior decreased significantly over time. The variance around each of the slope means was also significant, indicating considerable variability in girls' symptom trajectories over time.

Correlations between symptom intercept and slope factors were significant and negative. With increasing means, as was the case with girls' depressive, eating disorder, and substance abuse symptoms, the negative correlation between intercept and slope indicated that girls reporting higher initial levels were more likely to show slower rates of growth over time than those with lower initial levels. With decreasing means, as was the case with girls' antisocial symptom trajectory, a negative correlation between intercept and slope indicated that girls reporting higher initial levels were more likely to decrease at faster rates than girls reporting lower initial levels.[3]

Finally, for each of our best fitting univariate models, we plotted the estimated slope factor loadings. The relative size of each factor

[3] Several factors might explain the negative intercept–slope correlations observed here. First, the correlation between the intercept and slope depends on the time at which initial status is estimated. In growth curve analyses, a different correlation between initial status and change can be obtained depending on the time of initial status (cf. S. C. Duncan & Duncan, 1996; Rogosa, 1988). Second, regression to the mean could produce these negative correlations, in which extremely high (and low) intercept values that are partially a function of measurement error are likely to be closer to the sample mean at repeat assessment (see Marsh, Craven, Hinkley, & Debus, 2003, for a similar suggestion). Third, the negative intercept–slope correlations may be a product of actual developmental deceleration. Girls at the higher levels may have initiated their symptomatic behavior at earlier ages and were either remitting or escalating at slower rates.

Table 2
Parameter Estimates, Standard Errors, and Critical Ratios for Univariate Growth Models

	Linear model results						Nonlinear model results					
	Depression			Eating disorder			Antisocial behavior			Substance abuse		
Parameter	Est.	*SE*	Critical ratio	Est.	*SE*	Critical ratio	Est.	*SE*	Critical ratio	Est.	*SE*	Critical ratio
Factor mean												
INT at age 13	1.32	.02	67.91	0.50	.02	20.94	1.73	.03	51.21	0.12	.01	9.01
Slope	0.03	.01	4.93	0.04	.01	5.32	−0.04	.01	−5.15	0.01	.00	2.49
Factor variance												
INT at age 13	0.10	.01	7.72	0.17	.02	8.99	0.34	.05	7.55	0.04	.01	2.05
Slope	0.01	.00	5.97	0.01	.00	5.44	0.01	.00	3.05	0.02	.00	2.05
Factor correlation: INT with slope	−0.41	.09	−4.29	−0.61	.15	−3.86	−0.55	.13	−4.19	−0.38	.09	−3.85
Residual variance												
Age 13	0.01	.01	1.27	0.06	.01	5.25	0.09	.04	2.31	0.03	.01	3.31
Age 14	0.05	.01	8.42	0.06	.01	7.33	0.09	.03	3.95	0.04	.01	7.25
Age 15	0.07	.01	11.71	0.07	.01	11.26	0.15	.02	9.48	0.05	.00	10.82
Age 16	0.06	.01	10.38	0.05	.01	11.01	0.17	.01	11.47	0.05	.01	9.77
Age 17	0.06	.01	7.68	0.05	.01	7.74	0.08	.02	4.00	0.03	.01	4.86
Age 18	0.09	.02	4.65	0.08	.02	5.13	0.04	.03	1.31	0.01	.02	1.57

Note. Critical ratios of 1.96 indicate estimates are significant at $p < .05$. Est. = unstandardized model estimate, including factor mean, variance, and correlation; *SE* = standard error of each estimated parameter; Critical ratio = estimate divided by the standard error; *z* statistic was used to establish statistical significance of parameter; INT = intercept.

loading reflects the pattern of developmental change across the 5-year span. The graphs for depression, eating pathology, antisocial behavior, and substance abuse are presented in Figure 1. Because the factor loadings for the slope for antisocial behavior were inverted because of our positive scaling of the slope factor, we reestimated the model by using a negatively scaled slope for antisocial behavior. These graphs suggest that girls' depression and eating disorder symptoms grew at fairly constant rates between age 13 and age 18. The graph for girls' antisocial behavior suggests that the trajectory was relatively flat across the first 3 years but then declined more rapidly after age 15. The graph for substance abuse symptoms indicates fairly rapid escalations in self-reported substance abuse between age 13 and age 16, followed by less rapid growth and even decelerations in the level of substance abuse around age 18.

Temporal Relations Between Symptom Domains

To examine the developmental relations among the four symptom domains, we tested an associative LGM in which the growth factors for all four symptom domains were intercorrelated. Additionally, when specifying the antisocial behavior and substance abuse symptom portions of the associative model, we used the actual slope/shape scaling values generated by the unspecified univariate models as start values for their respective slope/shape factors.[4] The associative model fit the data well, $\chi^2(200, N = 483) = 639.59$, $p < .001$; CFI = .95; TLI = .96; RMSEA = .05 (90% CI = .03, .08). Parameter estimates for the associative model are presented in Table 3. Although the results of the associative model are not exactly identical to the univariate LGM results in terms of factor loadings, factor means and variances, and factor correlations, they tend to be very similar (S. C. Duncan & Duncan, 1996). Because this was the case with our data as well, in this section we focus primarily on the correlations between the developmental factors from the different symptom domains. These correlations are presented in Table 3.

All four intercepts were significantly associated with one another; girls with higher initial levels in one domain tended to have higher initial levels in the other domains. All four slope factors were also significantly and positively correlated, indicating, with one exception, that girls showing growth in one domain tended also to show growth in other domains. The exception was the slope correlations involving girls' antisocial trajectories; here, a positive correlation signified that greater increases in depression, eating disorder, and substance abuse symptoms corresponded with less rapid decreases in antisocial behavior on average.

Next we reparameterized the associative model to test whether initial levels in each symptom domain predicted future growth in other symptom domains. To test the unique predictive effects of each symptom domain at baseline, we regressed each slope factor on all four intercepts. As expected, given the significant intercept–slope correlations within symptom domains, initial symptom levels were predictive of significant change ($\beta = -.57$ to $-.33$, $ps < .001$) in the same domain. Consistent with the associations between intercepts and slopes within a given symptom domain, higher baseline levels predicted less rapid symptom change over time. The direction of effect was markedly different when predicting across domains while controlling for the effects of all other

[4] We used the actual slope estimates from the unspecified univariate models as start values in our associative model because we felt that having the same degrees of freedom for each symptom trajectory would allow for a more interpretable test of the temporal associations among symptom domains. This was not a requirement, however, so we also computed the associative model (and second-order models below) using the same unspecified structure modeled in the univariate section. The results from both approaches were essentially identical in terms of absolute and relative fit.

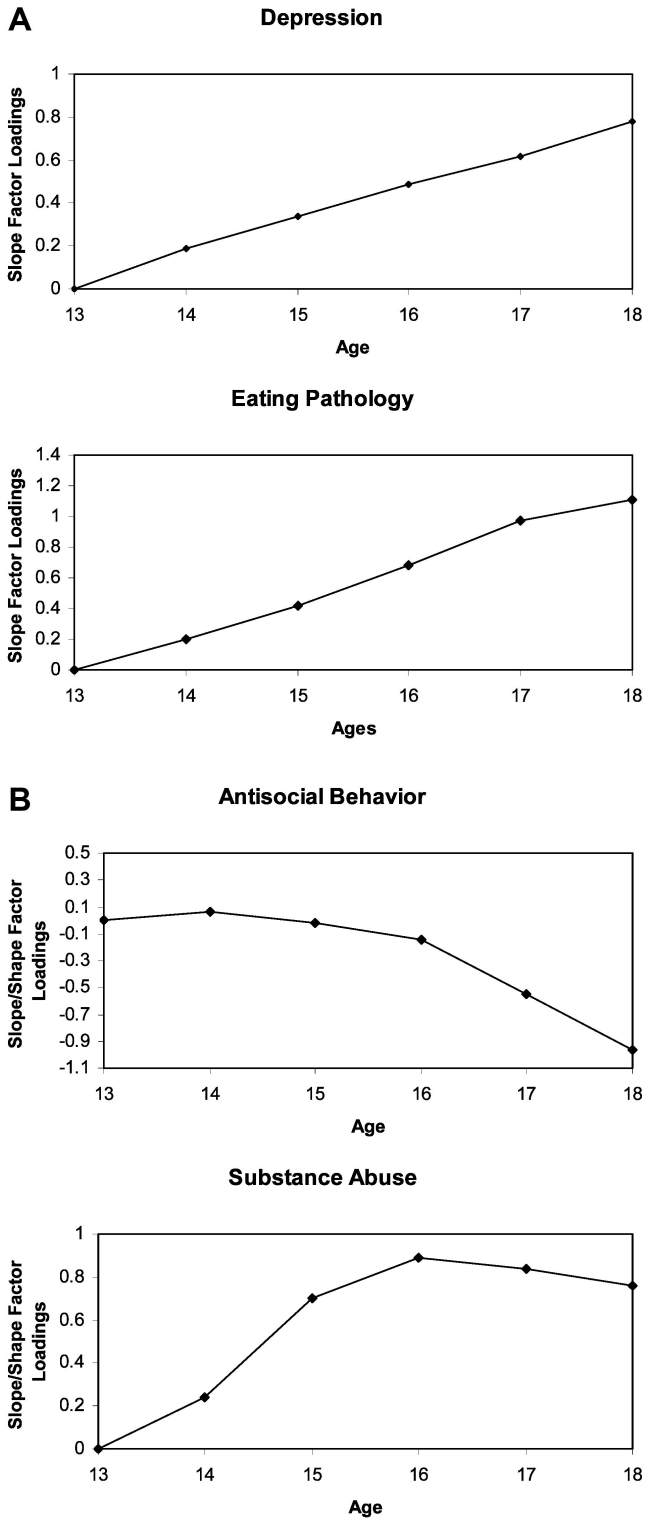

Figure 1. Estimated slope factor loadings from the univariate latent growth models for depression, eating disorder, antisocial, and substance abuse symptoms.

symptom domains. Initial depression level predicted increases in eating pathology ($\beta = .37$, $p < .05$) and substance abuse symptoms ($\beta = .40$, $p < .01$) and slower decreases in antisocial behavior ($\beta = .51$, $p < .01$). Initial eating pathology level predicted increases in substance abuse symptoms ($\beta = .36$, $p < .05$). Initial levels of antisocial symptoms predicted increases in depressive ($\beta = .39$, $p < .05$) and substance abuse ($\beta = .56$, $p < .01$) symptoms. Finally, initial substance abuse level predicted slower decelerations in antisocial behavior ($\beta = .45$, $p < .01$). Thus,

Table 3
Parameter Estimates, Standard Errors, and Critical Ratios for the Associative Growth Model

Parameter	Est.	*SE*	Critical ratio
Factor mean			
Depression intercept	1.32	.02	69.80
Eating disorder intercept	0.51	.03	17.60
Antisocial behavior intercept	1.71	.04	42.82
Substance abuse intercept	0.11	.02	5.87
Depression slope	0.03	.01	4.67
Eating disorder slope	0.04	.01	5.28
Antisocial behavior slope	−0.03	.01	−3.36
Substance abuse slope	0.01	.01	2.34
Factor variance			
Depression intercept	0.10	.01	7.81
Eating disorder intercept	0.17	.02	9.11
Antisocial behavior intercept	0.37	.04	9.29
Substance abuse intercept	0.08	.01	8.51
Depression slope	0.01	.00	6.53
Eating disorder slope	0.01	.01	5.40
Antisocial behavior slope	0.01	.00	4.56
Substance abuse slope	0.02	.00	3.04
Intercept/slope correlation within symptom domain			
Depression	−.42	.08	−5.22
Eating disorder	−.61	.15	−4.07
Antisocial behavior	−.54	.12	−4.23
Substance abuse	−.39	.09	−4.37
Intercept correlation between domains			
Depression WITH			
Eating disorder	.67	.06	10.61
Antisocial behavior	.59	.06	8.52
Substance abuse	.47	.07	6.75
Eating disorder WITH			
Antisocial behavior	.49	.05	9.18
Substance abuse	.28	.06	4.54
Antisocial behavior WITH			
Substance abuse	.67	.07	9.09
Slope correlation between domains			
Depression WITH			
Eating disorder	.52	.07	7.41
Antisocial behavior	.41	.10	4.83
Substance abuse	.39	.09	4.39
Eating disorder WITH			
Antisocial behavior	.37	.08	5.02
Substance abuse	.38	.08	4.75
Antisocial behavior WITH			
Substance abuse	.47	.11	4.32

Note. Critical ratios of 1.96 indicate estimates are significant at $p < .05$. Est. = unstandardized model estimate, including factor mean, variance, and correlation; *SE* = standard error of each estimated parameter; Critical ratio = estimate divided by the standard error; *z* statistic was used to establish statistical significance of parameter; WITH = correlation between factors (e.g., depression intercept and eating disorder slope).

several of the symptom domains acted as unique risk factors for growth in other domains, and patterns of unidirectional and bidirectional prospective effects emerged.

Hierarchical Structure of Co-Occurring Trajectories

Our final goal was to investigate the hierarchical factor structure of the associative model. Initially we fit a single pair of higher order growth parameters to the associative model by using the "factor-of-curve" modeling procedure (McArdle, 1988). In the factor-of-curve model, one examines whether a higher order factor adequately describes relationships among lower order growth factors (T. E. Duncan et al., 1999; McArdle, 1988). As such, the factor-of-curve model is a second-order extension of the associative model, in which the second-order intercept and slope factors are estimated from the first-order univariate latent factors. Thus, each first-order portion of the associative model describes individual differences within each univariate symptom domain, and the second-order common factor model describes the individual differences among the first-order LGMs. To specify the factor-of-curve model, the covariances among first-order latent growth curves' variances are fixed to a value of zero, and factor loadings between the first- and second-order factors are restricted to be equal over time for each symptom domain; this imposes a form of factorial invariance that ensures similar scaling among multiple second-order factor scores (T. E. Duncan et al., 1999; also see McArdle, 1988, for a more formal discussion of the mathematical representation of this model). We used girls' depression as the reference scaling for the single higher order factor model.

The single higher order model fit the data poorly, $\chi^2(221, N = 485) = 906.25$, $p < .001$; CFI = .88; TLI = .89; RMSEA = .11 (90% CI = .08, .13). As such, the data did not support the idea that girls' trajectories in each of the four symptom domains could be explained by a common set of growth factors across these years of adolescence.

We then tested our a priori two-factor higher order model in which we estimated separate intercept and slope factors on the basis of what depression and eating pathology had in common and what antisocial behavior and substance abuse had in common. We used girls' depression and antisocial behavior as the reference scaling for what we labeled the *internalizing* and *externalizing second-order factors*, respectively. The two-factor model fit the data reasonably well, $\chi^2(214, N = 485) = 682.29$, $p < .001$; CFI = .93; TLI = .93; RMSEA = .06 (90% CI = .04, .07).

Factor loadings, intercept and slope means and variances, factor correlations, factor intercepts, and residual variances are presented in Table 4. Parameter estimates for the two-factor model indicated that both intercept factors were significantly different from zero, that the means of both slope factors were also significant, and that the amount of individual variation around both intercept and both slope factors was significant. In addition, all common factor-of-curve loadings were significant. The higher order internalizing intercept factor accounted for approximately 75% and 63% of the variation in the first-order intercepts for depression and eating disorder symptoms, respectively, whereas the externalizing intercept factor accounted for 79% and 95% of the first-order intercepts for antisocial behavior and substance abuse, respectively. Approximately 73% and 93% of the variation in the first-order slope factors for depression and eating disorder symptoms, respectively, and 65% and 59% of the variation in the first-order slope factors for antisocial problems and substance abuse were accounted for by the internalizing and externalizing higher order slope factors, respectively.

Table 4
Parameter Estimates, Standard Errors, and Critical Ratios for the Two-Factor Higher Order Factor-of-Curves Model

Parameter	Est.	*SE*	Critical ratio
Factor loading			
Internalizing INT			
Depression	1.00	—	—
Eating disorder	0.68	0.07	9.71
Internalizing slope			
Depression	1.00	—	—
Eating disorder	0.67	0.09	7.44
Externalizing INT			
Antisocial behavior	1.00	—	—
Substance abuse	1.60	0.15	10.67
Externalizing slope			
Antisocial behavior	1.00	—	—
Substance abuse	1.36	0.22	6.21
Factor mean			
Internalizing INT	1.32	0.02	65.62
Internalizing slope	0.03	0.01	3.22
Externalizing INT	1.72	0.04	43.75
Externalizing slope	−0.03	0.01	−3.41
Factor variance			
Internalizing INT	0.13	0.02	6.71
Internalizing slope	0.01	0.00	5.13
Externalizing INT	0.43	0.06	6.91
Externalizing slope	0.02	0.00	4.80
Factor intracorrelation			
Internalizing	−.67	.06	−10.87
Externalizing	−.59	.11	−5.36
Factor intercorrelation			
INT with INT	.57	.05	9.40
Slope with slope	.41	.09	6.11
Factor intercept			
Depression INT	0.00	—	—
Depression slope	0.00	—	—
Eating disorder INT	−0.43	0.10	−4.53
Eating disorder slope	0.03	0.01	3.17
Antisocial behavior INT	0.00	—	—
Antisocial behavior slope	0.00	—	—
Substance abuse INT	−2.18	0.26	−8.38
Substance abuse slope	0.34	0.03	11.91
Residual variance			
Depression INT	−0.01	0.01	−1.49
Depression slope	0.00	0.00	4.17
Eating disorder INT	0.04	0.01	6.60
Eating disorder slope	0.00	0.00	0.03
Antisocial behavior INT	0.01	0.02	1.14
Antisocial behavior slope	−0.00	0.00	−1.47
Substance abuse INT	0.56	0.08	6.83
Substance abuse slope	0.04	0.01	5.59

Note. Critical ratios of 1.96 indicate estimates are significant at $p < .05$. Internalizing factor comprises depression and eating disorder symptoms. Externalizing factor comprises antisocial behavior and substance abuse symptoms. Dashes indicate data are nonapplicable because estimates were fixed to scale the higher order factors. Est. = unstandardized model estimate, including factor mean, variance, and correlation; *SE* = standard error of each estimated parameter; Critical ratio = estimate divided by the standard error; *z* statistic was used to establish statistical significance of parameter; INT = intercept.

Finally, the internalizing and externalizing intercept factors were positively and significantly correlated ($r = .57$, $p < .05$), as were the two slope factors ($r = .41$, $p < .01$). Thus, girls with higher initial internalizing symptom scores had elevated externalizing symptom scores as well. In light of the negative externalizing slope, growth in internalizing was significantly associated with slower declines in externalizing problems.

On the whole, it might appear odd that the same set of higher order growth factors could account for the first-order variation in girls' antisocial and substance abuse symptoms given that the respective aggregate trends were changing in opposite directions. However, it should be remembered that there was significant variation in the slopes for substance abuse and antisocial behavior, with some girls likely showing increases and some showing decreases in both symptom domains. In an attempt to interpret this finding, we used the estimated factor loadings at each age to plot each girl's antisocial behavior and substance abuse trajectories; we then examined the frequency with which girls' symptom development in these two domains moved in similar or different directions. We found that 31% of the sample had downward antisocial behavior trajectories and upward substance abuse trajectories (no case of the opposite was found), 44% of the sample had synchronous antisocial behavior and substance abuse trajectories (e.g., both increased or decreased), and 25% had one stable trajectory and a second trajectory that moved either upward or downward. Thus, by moving from a nomothetic focus on group-level trends to an ideographic focus on individuals' patterns of codevelopment, we can see that there was reasonable synchrony in how individual girls' antisocial behavior and substance abuse symptoms changed from age 13 to 18, despite contradictory sample-level estimates. It is our sense that this developmental synchrony was captured by the second-order slope factor for girls' antisocial behavior and substance abuse symptoms.

To evaluate further the acceptability of this internalizing–externalizing higher order LGM, we tested a series of alternate higher order LGMs. For example, depression, eating disorders, and substance abuse were grouped to reflect the idea that girls with mood problems might rely on two different types of mood regulation strategies. In this particular model, only the first-order growth factors were needed for antisocial behavior. None of our alternate higher order models provided an acceptable accounting of the data.[5]

We also tested a second multivariate approach proposed by McArdle (1988), the "curve-of-factor" model (see also T. E. Duncan et al., 1999). In contrast to the factor-of-curve model tested above, the curve-of-factor model fits a growth curve to factor scores representing what different symptom domains have in common at each time point. The observed variables for all symptom domains at each age are factor analyzed to produce factor scores (e.g., a factor score that comprises what is common to depression, eating disorder, antisocial, and substance abuse symptoms at age 13), which are then used for modeling higher order growth curves (T. E. Duncan et al., 1999; Hancock et al., 2001; McArdle, 1988). The curve-of-factor alternative to our internalizing and externalizing factor-of-curve model provided a poor accounting of the data, $\chi^2(202, N = 485) = 956.58$, $p < .001$; CFI = .84; TLI = .85; RMSEA = .12 (90% CI = .11, .13); consequently, we did not explore other curve-of-factor alternatives.

Discussion

Single-Syndrome Trajectories

The first aim of this study was to generate descriptive data on depressive, eating disorder, antisocial, and substance abuse symptom trajectories in a community sample of female adolescents. Our results suggest that between the ages of 13 and 18, girls' self-reported symptoms of depression and eating disorder symptom trajectories showed evidence of linear growth as well as significant levels of variability around the aggregate trends. The rate of growth in girls' depressive symptoms was significant albeit modest, increasing on average about a quarter of a standard deviation between age 13 and age 18. These results dovetail with other studies that have reported sample-level increases in depressive symptoms in female adolescents (Cole et al., 2002; Hankin et al., 1998).

Girls' eating disorder symptom trajectories also showed significant sample-level growth as well as significant individual variability in the rate of growth. Like the growth in depressive symptoms, the growth in girls' eating disorder symptoms tended to be fairly constant over time. However, in contrast to the depressive symptom trajectory, the rate of growth in girls' eating disorder symptomatology was more pronounced, increasing more than half a standard deviation between ages 13 and 18. To our knowledge, this study and prior investigations of this sample (Stice et al., 2004) may be the first to report normative increases in eating disorder symptoms for female adolescents.

Girls' substance abuse trajectory also increased significantly albeit modestly during this period (e.g., approximately a quarter of a standard deviation between ages 13 and 18). Again, the amount of variability around the sample-level growth trajectory was significant, indicating considerable individual differences in substance abuse progression. In contrast to girls' depressive and eating disorder symptom trajectories, growth in this domain tended to be somewhat uneven or nonlinear. Specifically, the shape of the substance abuse trajectory suggested that girls reported more rapid increases in substance abuse problems between the ages of 13 and 16 but then appeared to begin to decelerate between 16 and 18 years. This pattern of increase in girls' substance abuse symptoms is consistent with other investigations and is likely linked to less restrictive living situations as well as some of the risks associated with greater tolerance of substance use (Chassin & Ritter, 2001; Chen & Kandel, 1995; Hankin et al., 1998; Kandel, Huang, & Davies, 2001; O'Malley et al., 2000). We are less certain about the factors that might account for girls' apparent slowing of substance abuse behavior between ages 16 and 18. Perhaps, this decline can be linked to girls' preparation for their transition to adult roles (e.g., college, marriage, employment), which have been shown to reduce substance abuse symptoms in young adults (Collins & Shirley, 2001).

[5] Additionally, we were concerned that our second-order results might be due to method artifact, namely that the internalizing factor simply represented interview data whereas the externalizing factor represented questionnaire data. Fortunately, we replicated our second-order results when we replaced the K-SAD depression symptom composite with items from Buss and Plomin's (1984) Emotionality Scale, which girls completed in questionnaire form at all assessments to report on their negative affect and mood traits.

As has been found in other prospective studies (Bongers et al., 2003), girls' antisocial behavior showed a downward trajectory across adolescence. However, the rate at which girls' antisocial behavior decreased was fairly modest (e.g., about a quarter of a standard deviation from age 13 to age 18) as well as nonlinear in shape. Specifically, the nonlinear pattern suggests that girls reported fairly constant or static levels of conduct problems until age 15 but that after age 15, girls reported declining levels of antisocial behavior. This nonlinear pattern of antisocial behavior development is suggestive of Moffitt's (1993) theory that early adolescence is defined by normative elevations in conduct problems but that these behaviors begin to desist for most, but not all, adolescents during the later teenage years; as Moffitt (1993) also showed, an important minority of adolescents will persist in their antisocial behavior into adulthood.

Temporal Associations Between Symptom Domains

Our second objective was to examine the prospective relations among co-occurring symptoms. Results suggest that girls' symptoms of depression, eating pathology, antisocial behavior, and substance abuse were significantly correlated with each other concurrently and across time. However, the four symptom domains examined were differentially predictive of each other's growth, suggesting that symptom co-occurrence may arise in part because certain symptom domains are risk factors for other symptom domains. We found a bidirectional association between depressive and antisocial symptoms, with initial levels of each uniquely predicting future change in the other. Specifically, initial levels of antisocial behavior predicted increases in depressive symptoms, whereas initial levels of depressive symptoms predicted slower rates of deceleration in antisocial behavior. The former effect is consonant with prior studies with both female adolescents (Silberg et al., 2003) and male adolescents (Kim, Capaldi, & Stoolmiller, 2003; Rohde et al., 1991). This finding appears to be consistent with a failure model (Capaldi, 1992), in which developmental failures associated with antisocial behavior appear to increase adolescents' vulnerability to symptoms of depression. There is less evidence in the literature of early depressive symptomatology contributing to changes in girls' antisocial behavior. The hopelessness that often characterizes depression may increase the likelihood that girls engage in risky behaviors that typify antisocial behavior (e.g., fighting, theft, and property destruction). In addition, depression may be associated with greater irritability, which increases the likelihood of conflict with parents and other authority figures, which is another common antisocial symptom. As Joiner (2000) suggested, the residue of depression may have implications for subsequent experiences of depressive symptomatology as well as later problems with antisocial behavior.

A unidirectional relation between depression and eating pathology emerged, wherein initial levels of depressive symptoms predicted future growth in eating pathology. This finding is consistent with prior evidence that negative affect predicts future increases in bulimic symptoms, binge eating, and general eating pathology (Johnson et al., 2002; Stice et al., 2002). Thus, findings provide support for the assertion that depression increases the risk that female adolescents will turn to binge eating to provide comfort or distraction from adverse emotions (McCarthy, 1990). Individuals might also use radical compensatory behaviors, such as vomiting, to reduce distress about impending weight gain consequent to overeating or because they believe that purging serves as an emotional catharsis (Stice et al., 2002).

A unidirectional relationship between initial levels of depression and substance abuse growth also emerged. One of the more widely accepted etiologic theories is that affective disturbances increase the risk for onset and maintenance of substance abuse (Cooper, Frone, Russell, & Mudar, 1995; Newcomb & Bentler, 1988; Stasiewicz & Maisto, 1993). Individuals with affective disturbances are thought to consume psychoactive substances because they improve mood and provide distraction from adverse emotions (McCollam, Burish, Maisto, & Sobell, 1980). The fact that mood disturbances seemed to increase during adolescence for girls (Hankin et al., 1998) suggests that negative disturbance might be a particularly strong predictor of substance abuse increase for this population.

Although it is established that the trajectories of antisocial behavior and substance abuse are highly interwoven, most of this research has been conducted with boys (Angold et al., 1999). Our results extend this pattern to girls and also suggest that there is a reciprocal pattern of influence between antisocial behavior and substance abuse. That initial antisocial behavior predicted growth in substance abuse is consistent with data indicating that substance abuse is very rare for those free of conduct problems and may subsequently reflect girls' maladaptive efforts to cope with social disruptions caused by delinquent interpersonal behavior (Capaldi, 1992; Patterson, Reid, & Dishion, 1992). That initial levels of substance abuse predicted slower decelerations in antisocial problems might be due to the fact that substance abuse can increase dysregulation and problems with judgment that enable or sustain antisocial behavior (Brown, Gleghorn, Schuckit, Myers, & Mott, 1996).

Finally, initial eating pathology predicted future growth in substance abuse, but not vice versa. This finding converges with evidence in adulthood of a prospective, unidirectional relationship between earlier eating pathology and subsequent growth in substance abuse (Johnson et al., 2002). Theoretically, the strong drive for thinness that typifies eating pathology may increase the risk for recurrent use of stimulant drugs for the purpose of weight loss, which would increase the risk for the use-related negative consequences. Binge eating and compensatory behaviors may also promote feelings of shame and guilt (Stice et al., 2001), which might lead to attempts to regulate affect through substance use.

In sum, our findings suggest that symptomatic co-occurrence may arise because certain symptom domains are risk factors for other symptom domains. Counterarguments to the idea of true co-occurrence, however, would point to issues of method covariance or to the possibility that the girls in this sample were simply reporting on a single undifferentiated form of psychological distress. This accounting, however, is inconsistent with the heterotypic associations observed (e.g., depression covarying with substance abuse) and the evidence of unidirectional relations. Moreover, it is unlikely that these effects are a product of reporter bias because the multivariate models examined the unique predictive effects of each symptom domain while controlling for the other domains. Within such models, reporter bias would have manifested itself in the associations between intercepts and slopes within the same domain.

Hierarchical Structure of Co-Occurring Trajectories

Our third aim was to determine the number of higher order growth factors needed to explain the variation in and covariation among girls' depression, eating disorder, antisocial, and substance abuse symptoms. Results suggest that a single common higher order factor did not account adequately for the covariation among the first-order symptom growth factors. Had a single higher order dimension provided a tenable accounting of the data, this latent factor might suggest that depression, eating disorder, antisocial, and substance abuse symptoms covary over time because of a core or shared underlying psychological process. Such a broadband factor might be representative of general maladjustment, as has been suggested by personality theorists (Graham, 1993; Tellegen, 1982). However, it could still be that symptom co-occurrence during adolescence is the product of what once was a basic affective or motivational form of maladjustment. Over time, and under the sway of diverse developmental and environmental forces, a core psychological process may branch out into various behavioral outcomes in the same individual. For example, Essex, Klein, Cho, and Kraemer (2003) reported that the timing of exposure to maternal depression determined whether affectively irritable infants developed conduct or mood problems first. In other words, we cannot rule out from these data that the co-occurring syndromes might have once shared a developmental pathogenesis, such as temperamental irritability.

Our results do suggest, however, that between the ages of 13 and 18 years, two higher order factors provided a tenable accounting of the associations among the first-order latent growth factors. Our best fitting model applied one pair of higher order growth factors to the changes in girls' depressive and eating disorder symptoms and a second pair of higher order growth factors to the changes in girls' antisocial and substance abuse symptoms. Despite significant overall increases in the internalizing factor and significant overall decreases in the externalizing factor, both showed significant change over time and were positively correlated. Further, alternate second-order models did not fit the data well. For example, an untenable fit was provided by a model that grouped eating disorder and substance abuse symptoms, reflecting theories of maladaptive approaches to mood regulation, and depression and antisocial symptoms, reflecting Karpman's (1941) notion of neurotic psychopathy. In addition, McArdle's (1988) second approach to a multivariate LGM, the curve-of-factor model, poorly fit these data.

Our second-order results are consistent with Krueger's (1999) analyses of the National Comorbidity Survey as well as Krueger et al.'s (1998) analysis of the Dunedin Multidisciplinary Health and Development Study, in which separate but related internalizing and externalizing higher order factors accounted for a panel of common psychiatric disorders. However, our findings appear to extend Krueger's findings in two important ways. First, the prospective results from our first-order associative LGM demonstrate that there is obvious value to distinguishing between putative domains of psychopathology. That girls' depression, eating disorder, antisocial, and substance abuse symptoms operated differently as risk factors for one another suggests that there may be reliable temporal sequencing of co-occurring forms of psychopathology (Angold et al., 1999). A narrow read of Krueger's (1999) work might have resulted in the conclusion that the higher order accounting of common co-occurring symptomatology is the more important unit of analysis. However, like the series of studies by Krueger and colleagues (Krueger, 1999; Krueger et al., 1998), our second-order findings also call into question the tendency to study syndromes in isolation (see also Clark, Watson, & Reynolds, 1995). The presence of core psychopathological processes that underlie the expression and progression of constellations of different symptom domains seems highly likely and worthy of further study. Moreover, given that we used just four symptom scales, two each for the internalizing and externalizing common factors, the inclusion of more symptom domains may have shed a different, potentially more valid light on the common factors underlying the development and progression of co-occurring problems. For example, including measures of attention-deficit/hyperactivity disorder might have yielded a common factor that replaced what here we called the externalizing factor in favor of a factor that captured the temperamental aspects of disinhibition or inhibitory control deficits more specifically.

Limitations

It is important for one to consider the limitations of this study when interpreting our findings. First, although adolescents appear to be the most valid reporters of much of their own emotional and behavioral difficulties (e.g., Cantwell, Lewinsohn, Rohde, & Seeley, 1997), reporter bias may artificially inflate the magnitude of correlations between constructs. Future studies should collect multiple-reporter data. Second, as just referred to, it would have been desirable if we had collected data on other psychiatric conditions that show high comorbidity, particularly anxiety symptoms. It is possible that our second-order results might have differed had additional symptom domains been included. Third, for a number of girls in the present study, the onset of psychopathology undoubtedly started prior to age 13. Earlier measurement of girls' co-occurring symptoms might reveal a different temporal picture than the one provided herein. Finally, as with all longitudinal data, there is no way to rule out the possibility that some omitted third variable might explain the prospective effects observed in our study (e.g., temperamental emotionality or neurocognitive impairment). Therefore, it would be useful if randomized treatment trials for one symptom domain assess the impact of treatment on other symptom domains.

Nonetheless, the longitudinal nature of this study provides some assurance that the temporal effects and model results are tenable. Confidence can also be placed in these findings given that we used multiple model-fitting strategies and did not engage in post hoc model respecification. Finally, the large and well-retained community-recruited sample augments the generalizability of the results.

Conclusions and Implications

In sum, the present study sheds new light on the development of co-occurring problems in female adolescents. Results suggest that female adolescents' symptoms of depression, eating pathology, antisocial behavior, and substance abuse covary over time and that their co-occurrence may be due partially to the fact that over time certain symptom domains increase the risk of symptom growth in other domains. Finally, there is evidence that growth in depressive

and eating symptoms, on the one hand, and antisocial and substance abuse symptoms, on the other hand, is best represented by separate hierarchical growth factors. Such higher order symptom models may ultimately prove useful in disentangling specific and general risk factors for these co-occurring domains of symptomatology.

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