



2-2018

Core Dimensions of Anxiety and Depression Change Independently During Adolescence

Christopher C. Conway
College of William and Mary, conway@wm.edu

Richard E. Zinbarg

Susan Mineka

Michelle G. Craske

Follow this and additional works at: <https://scholarworks.wm.edu/aspubs>

Recommended Citation

Conway, Christopher C.; Zinbarg, Richard E.; Mineka, Susan; and Craske, Michelle G., Core Dimensions of Anxiety and Depression Change Independently During Adolescence (2018). *JOURNAL OF ABNORMAL PSYCHOLOGY*, 126(2).
10.1037/abn0000222

This Article is brought to you for free and open access by the Arts and Sciences at W&M ScholarWorks. It has been accepted for inclusion in Arts & Sciences Articles by an authorized administrator of W&M ScholarWorks. For more information, please contact scholarworks@wm.edu.



Published in final edited form as:

J Abnorm Psychol. 2017 February ; 126(2): 160–172. doi:10.1037/abn0000222.

Core Dimensions of Anxiety and Depression Change Independently During Adolescence

Christopher C. Conway¹, Richard E. Zinbarg^{2,3}, Susan Mineka², and Michelle G. Craske⁴

¹Department of Psychology, College of William & Mary, Williamsburg, USA

²Department of Psychology, Northwestern University, Evanston, IL, USA

³The Family Institute at Northwestern University, Evanston, IL, USA

⁴Department of Psychology, University of California, Los Angeles, CA, USA

Abstract

The developmental trajectories of emotional disorder symptoms during adolescence remain elusive, owing in part to a shortage of intensive longitudinal data. In the present study, we charted the temporal course of the tripartite model of anxiety and depression—which posits an overarching negative affect dimension and specific anhedonia and anxious arousal dimensions—over adolescence and emerging adulthood in order to construct a developmental map of the core dimensions of emotional disorders. We recruited 604 high school juniors, overselecting those at high risk for emotional disorders, and assessed the tripartite symptom domains five times annually. Latent curve modeling revealed that negative affect and anxious arousal declined over follow up, whereas anhedonia did not. Moreover, the correlation in rate of change varied across pairs of symptom domains. Change in negative affect was moderately correlated with change in anxious arousal, but change in anhedonia was not significantly related to change in any other domain. Symptom trajectories, and the pattern of covariation among trajectories, were equivalent across gender and comorbidity status. We discuss implications of these findings for developmental models of anxiety and depression as well as transdiagnostic frameworks for emotional disorders.

General Scientific Summary

We charted a map of how anxiety and depression grow through adolescence, a period of peak risk for emotional disorders. We found that the core elements of anxiety and depression do not develop at the same rate or even in the same direction, on average. There may thus be multiple pathways to emotional disorder during adolescence.

Keywords

anxiety; depression; latent curve model; tripartite model

Core Dimensions of Anxiety and Depression Change Independently During Adolescence

The trajectories of emotional disorder symptoms are perhaps most unstable in adolescence. Anxiety and depressive symptom levels can change dramatically during this period, as some conditions (e.g., unipolar depression) tend to emerge for the first time and others (e.g., phobias) become entrenched (e.g., Hankin & Abramson, 2001; Lewinsohn, Clarke, Seeley, & Rohde, 1994; Kessler et al. 2005). Developmental trends in these symptom domains defy simple explanations. Not all emotional disorder symptoms change at the same speed, or even in the same direction, during adolescence, despite high rates of diagnostic comorbidity (e.g., McLaughlin & King, 2015). Moreover, there is wide variation around average trends, reflecting a diversity of possible trajectories.

Mapping the arcs of anxiety and depression over adolescence clearly requires intensive longitudinal data. There are surprisingly few prospective studies with a follow up structure suited to examine the rate and shape of symptom change. The available research generally suggests that depressive symptoms increase steadily through adolescence and reach a plateau at the transition to adulthood (Galambos, Barker, & Krahn, 2006; Galambos, Leadbeater, & Barker, 2004; Ge, Lorenz, Conger, Elder, & Simons, 1994; Kim, Capaldi, & Stoolmiller, 2003). However, several studies report zero growth, or even depression symptom improvement, from mid- to late-adolescence (e.g., Galambos, Barker, & Krahn, 2006; Measelle, Stice, & Hogansen, 2006). Anxiety disorder symptoms appear to decline gradually over the second decade of life (McLaughlin & King, 2015; van Oort, Greaves-Lord, Verhulst, Ormel, & Huizink, 2009), although contradictory results have been reported (Leadbeater, Thompson, & Gruppuso, 2012; Olino, Stepp, Keenan, Loeber, & Hipwell, 2014), and trajectories likely vary across the different anxiety disorders (Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2008; van Oort et al., 2009).

A focus on the core dimensions underlying anxiety and depression may provide a more coherent picture of emotional disorder symptom change in adolescence. Negative affect is a symptom dimension common to the full spectrum of anxiety and depressive disorders, and its growth could be responsible for disorders changing together. Structural modeling of the emotional disorder domain shows that disorders also are characterized by more specific (i.e., unique) psychopathological dimensions, and these could account for divergent symptom trajectories (e.g., Krueger, 1999; Markon, 2010; Prenoveau et al., 2010). Traditional assessments of emotional disorders do not adequately distinguish the common versus specific components of anxiety and depression (Clark & Watson, 1991). Therefore, prior longitudinal studies may have overestimated the similarity of emotional disorder trajectories by emphasizing shared symptom dimensions of anxiety and depression (e.g., negative affect).

The tripartite model—along with closely related formulations, such as the integrative hierarchical model (Mineka, Watson, & Clark, 1998) and the trilevel model (Naragon-Gainey, Prenoveau, Brown & Zinbarg, in press; Prenoveau et al., 2010)—of anxiety and depression aims to separate the common and specific symptom dimensions of emotional disorders (Clark & Watson, 1991). This model stipulates that (i) negative affect is common

to all emotional disorders and is a major reason for their comorbidity, (ii) anhedonia (i.e., low positive affect) is more narrowly associated with depression and social anxiety, and (iii) anxious arousal is specifically associated with anxiety (especially panic disorder).

A wealth of cross-sectional research has validated the tripartite configuration in diverse community and clinical samples (e.g., Kotov, Gamez, Schmidt, & Watson, 2010; Watson, Gamez, & Simms, 2005). The dimensions are distinct yet moderately correlated (Naragon-Gainey, Watson, & Markon, 2009; Watson & Naragon-Gainey, 2014; Watson et al., 1995; Zinbarg & Barlow, 1996), explain complex emotional disorder comorbidity patterns (Brown, Chorpita, & Barlow, 1998), and have mostly independent etiologies and correlates (e.g., Watson et al., 1995).

There has been no systematic research, however, on the temporal course of tripartite model dimensions. Therefore, it remains to be seen whether these components of anxiety and depression change at the same rate, or in the same direction, over adolescence and young adulthood. It is possible that the three dimensions represent distinct routes to emotional disorder that follow divergent naturalistic courses. On the other hand, they may converge over time to form one common pathway to disorder.

Although the tripartite model was not originally formulated as a developmental theory, some hypotheses regarding temporal dynamics can be derived. Negative affect is posited as a common thread across emotional disorder symptoms that correlates more highly with the other two tripartite model dimensions than they do with each other, and change in negative affect might therefore be expected to correlate with change in anhedonia and anxious arousal over time. In contrast, anhedonia and anxious arousal are thought to reflect largely independent systems, based on unique neurobiological and psychological underpinnings (e.g., Forbes & Dahl, 2005; Forbes et al., 2009; Klein, Kotov, & Bufferd, 2011; Kotov, Watson, Robles, & Schmidt, 2007). In cross-sectional analyses, these symptom domains exhibit appropriate levels of discriminant validity (Brown et al., 1998; Watson et al., 1995). Therefore, it could be argued that the developmental trajectories of these dimensions should be largely unrelated.

Prior longitudinal research offers little empirical guidance for predictions about correlations among developmental trajectories of the core dimensions of anxiety and depression. In one early adolescent sample, latent curve modeling analyses revealed no significant correlation between change in depressive symptoms and change in generalized, physical, separation, or social anxiety symptoms (McLaughlin & King, 2015). In contrast, in a large sample of Dutch adolescents, the corresponding estimates (r range: .32-.71) were moderate-to-large over five years of follow up (Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2009). Two other longitudinal studies also reported substantial correlations ($r_s \approx .60$) in growth of anxiety and depression (Leadbeater et al., 2012; Olinio et al., 2014). However, it is important to bear in mind that these estimates may be inflated to some extent by negative affect pervading the assessment of anxiety and depressive constructs.

Present Study

In the present study, we charted the growth of basic psychopathological dimensions undergirding anxiety and depressive disorders during adolescence and emerging adulthood. We examined negative affect, anhedonia, and anxious arousal over five annual assessment waves in a high risk sample of high school juniors. We had three research questions. First, what are the direction and rate of symptom change? Second, how much variation exists around the average symptom trajectory, and do the trajectories from different symptom domains covary? Third, do symptom trajectories, and the degree of covariation among those trajectories, differ by gender and comorbidity status?

Two opposing theoretical perspectives led to competing hypotheses regarding the trajectory of mean-level symptom change (Question 1). On one hand, drawing from data on change in personality trait levels over the life course, the maturity principle (Caspi, Roberts, & Shiner, 2005) holds that people tend to become more emotionally stable at the transition to adulthood. That is, negative affect is theorized to wane as people make normative commitments to conventional social roles, such as long-term romantic relationships, lifelong friendships, occupations, and families (e.g., Robins, Caspi, & Moffitt, 2002; Robins, Fraley, Roberts, & Trzesniewski, 2001). Perhaps because entry into many of these stabilizing social institutions occurs in emerging adulthood, the transition to young adulthood is the period of greatest change (i.e., reduction) in trait negative affectivity (see Roberts, Walton, & Viechtbauer, 2006, for a meta-analysis). Therefore, it could be hypothesized that those emotional disorder symptom dimensions most saturated with trait negative affectivity—in the present study, negative affect and anxious arousal (see Mineka et al., 1998)—should show reductions over the present study's timeframe, which spans the transition from adolescence to young adulthood.

On the other hand, the major social transitions accompanying emerging adulthood may trigger anxiety and depressive symptoms among vulnerable people. The list of developmental challenges at the transition to adulthood is long: for instance, leaving home, beginning and ending romantic relationships, succeeding in academics, and choosing a career (e.g., Helson, Kwan, John, & Jones, 2002). For that reason, Arnett (2000) referred to emerging adulthood as “demographically dense.” Real, perceived, or anticipated failure to successfully negotiate any of these age-graded social roles theoretically could provoke anxiety and depression, especially among those prone to negative affect. As explained below, our participants were over-sampled for adolescents endorsing high levels of trait negative affectivity. Thus, consistent with a diathesis-stress viewpoint (e.g., Beck, 1979), it could be argued that the transition to adulthood—with the demands of achieving normative life tasks more salient than ever—should activate negative affect, anxious arousal, and anhedonic symptoms in the present sample (cf. Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013).

We expected to find significant variability in both the initial level and the amount of change across all dimensions (Question 2). Further, based on prior theorizing—albeit mostly in the context of cross-sectional symptom associations—about the structure of anxiety and depression (e.g., Clark & Watson, 1991), we hypothesized that growth in negative affect would, to some extent, correlate with both anhedonia and anxious arousal. In contrast, in line

with prior theory and empirical evidence emphasizing their discriminant validity (e.g., Mineka et al., 1998), growth in anhedonia and anxious arousal was predicted to be mostly independent. As described earlier, these specific dimensions of the tripartite model are hypothesized to represent symptom dimensions that are distinctive to different emotional disorders.

We examined a pair of background variables theoretically linked to the nature of emotional disorder (co)development during adolescence and the transition to adulthood (Question 3). We examined gender differences in symptom trajectories in light of the diverging emotional disorder prevalence rates during this developmental stage (e.g., Ge, Conger, & Elder 2001). We generated competing predictions with respect to gender. On the one hand, women are expected to show steeper symptom increases (or flatter declines) because of the higher risk for anxiety and depression during this period. On the other hand, if gender differences in adolescence are largely mediated by neuroticism (e.g., Eaton et al., 2011), one would expect no differences in symptom trajectories across gender because both men and women in the present study were over-sampled on the basis of elevated neuroticism (see Methods). We also predicted that diagnostic history would influence symptom development. We concentrated specifically on those who had a history of anxiety *and* depressive diagnoses. We hypothesized that comorbidity would predict not only the shape of symptom trajectories (i.e., steady or increasing symptom patterns), but also the extent to which symptoms changed together over time. That is, we expected that comorbid cases, by virtue of possessing vulnerabilities for both anxiety and depression, would evidence a stronger coupling of change across symptom domains over follow up.

Method

Participants

Participants were part of the Youth Emotion Project (YEP), a longitudinal study of the development of emotional disorders. High school juniors were recruited in three cohorts from schools in suburban Los Angeles and suburban Chicago. Those students who provided assent and parental consent ($N = 1,976$) were administered the Neuroticism scale of the revised Eysenck Personality Questionnaire (EPQ-R-N; Eysenck & Eysenck, 1975). A subsample of 1,269 students was invited to participate in the YEP on the basis of EPQ-R-N responses, overselecting for those in the highest Neuroticism tertile. There were 627 students who completed the baseline assessment. Females were the larger portion of this sample (68.7%), owing to greater likelihood of completing the screening questionnaire (56% female), scoring higher on the EPQ-R-N, and accepting the invitation to participate. At baseline, participants were an average of 16.9 years old ($SD = 0.37$). Participants identified as Caucasian (50.2%), Hispanic/Latino (13.8%), African American (13.4%), Asian/Pacific Islander (4.7%), other race/ethnicity (5.3%), and multiracial (12.6%). Additional details regarding screening and selection procedures are available elsewhere (Zinbarg et al., 2010).

Measures

Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995)—The 90-item MASQ was constructed to assess the general and specific symptom domains of the

tripartite model of anxiety and depression. The MASQ has three general dimensions that feature symptoms common to multiple anxiety and depressive disorders. The 15-item General Distress: Mixed Symptoms scale includes symptoms that appear in the criterion sets for both anxiety and depressive conditions (e.g., irritability, concentration difficulty, insomnia) and represents the tripartite model spectrum of negative affect that characterizes all emotional disorders. The General Distress: Anxious Symptoms scale assesses anxious mood and other symptoms that empirically do not differentiate among various anxiety disorders. Analogously, the General Distress: Depressive Symptoms scale includes items assessing depressed mood and other nonspecific symptoms of mood disorder. The General Distress: Mixed Symptoms (hereafter Mixed Symptoms) scale was preferred to the other two because it captures symptoms of emotional distress that are identified in modern nosologies as being symptoms of both anxiety and depressive disorders (Watson et al., 1995).

Two scales contain items selected to index specific symptom dimensions of either anxiety or depression. Anxious Arousal features 17 items related to somatic tension and hyperarousal (e.g., dizziness, shortness of breath, shaking hands) that reflect the anxious arousal dimension of the tripartite model. In contrast, the 22-item Anhedonic Depression scale assesses the anhedonia tripartite domain with items related to loss of interest and positive affective states (e.g., enjoyment, happiness). On all scales, respondents indicated on a five-point scale to what extent they had experienced each symptom (1 = not at all, 5 = extremely) “during the past week, including today.”

Prior research on the latent architecture of the MASQ Anhedonic Depression scale in this and other samples (Kendall et al., 2016; Keogh & Reidy, 2000; Watson, Clark, Weber, & Assenheimer, 1995) indicates that it is multidimensional. That is, during MASQ development, some items (i.e., those tapping the low end of positive affect such as “Felt really bored,” “Felt withdrawn from other people,” “Felt unattractive”) cohered with MASQ items intended to assess negative affect (Watson et al., 1995). A recent investigation into the latent structure of the MASQ Anhedonic Depression scale using bifactor analysis found support for an overarching general factor uniting all 22 items, but also for separate group factors for the 8 items tapping low positive affect (and likely contaminated with negative affect variance) and the remaining 14 tapping the high end of positive affect (Kendall et al., 2016). Kendall et al. recommended separating these item sets in future research. Thus, to achieve maximum discrimination between anhedonia and the other two tripartite dimensions, we omit the 8 items thought to be saturated with negative affect and include in our Anhedonic Depression scale only those 14 items assessing high positive affect (e.g., “Felt really cheerful,” “Looked forward to things with enjoyment,” “Felt really good about myself”). Following MASQ scoring conventions, these 14 items were reverse-scored such that higher values represent greater anhedonia.

The MASQ has demonstrated excellent psychometric properties in cross-sectional research. Scale reliability values are consistently .80 and above across student, community, and patient samples (Watson et al., 1995). The pattern of scale correlations is in line with tripartite model predictions, such that the three General Distress dimensions are highly intercorrelated, the General Distress dimensions are moderately correlated with Anxious

Arousal and Anhedonic Depression, and Anxious Arousal and Anhedonic Depression are weakly correlated (Watson et al., 1995). Also supporting the discriminant validity of the specific dimensions, Anxious Arousal is far more related than Anhedonic Depression to conventional measures of anxiety, whereas Anhedonic Depression is more closely connected than Anxious Arousal to measures of depression. In the present study, scale reliability estimates at baseline were all above .88 (range: .88 to .93).

Structured Clinical Interview for DSM-IV, Non-Patient Edition (SCID-I/NP)—The SCID-I/NP (First et al., 2002) assesses DSM-IV (American Psychiatric Association, 1994) diagnoses. The SCID-I/NP was administered at baseline to assess lifetime history of emotional disorders (i.e., current and past diagnoses) up to and including Time 1. Diagnoses occurring after Time 1 were not examined in this study to preserve the temporal precedence of diagnostic history relative to symptom trajectories. Each completed SCID-I/NP was presented at a consensus meeting led by doctoral-level supervisors. To qualify for a diagnosis, participants had to meet DSM-IV criteria and be assigned a clinical severity rating (CSR; Di Nardo & Barlow, 1988) of four or greater for that diagnosis. CSRs are used to quantify symptom severity and the amount of impairment and distress present, and they are rated on a scale of 0–8, with a 0 indicating no notable symptoms, distress, or interference, 1–3 indicating subclinical severity, and ratings of four and higher indicating clinical significance. Inter-rater reliability for both CSRs (Pearson $r_s > .70$) and DSM-IV diagnoses (kappas $> .65$) were in the acceptable to good range (see Zinbarg et al., 2010, for more detail). The diagnostic frequencies (and percentages) at baseline were 112 (18%) major depressive disorder, 7 (1%) dysthymia, 6 (1%) panic disorder, 53 (9%) social phobia, 13 (2%) obsessive-compulsive disorder, 4 (1%) posttraumatic stress disorder, 13 (2%) generalized anxiety disorder, and 33 (5%) specific phobia. There were 48 comorbid cases (8% of sample) at baseline with a history of both an anxiety and depressive disorder.

Procedures

Participants were recontacted 10 months after baseline, and each subsequent wave, to complete the study assessment battery. Each successive assessment was administered between 10 and 18 months after the prior assessment, and the mean interval between follow ups was 0.94 year ($SD = 0.12$). If a participant was not reachable or available to complete a given assessment (time t) in the 18 months following the previous wave (time $t-1$), he or she was still contacted to participate in the following wave (time $t+1$). The available sample sizes for the MASQ at baseline and the four follow up points were 604, 443, 326, 340, and 360. We compared 69 baseline respondents who did not participate at any subsequent wave with those who did, and we found that attriters did not differ from other participants on any baseline demographic or psychopathology index, that they reported higher levels of baseline Anxious Arousal, $t(601) = 2.00, p < .05$. In addition, baseline variables generally were not related to missingness at any single wave. The exceptions were that elevated Anxious Arousal predicted missingness at Time 4, $t(601) = 2.54, p < .05$, and Time 5, $t(601) = 2.93, p < .05$, and elevated Mixed Symptoms predicted missingness at Time 5, $t(601) = 2.93, p < .05$.

The MASQ was completed through a secure website at each assessment wave, and participants were mailed a check upon completion. The Institutional Review Boards at Northwestern University and UCLA approved all study procedures.

Statistical Analysis

We specified latent curve models (LCMs) to understand how each symptom domain changed over time and the extent to which they changed in concert. LCM is well suited for those research objectives because it assumes, unlike other statistical frameworks for studying multivariate change (e.g., autoregressive cross-lagged models), an underlying growth process that shapes the trajectory of observed repeated measures (Bollen & Curran, 2006; Duncan & Duncan, 2004). Figure 1 presents a path diagram of a bivariate LCM that assumes linear change in both constructs—here we use Mixed Symptoms and Anxious Arousal as examples—over time. However, the multivariate LCM can easily be extended to include additional constructs and alternate functional forms of change (e.g., polynomial models); our example focuses on the linear bivariate case for simplicity. In the diagram, the LCM slope factor means are represented by the regression of each factor on a constant value of unity (shown in the triangle). The weights of these regressions (labeled α_2 and α_4) reflect the direction (i.e., sign of the coefficient) and rate (i.e., magnitude of the coefficient) of symptom change. Meanwhile, the random portion of each factor is represented by a separate latent variable influencing the growth factors. Thus, the slope factor variances (ψ_{22} and ψ_{44}) reflect the degree of between-person variability in symptom trajectories, whereas the slope factor covariance (ψ_{42}) indexes the extent to which the two symptom trajectories are correlated over time.

The first step in our analysis was to find the optimal functional form of change over time for each symptom domain independently. In univariate models, we evaluated a linear growth model in which slope factor loadings were specified as 0 (Time 1 [T1]), 1 (T2), 2 (T3), 3 (T4), and 4 (T5), and all intercept loadings were fixed at 1. We compared that linear model, which assumes a constant rate of symptom change over follow up, with a quadratic model that allows for a nonlinear trajectory (i.e., acceleration or deceleration in growth). In quadratic models, we reparamaterized the factor loadings such that time was centered on T3 (i.e., linear trend factor loadings: -2, 1, 0, 1, 2) to avoid estimation problems related to linear dependence among the linear and quadratic factors (Stoolmiller, 1995). A likelihood ratio test (LRT) of nested models was used to judge whether the addition of quadratic effects significantly improved model fit.

After we characterized the form of symptom growth in univariate analyses, we examined correlations among symptom trajectories. In a trivariate LCM, we examined the cross-construct covariances between the intercepts and rates of change for each pair of symptom dimensions. In this trivariate model, cross-construct within-time error covariances (e.g., the covariance between the Time 3 Mixed Symptoms error term and Time 3 Anxious Arousal error term) were freely estimated. A LRT indicated that allowing these error covariances significantly improved fit relative to a model in which they were fixed to zero, $\chi^2(3) = 389.46, p < .001$. Further, these error covariances were constrained to equality over time for

model parsimony; another LRT showed that these constraints did not significantly degrade model fit for any of the three pairs of symptom dimensions, $\chi^2(4) < 7.50, ps > .10$.

After determining the best fitting trivariate LCM, we compared it to the latent curve model with structured residuals (LCM-SR; Curran, Howard, Bainter, Lane, & McGinley, 2014), an alternate—and closely related—model of multivariate change. The LCM-SR is a hybrid of LCM and autoregressive cross-lagged models that specifies autoregressive and cross-lagged paths among adjacent *residuals* of the manifest variables, as opposed to the manifest variables themselves. Thus, the LCM-SR is an extension of the LCM that includes autoregressive and cross-lagged regressions of residual terms of repeated measures at time t on residual terms of repeated measures at time $t-1$ (regressions onto more distal repeated measure residuals [e.g., $t-2$] are possible but not tested here). These additional parameters represent the degree to which departures from one's expected trajectory at time t in one construct predict deflections from one's expected trajectory at time $t+1$ in the other construct.

After empirically comparing the LCM and LCM-SR (and retaining the LCM), we examined individual differences in symptom change as a function of gender and comorbidity. This testing was performed in a multiple-groups structural equation modeling context, which allows model parameters to vary across population subgroups. We chose the multiple-group approach instead of specifying the covariates as exogenous predictors of growth factors because the latter procedure only permits an examination of covariate effects on factor means (see Bollen & Curran, 2006), whereas our primary interest was in how gender and comorbidity influenced growth factor *covariances* (i.e., correlation of growth trajectories across symptom dimensions). Testing followed a two-step process. First, in univariate LCMs, we compared models in which factor means were constrained to equality across the subgroups or varied freely. The LRT of the fit of these nested models indicated whether the shape of change in each symptom dimension was constant across subgroups. Second, in a series of bivariate LCMs, we compared models in which the factor variances and covariances were equated versus freely estimated across subgroups. A significant LRT in this step would indicate that the sample variance in the rate of change or the correlation in change across symptom domains differed across subgroups. If an LRT was significant in either step, we inspected the model modification indices (MIs) to determine which, if any, of the model constraints led to poor fit. It should be noted that such *post hoc* inspection of MIs can lead to model overfitting, or picking up on chance variation in a given sample, and we therefore proceed with caution when interpreting MIs (e.g., MacCallum, 2001). Due to the increasing number of significance tests, we followed an analog of the protected t -test procedure whereby we only proceeded to examine the MIs for means and (co)variances if the omnibus LRT was significant (Cohen, Cohen, West, & Aiken, 2003).

We assessed model fit with the comparative fit index (CFI), Tucker-Lewis index (TLI), root mean square error of approximation (RMSEA), and standardized root mean square residual (SRMR). We compared sample estimates of these criteria to guidelines for acceptable fit offered by Hu and Bentler (1999). We also judged model fit on the basis of localized areas of strain and the size and interpretability of parameter estimates. We used the robust maximum likelihood estimator in Mplus (version 7.3, Muthén & Muthén, 1998–2014) for all analyses

to account for non-normality in MASQ scale distributions, and missing data due to attrition were accommodated using full information maximum likelihood estimation procedures (see Allison, 2003).

Results

Descriptive Statistics

Table 1 presents the means and standard deviations of each symptom domain across time. Average symptom levels were comparable to undergraduate norms reported in studies of MASQ psychometrics (Watson et al., 1995). Also, Mixed Symptoms and Anxious Arousal appeared to decline over time, whereas there was a small overall increase in Anhedonic Depression. The corresponding effect sizes representing change between the symptom mean at T1 to the symptom mean at T5, expressed in Cohen's d (using standard deviations at T1), were -0.47 , -0.41 , and 0.06 for Mixed Symptoms, Anxious Arousal, and Anhedonic Depression, respectively.

Measurement Invariance

Measurement invariance was examined prior to fitting LCMs to verify that any observed symptom change over time was true change in the underlying construct rather than variability across time in the measurement properties of MASQ items (see Horn & McArdle, 1992). To evaluate measurement invariance, we estimated a series of confirmatory factor analyses (CFAs) for each symptom dimension involving the two most distant waves (i.e., T1 and T5). That is, we compared CFAs with cross-time equality constraints on factor loadings and indicator intercepts with models in which those parameters were allowed to vary freely. In this analysis, each scale item was treated as a separate indicator of a latent variable representing the relevant symptom dimension (e.g., each of the 17 Anxious Arousal items was treated as an indicator of an Anxious Arousal factor). For each scale we tested for weak (i.e., loadings but not intercepts) and strong (i.e., loadings and intercepts) measurement invariance.

The results supported partial invariance for all three scales. According to LRTs and comparison of other model fit indices (e.g., CFI, RMSEA), 1 factor loading was allowed to vary across time for Anhedonic Depression, whereas all factor loadings were time-invariant for Mixed Symptoms and Anxious Arousal. Regarding the intercepts, 4 (out of 15) on the Mixed Symptoms scale, 6 (out of 14) on the Anhedonic Depression scale, and 2 (out of 17) on the Anxious Arousal scale were non-invariant over time. Thus, we concluded that partial invariance obtained and we could proceed with the LCM. (Full results are available upon request from the first author.)

Unconditional Univariate Latent Curve Models

This series of LCMs aimed to quantify the rate and variability of symptom growth over time. The observed patterns of change are depicted in Figure 2. We first compared linear and quadratic models to evaluate the constancy of change in each symptom domain. Linear models provided adequate fit for Mixed Symptoms, $\chi^2(14) = 25.52$, $p < .05$; CFI = .97; TLI = 0.98; RMSEA = 0.04; SRMR = .06; Anxious Arousal, $\chi^2(14) = 40.40$, $p < .001$; CFI = .

89; TLI = 0.92; RMSEA = 0.08; SRMR = .13; and Anhedonic Depression, $\chi^2(14) = 29.44$, $p < .01$; CFI = .96; TLI = 0.97; RMSEA = 0.04; SRMR = .06. However, chi-square difference tests indicated that the addition of a quadratic factor—including the factor mean, variance, and covariances among the intercept and linear trend factors—significantly improved model fit for each dimension: Mixed Symptoms, $\chi^2(4) = 56.11$, $p < .001$; Anxious Arousal, $\chi^2(4) = 64.72$, $p < .001$; Anhedonia, $\chi^2(4) = 47.72$, $p < .001$ (see Table 2 for quadratic model fit statistics).

Table 2 shows the parameter estimates—including factor means, variances, and correlations—from the quadratic LCMs. As expected, the means of all intercept factors differed from zero. More significantly, the large intercept variance estimates indicated wide cross-sectional variation on each symptom dimension. The linear trend estimates¹ for Mixed Symptoms and Anxious Arousal were negative—indicative of a downward trajectory—and statistically significant. There was also significant variability around the linear trend estimates, suggesting that not all participants exhibited an equally precipitous drop in symptoms over follow up.

Moreover, a statistically significant quadratic factor mean was observed for Mixed Symptoms. The positive sign of the coefficient indicated that the decline in Mixed Symptoms decelerated over the course of follow up, such that, on average, the linear trend became approximately 0.56 units² more positive at successive intervals. The quadratic trend for Anxious Arousal was positive but not statistically significant. However, there was significant variability around that estimate, indicating a wide degree of variation in the expected curvature in Anxious Arousal symptom change over time.

The Anhedonic Depression trajectory diverged from that of Mixed Symptoms and Anxious Arousal. The average linear trend over follow up was positive, although it only approached conventional levels of statistical significance. The mean quadratic trend was positive but not statistically significant. Further, significant variability in the quadratic factor revealed that the amount of change in change over time varied across participants.

Unconditional Trivariate Latent Curve Model

The next analysis allowed us to examine covariances in properties of symptom change. The correlations among intercept factors—representing symptom levels at Time 3, since time was centered at the middle assessment wave—provided insight into cross-sectional associations between symptom domains. Mixed Symptoms had a strong association with Anxious Arousal ($r = .73$). In contrast, the intercept correlations of Anhedonic Depression with Mixed Symptoms ($r = .36$) and Anxious Arousal ($r = .20$) were small-to-moderate.³

¹In the quadratic model, the (simple) linear trend estimates are interpreted as the slope of the tangent to the curve at the model intercept (i.e., Time 3). The linear trend estimates from the quadratic LCMs were virtually identical to the slope factor mean estimates from linear LCMs (results available upon request).

²The rate of change in the linear trend per unit time is double the quadratic factor mean (e.g., for Mixed Symptoms: 2×0.28).

³In an exploratory analysis, we respecified the intercept location at the other assessment waves to figure out whether cross-sectional associations between the symptom domains changed over the course of the study. We did not detect any systematic trends in the size or pattern of intercept intercorrelations across waves. The range of intercept correlations for Mixed Symptoms and Anxious Arousal was .71-.78; that of Mixed Symptoms and Anhedonic Depression was .24-.36; and that of Anxious Arousal and Anhedonic Depression was .09-.20.

Table 3 presents zero-order correlations between growth factors. We focus on the cross-construct correlations between linear and quadratic factors to understand the degree of correspondence in change over time among tripartite model dimensions. The linear change in Mixed Symptoms was significantly and positively related to that in Anxious Arousal ($r = .59$). In contrast, the correlation in linear change between Mixed Symptoms and Anhedonic Depression ($r = .27$) did not reach statistical significance, and that between Anhedonic Depression and Anxious Arousal ($r = -.02$) was non-significant and almost nil. Constraining the covariances among linear factors to equality produced a significant decrement in model fit, $\chi^2(2) > 11.30, p < .01$, indicating that these correlations differ significantly in magnitude. An analogous pattern of correlations emerged for the quadratic factors. The Mixed Symptoms quadratic trend (i.e., the upward bend in trajectory over follow up) was strongly related to that of Anxious Arousal ($r = .75$) but not Anhedonic Depression ($r = .35$). There was a small association between quadratic trends in Anhedonic Depression and Anxious Arousal ($r = .15$).⁴ As with the linear factors, fixing all three quadratic factor covariances to equality significantly worsened model fit, $\chi^2(2) > 6.47, p < .05$, again suggesting that tripartite quadratic trajectories are not all related equally.

Alternate Model for Multivariate Change

As described above, we aimed to compare the fit of the LCM to the LCM-SR to determine whether any prospective relations among the symptom constructs were evident after accounting for the latent growth processes thought to shape tripartite symptom trajectories. We began by specifying a trivariate LCM-SR to correspond to our final trivariate LCM. As in the LCM, we specified a quadratic functional form for all three constructs. The trivariate LCM-SR produced an improper solution; the estimates for the variances of the linear and quadratic trends for all symptom constructs were negative. (We also observed a negative variance estimate for the linear trend in sensitivity analyses that omitted the quadratic factor.) In *ad hoc* analyses we constrained the variances of the linear and quadratic factors to zero. These constraints led to a reasonable model fit, $\chi^2(93) = 81.95, p = .80$; CFI = 1.00; TLI = 1.00; RMSEA = 0.00; SRMR = .05; however, none of the cross-lagged paths were significantly different from zero (all standardized cross-lags were smaller than $|.04|$). In other words, they did not explain the longitudinal correlation among symptom domains above and beyond the latent growth trajectories. We interpreted this set of results as indicating that cross-lagged paths across symptom constructs do not add meaningfully to our understanding of the nature of symptom co-development in this sample. Thus, we rely on the multivariate LCM for interpretations of the direction, size, and covariance of symptom trajectories, and we carry it forward to examine predictors of individual differences in those trajectories.

⁴We mentioned above that our main analyses omitted the 8 Anhedonic Depression items previously found to be saturated with negative affect variance and meaningfully distinguished in bifactor analyses from the 14 Anhedonic Depression items tapping the high end of positive affect (Kendall et al., 2015; Watson et al., 1995). Here we present the pattern of growth for a dimension comprised of those 8 Anhedonic Depression items that appear to assess the low end of positive affect. Linear growth for this dimension was negative ($b = -0.82, p < .001$) with significant interindividual variability around that estimate. The quadratic factor mean was not significantly different from zero. In parallel process growth models, the Anhedonic Depression intercept was very closely related to the intercepts for Mixed Symptoms ($r = .93$) and Anxious Arousal ($r = .70$). Moreover, the correlation in Anhedonic Depression's linear trend with Mixed Symptoms ($r = .77$) was remarkably high; it had a more moderate longitudinal association with Anxious Arousal ($r = .33$). Paralleling the pattern of linear trend associations, the correlations among quadratic factors were .90 for Mixed Symptoms and .64 for Anxious Arousal. Overall, these results supported the notion that a subset of MASQ Anhedonic Depression items may reflect negative affect and therefore might not be optimal indicators of the anhedonia dimension (for details, see Kendall et al., 2015).

Multiple-Group Multivariate Growth Models

When growth factor means were constrained to equality across gender, model fit worsened only in the Anhedonic Depression model, $\chi^2(2) = 10.79, p < .01$. Inspection of the MIs revealed that only the model intercept differed across gender, $\chi^2(1) = 8.17, p < .01$. Specifically, men ($M = 44.66$) had higher Anhedonic Depression levels at the study mid-point than women ($M = 41.80$). No factor variances or cross-construct covariances varied as a function of gender.

Factor means were found to differ across comorbidity status for all three symptom constructs, $\chi^2(3) > 10.30, ps < .01$. For Mixed Symptoms, MIs indicated that the intercept factor mean was higher for comorbid cases (comorbid group: $M = 33.16$; non-comorbid group: $M = 28.44$), $\chi^2(1) = 18.80, p < .001$. Further, the quadratic factor mean was larger for comorbid cases (comorbid group: $M = 1.15, SE = 0.45, p < .05$; non-comorbid group: $M = 0.20, SE = 0.11, p = .06$), $\chi^2(1) = 15.49, p < .001$. For Anhedonic Depression, the intercept factor mean was also higher for comorbid cases (comorbid group: $M = 46.56, SE = 1.53, p < .001$; non-comorbid group: $M = 42.34, SE = 0.50, p < .001$), $\chi^2(1) = 9.31, p < .01$. For Anxious Arousal, only the quadratic factor mean differed across groups (comorbid group: $M = 0.86, SE = 0.25, p < .001$; non-comorbid group: $M = 0.06, SE = 0.10, p = .56$), $\chi^2(1) = 15.73, p < .001$.⁵ Factor variances and cross-construct covariances did not vary significantly by comorbidity status.

Discussion

We mapped the trajectory of core dimensions of emotional disorders across five annual assessment waves beginning in adolescence. We observed reductions over follow up in negative affect and anxious arousal symptom levels, but no significant change in anhedonia. We also found that the rate of change in these symptoms appeared to change over time; that is, emotional disorder development during this period was not entirely linear. Just as important, we demonstrated that the rate of symptom change—and changes in the rate of change—are not necessarily correlated across tripartite domains. Specifically, anhedonia progressed fairly independently from negative affect and anxious arousal.

The patterns of change in tripartite domains suggest that emotional disorder symptoms do not uniformly rise or fall during emerging adulthood. Consistent with the maturity principle (Caspi et al., 2005), which posits that accomplishment of normative life tasks (e.g., choosing a career, beginning serious romantic relationships) at the transition to adulthood should be accompanied by reductions in trait negative affectivity, we found declining trajectories for negative affect and anxious arousal (cf. Robins et al., 2001). In contrast, adolescents did not “mature out” of anhedonic symptoms at the transition to adulthood. If anything, these symptoms appeared to worsen somewhat over this period. This observation accords with findings from community studies that document substantial continuity of depressive

⁵Supplementing our analyses of comorbidity effects on growth factor means, we regressed these factor means on a trichotomous independent variable reflecting EPQ-R Neuroticism tertiles (see Methods), a proxy for baseline emotional disorder risk (similar to a history of diagnostic comorbidity). In line with previous studies in community and patient samples (e.g., Brown & Rosellini, 2011; Galambos et al., 2006), we found that higher Neuroticism levels were associated with significantly greater baseline levels and steeper linear declines across all three symptom dimensions, consistent with regression to the mean (full results available upon request).

symptoms and disorders from adolescence to emerging adulthood, including peak rates of depressive disorder recurrence (e.g., Rohde et al., 2013). Moreover, the persistence of anhedonia is in line with the theory that the “demographically dense” transition to adulthood—featuring novel social challenges such as leaving home and navigating long-term romantic relationships—set the stage for depression among vulnerable people (see Arnett, 2000). However, such changes in anhedonia might also be consistent with a wide range of other developmental processes (e.g., normative development of reward processing systems, less time for engagement in pleasurable activities). Additional research is needed to determine to what extent developmental challenges at the transition to adulthood shape the naturalistic course of emotional disorder features.

Our findings extend prior developmental studies by distinguishing between general and differentiating features of emotional disorders. That is, previous work typically has involved assessments that conflate the core dimensions of anxiety and depression (cf. Clark & Watson, 1991). Therefore, the naturalistic course of the specific and distinctive variance in observed scores on depressive symptom measures have been associated with fluctuations in anxiety to an unknown extent (and vice versa). By modeling the tripartite dimensions over time, we charted separate courses for negative affect, which characterizes all emotional disorders, and more specific components of anxiety and depression. This distinction proved revealing, as negative affect declined monotonically over late adolescence, whereas anhedonia—a specific dimension to unipolar depression and social phobia—exhibited comparatively little growth (in the opposite direction). This result aligns with previous treatment research that shows anhedonia is the emotional disorder dimension least likely to change with intervention (Craske et al., in press).

Just as important as the symptom trajectories were the correlations among those trajectories. We showed, for the first time, that negative affect, anhedonia, and anxious arousal symptoms do not travel together as a “lump” across development. The correspondence between linear change in anhedonia and negative affect was nearly half (standardized coefficient $\sim .30$) that of anxious arousal and positive affect ($\sim .60$). The standardized relation between anhedonia and anxious arousal linear factors was virtually nil. Although this pattern of correlations does not rule out meaningful associations between aspects of change in anhedonia with other tripartite dimensions, it did appear that anhedonia development was more weakly related to negative affect and anxious arousal than those two constructs were with one another. This finding is consistent with our knowledge of the cross-sectional architecture of emotional disorders (e.g., Mineka et al., 1998), and they support the notion that anxious arousal and anhedonia represent independent pathways to emotional disorders.

The nature of symptom growth—including relations among symptom trajectories—did not differ significantly for men and women. This result might be attributable to our sampling procedure, which overselected men at risk for emotional disorders. In other words, our data suggest that, if risk status is held constant, gender differences in the time course of symptom development in late adolescence are small. Alternatively, this pattern may be specific to the developmental stage under study (i.e., the transition from adolescence to adulthood). In early adolescence, gender differences in the rate of depression change, and perhaps the correlation between the rate of depression and anxiety symptom change, should be more apparent

according to established theory and longitudinal data (e.g., Ge et al., 1994; Lubke et al., 2015). More generally, we might expect symptom trajectories to respond to emotion-relevant biological (e.g., hormonal) and social (e.g., peer relations) processes as they come online, which can occur at different ages, and in qualitatively different fashion, for men and women (see, e.g., Hankin & Abramson, 2001).

Unlike gender, comorbidity appeared to have a systematic influence on the developmental map of tripartite symptoms. We found a pattern of elevated intercept levels among comorbid cases, as might be expected given the positive association between comorbidity and overall severity of emotional disorders (e.g., Brown, Campbell, Lehman, Grisham, & Mancill, 2001). We also found that when symptoms tended to decline over follow up—as was the case for negative affect and anxious arousal—the typical path of improvement was slowed over time in the comorbid group. That is, the normative trajectory of symptom reductions was not as steep—as reflected in larger quadratic factor means—among comorbid cases. However, counter to predictions, there was no evidence of differences in cross-construct covariances of symptom trajectories by comorbidity status. This result implies that the developmental trajectories of anxiety- and depression-specific symptoms are no more closely correlated for comorbid cases than for others. Stated differently, the unique components of anxiety and depression grow independently over time even among those who possess vulnerabilities for both anxiety and depression.

Several study limitations point to directions for future research. First, as observed earlier, the present sample was overselected for elevated trait negative affectivity, and results may not be characteristic of the general adolescent population. Although the average symptom levels in our sample were no higher than those reported in normative undergraduate samples (Watson et al., 1995), it is possible that regression to the mean may have biased our trajectory estimates, at least for high risk subgroups. We note, however, that a recent simulation study suggests that this type of oversampling does not seem to bias appreciably effect size estimates in behavioral research (Hauner, Zinbarg, & Revelle, 2014). Second, our analysis spans a critical period of emotional disorder development, but our results cannot be assumed to apply to other developmental epochs. Theory suggests that trait negative affectivity should continue to decline as young adults age (e.g., Caspi et al., 2005), but very few longitudinal data on the progression of relevant emotional disorder symptoms are available. Analogously, fluctuations in anhedonia may be most pronounced in early adolescence, when the incidence of depressive disorders climbs markedly. In fact, disrupted reward processing is evident even in disorder-free children who are at risk for depression (Gotlib et al., 2010). Thus, future research is needed to discover the time course of anhedonic (and other) symptoms in early developmental epochs. Third, the MASQ has undergone psychometric development since the start of the Youth Emotion Project. Its successor, the Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007), includes symptom dimensions that provide coverage of additional specific features thought to differentiate anxiety and depression (e.g., lassitude, traumatic intrusions). Future empirical work with the IDAS and other multifaceted transdiagnostic models such as the trilevel model (e.g., Naragon-Gainey, Prenoveau, Brown & Zinbarg, in press; Prenoveau et al., 2010) will shed more light on change in emotional disorder risk across development. Fourth, in some research contexts it can be theoretically and empirically challenging to differentiate symptom dimensions from related temperament

and personality processes (e.g., Krueger & Tackett, 2003). Our focus here was on change in emotional disorder symptoms, but it remains unclear to what extent the personality dimensions underlying anxiety and depression follow the same pattern of development. Future work is needed to directly compare, and examine causal influences between, symptom versus personality development over time (e.g., Clark, 2005; Morey & Hopwood, 2013). Fifth, 11% of the baseline sample did not participate in any follow up wave, and attritors endorsed higher levels of baseline anxious arousal symptoms. Although we used full information maximum likelihood, which is preferred to other methods for handling missing data (e.g., listwise deletion), differential attrition may still limit to some extent the generalizability of the results. Finally, (multiple-indicator) latent variable repeated measures can be incorporated into LCM to remove measurement error from growth factor indicators (e.g., Preacher, Wichman, MacCallum, & Briggs, 2008). Here we used single indicator repeated measures (i.e., MASQ scale means) to limit model complexity; however, it must be acknowledged that the factor indicator variance included measurement error.

In sum, we charted the trajectories of core dimensions of emotional disorders from adolescence into emerging adulthood. We found that the developmental trends for negative affect, anhedonia, and anxious arousal can look very different—and occasionally proceed in opposite directions—during this period. We also discovered that the rates of change in these symptom domains are not always strongly (or even minimally) correlated. Together, these results suggest that divergent pathways to emotional disorders are possible during this key developmental stage.

Acknowledgments

This research was supported by a two-site grant from the National Institute of Mental Health (NIMH) to MGC (R01-MH065651) and to SM and REZ (R01-MH065652). The content is the responsibility of the authors and does not necessarily represent the official views of the NIMH.

References

- Allison PD. Missing data techniques for structural equation modeling. *Journal of Abnormal Psychology*. 2003; 112:545–557. [PubMed: 14674868]
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th. Arlington, VA: American Psychiatric Association; 1994.
- Arnett JJ. Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*. 2000; 55:469–480. [PubMed: 10842426]
- Beck, AT., editor. *Cognitive therapy of depression*. Guilford press; 1979.
- Bollen, KA., Curran, PJ. *Latent curve models: A structural equation perspective*. John Wiley & Sons; 2006.
- Brown TA. Temporal course and structural relationships among dimensions of temperament and DSM-IV anxiety and mood disorder constructs. *Journal of Abnormal Psychology*. 2007; 116:313–328. [PubMed: 17516764]
- Brown TA, Barlow DH. A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: implications for assessment and treatment. *Psychological Assessment*. 2009; 21:256–271. [PubMed: 19719339]
- Brown TA, Chorpita BF, Barlow DH. Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*. 1998; 107:179–192. [PubMed: 9604548]

- Brown TA, Rosellini AJ. The direct and interactive effects of neuroticism and life stress on the severity and longitudinal course of depressive symptoms. *Journal of Abnormal Psychology*. 2011; 120:844–856. [PubMed: 21381799]
- Caspi A, Roberts BW, Shiner RL. Personality development: Stability and change. *Annual Review of Psychology*. 2005; 56:453–484.
- Clark LA. Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology*. 2005; 114:505–521. [PubMed: 16351374]
- Clark DA, Beck AT, Brown GK. Sociotropy, autonomy, and life event perceptions in dysphoric and nondysphoric individuals. *Cognitive Therapy and Research*. 1992; 16:635–652.
- Clark LA, Watson D. Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*. 1991; 100:316–336. [PubMed: 1918611]
- Craske MG, Meuret A, Ritz T, Treanor M, Dour H. Treatment for anhedonia: A neuroscience driven approach. *Depression and Anxiety*. in press.
- Eaton NR, Keyes KM, Krueger RF, Balsis S, Hasin DS. An invariant dimensional liability model of gender differences in mental disorder prevalence: Evidence from a national sample. *Journal of Abnormal Psychology*. 2012; 121:282–288. [PubMed: 21842958]
- Eysenck, HJ., Eysenck, SBG. *Manual of the Eysenck personality Questionnaire (junior and adult)*. Hodder and Stoughton; 1975.
- First, MB., Spitzer, RL., Gibbon, M., Williams, JBW. *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition. (SCID-I/NP)*. New York: Biometrics Research, New York State Psychiatric Institute; 2002.
- Forbes EE, Dahl RE. Neural systems of positive affect: Relevance to understanding child and adolescent depression? *Development & Psychopathology*. 2005; 17:827–850. [PubMed: 16262994]
- Forbes EE, Hariri AR, Martin SL, Silk JS, Dahl RE. Altered striatal activation predicting real-world positive affect in adolescent major depressive disorder. *American Journal of Psychiatry*. 2009; 166:64–73. [PubMed: 19047324]
- Galambos NL, Barker ET, Krahn HJ. Depression, self-esteem, and anger in emerging adulthood: seven-year trajectories. *Developmental Psychology*. 2006; 42:350–365. [PubMed: 16569173]
- Galambos N, Leadbeater B, Barker E. Gender differences in and risk factors for depression in adolescence: A 4-year longitudinal study. *International Journal of Behavioral Development*. 2004; 28:16–25.
- Ge X, Lorenz FO, Conger RD, Elder GH, Simons RL. Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental psychology*. 1994; 30:467–483.
- Gotlib IH, Hamilton JP, Cooney RE, Singh MK, Henry ML, Joormann J. Neural processing of reward and loss in girls at risk for major depression. *Archives of General Psychiatry*. 2010; 67:380–387. [PubMed: 20368513]
- Hale WW III, Raaijmakers QA, Muris P, Van Hoof A, Meeus WH. One factor or two parallel processes? Comorbidity and development of adolescent anxiety and depressive disorder symptoms. *Journal of Child Psychology and Psychiatry*. 2009; 50:1218–1226. [PubMed: 19570045]
- Hankin BL, Abramson LY. Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin*. 2001; 127:773–796. [PubMed: 11726071]
- Hauner KK, Zinbarg RE, Revelle W. A latent variable model approach to estimating systematic bias in the oversampling method. *Behavior Research Methods*. 2014; 46:786–797. [PubMed: 24142836]
- Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*. 1999; 6:1–55.
- Kendall AD, Zinbarg RE, Mineka S, Bobova L, Craske MG. Prospective associations of low positive emotionality with first onsets of depressive and anxiety disorders: Results from a 10-wave latent trait-state modeling study. *Journal of Abnormal Psychology*. 2015; 124:933–943. [PubMed: 26372005]

- Kendall AD, Zinbarg RE, Bobova L, Craske MG. Measuring positive emotion with the Mood and Anxiety Symptom Questionnaire: Psychometric properties of the Anhedonic Depression scale. *Assessment*. 2016; 23:86–95. [PubMed: 25657305]
- Keogh E, Reidy J. Exploring the factor structure of the mood and anxiety symptom questionnaire (MASQ). *Journal of Personality Assessment*. 2000; 74:106–125. [PubMed: 10779936]
- Kessler RC, Berglund P, Demler O, Jin R, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*. 2005; 62:593–602. [PubMed: 15939837]
- Krueger RF. The structure of common mental disorders. *Archives of General Psychiatry*. 1999; 56(10): 921–926. [PubMed: 10530634]
- Kim HK, Capaldi DM, Stoolmiller M. Depressive symptoms across adolescence and young adulthood in men: Predictions from parental and contextual risk factors. *Development and Psychopathology*. 2003; 15:469–495. [PubMed: 12931838]
- Klein DN, Kotov R, Bufferd SJ. Personality and depression: Explanatory models and review of the evidence. *Annual Review of Clinical Psychology*. 2011; 7:269–295.
- Kotov R, Gamez W, Schmidt F, Watson D. Linking “big” personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin*. 2010; 136:768–821. [PubMed: 20804236]
- Kotov R, Watson D, Robles JP, Schmidt NB. Personality traits and anxiety symptoms: The multilevel trait predictor model. *Behaviour Research and Therapy*. 2007; 45:1485–1503. [PubMed: 17240351]
- Krueger RF, Tackett JL. Personality and psychopathology: Working toward the bigger picture. *Journal of Personality Disorders*. 2003; 17:109–128. [PubMed: 12755325]
- Leadbeater B, Thompson K, Gruppuso V. Co-occurring trajectories of symptoms of anxiety, depression, and oppositional defiance from adolescence to young adulthood. *Journal of Clinical Child & Adolescent Psychology*. 2012; 41:719–730. [PubMed: 22742519]
- Lewinsohn PM, Clarke GN, Seeley JR, Rohde P. Major depression in community adolescents: age at onset, episode duration, and time to recurrence. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1994; 33:809–818. [PubMed: 7598758]
- MacCallum RC. Working with imperfect models. *Multivariate Behavioral Research*. 2001; 38:113–139.
- Markon KE. Modeling psychopathology structure: A symptom-level analysis of Axis I and II disorders. *Psychological Medicine*. 2010; 40:273–288. [PubMed: 19515267]
- McLaughlin KA, King K. Developmental trajectories of anxiety and depression in early adolescence. *Journal of Abnormal Child Psychology*. 2015; 43:311–323. [PubMed: 24996791]
- Measelle JR, Stice E, Hogansen JM. Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *Journal of Abnormal Psychology*. 2006; 115:524–538. [PubMed: 16866592]
- Mineka S, Watson D, Clark LA. Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*. 1998; 49:377–412.
- Morey LC, Hopwood CJ. Stability and change in personality disorders. *Annual Review of Clinical Psychology*. 2013; 9:499–528.
- Muthén, LK., Muthén, BO. Mplus version 7. Los Angeles, CA: Muthén & Muthén; 1998–2014.
- Naragon-Gainey K, Watson D, Markon KE. Differential relations of depression and social anxiety symptoms to the facets of extraversion/positive emotionality. *Journal of Abnormal Psychology*. 2009; 118:299–310. [PubMed: 19413405]
- Olino TM, Stepp SD, Keenan K, Loeber R, Hipwell A. Trajectories of depression and anxiety symptoms in adolescent girls: A comparison of parallel trajectory approaches. *Journal of Personality Assessment*. 2014; 96:316–326. [PubMed: 24369925]
- Prenoveau JM, Zinbarg RE, Craske MG, Mineka S, Epstein AM. Testing a hierarchical model of anxiety and depression in adolescents: A tri-level model. *Journal of Anxiety Disorders*. 2010; 24:334–344. [PubMed: 20171054]

- Roberts BW, Walton KE, Viechtbauer W. Patterns of mean-level change in personality traits across the life course: A meta-analysis of longitudinal studies. *Psychological Bulletin*. 2006; 132:1–25. [PubMed: 16435954]
- Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*. 1970; 126:107–111.
- Robins RW, Fraley RC, Roberts BW, Trzesniewski KH. A longitudinal study of personality change in young adulthood. *Journal of Personality*. 2001; 69:617–640. [PubMed: 11497032]
- Rohde P, Lewinsohn PM, Klein DN, Seeley JR, Gau JM. Key characteristics of major depressive disorder occurring in childhood, adolescence, emerging adulthood, and adulthood. *Clinical Psychological Science*. 2013; 1:41–53.
- Stoolmiller, M. Using latent growth curve models to study developmental processes. In: Gottman, JM., editor. *The analysis of change*. Mahwah, NJ: Erlbaum; 1995. p. 103-138.
- Van Oort FVA, Greaves-Lord K, Verhulst FC, Ormel J, Huizink AC. The developmental course of anxiety symptoms during adolescence: the TRAILS study. *Journal of Child Psychology and Psychiatry*. 2009; 50:1209–1217. [PubMed: 19490305]
- Watson D, Gamez W, Simms LJ. Basic dimensions of temperament and their relation to anxiety and depression: A symptom-based perspective. *Journal of Research in Personality*. 2005; 39:46–66.
- Watson D, Naragon-Gainey K. Personality, emotions, and the emotional disorders. *Clinical Psychological Science*. 2014; 2:422–442. [PubMed: 25815243]
- Watson D, O'Hara MW, Simms LJ, Kotov R, Chmielewski M, McDade-Montez EA, Stuart S. Development and validation of the Inventory of Depression and Anxiety Symptoms (IDAS). *Psychological Assessment*. 2007; 19:253–268. [PubMed: 17845118]
- Watson D, Clark LA, Weber K, Assenheimer JS. Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology*. 1995; 104:15–25. [PubMed: 7897037]
- Watson D, Weber K, Assenheimer JS, Clark LA, Strauss ME, McCormick RA. Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology*. 1995; 104:3–14. [PubMed: 7897050]
- Zinbarg RE, Mineka S, Craske MG, Griffith JW, Sutton J, Rose RD, Waters AM. The Northwestern-UCLA youth emotion project: Associations of cognitive vulnerabilities, neuroticism and gender with past diagnoses of emotional disorders in adolescents. *Behaviour Research and Therapy*. 2010; 48:347–358. [PubMed: 20070951]

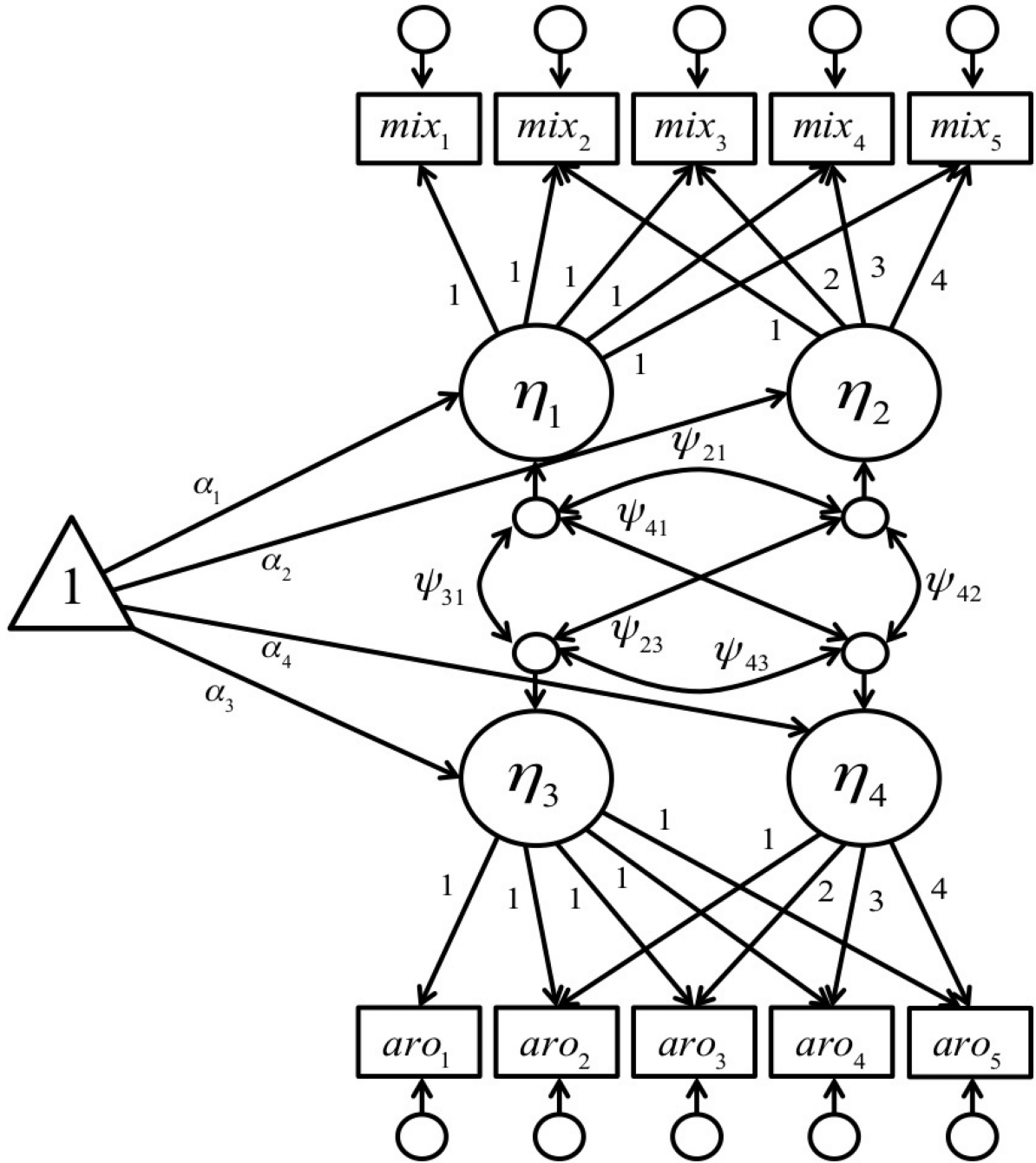


Figure 1. Path diagram for the bivariate linear latent curve model for Mood and Anxiety Symptom Questionnaire Mixed Symptoms and Anxious Arousal measured at five occasions. η_1 and η_3 represent intercept factors, whereas η_2 and η_4 represent linear trend factors. The ψ s represent factor variances and covariances, and the α s denote factor means. Following path diagram conventions, rectangles represent observed variables and circles represent latent variables. Growth factor means are represented by regressing the factors onto a constant (unity). Within-time residual covariances are omitted for clarity of presentation.

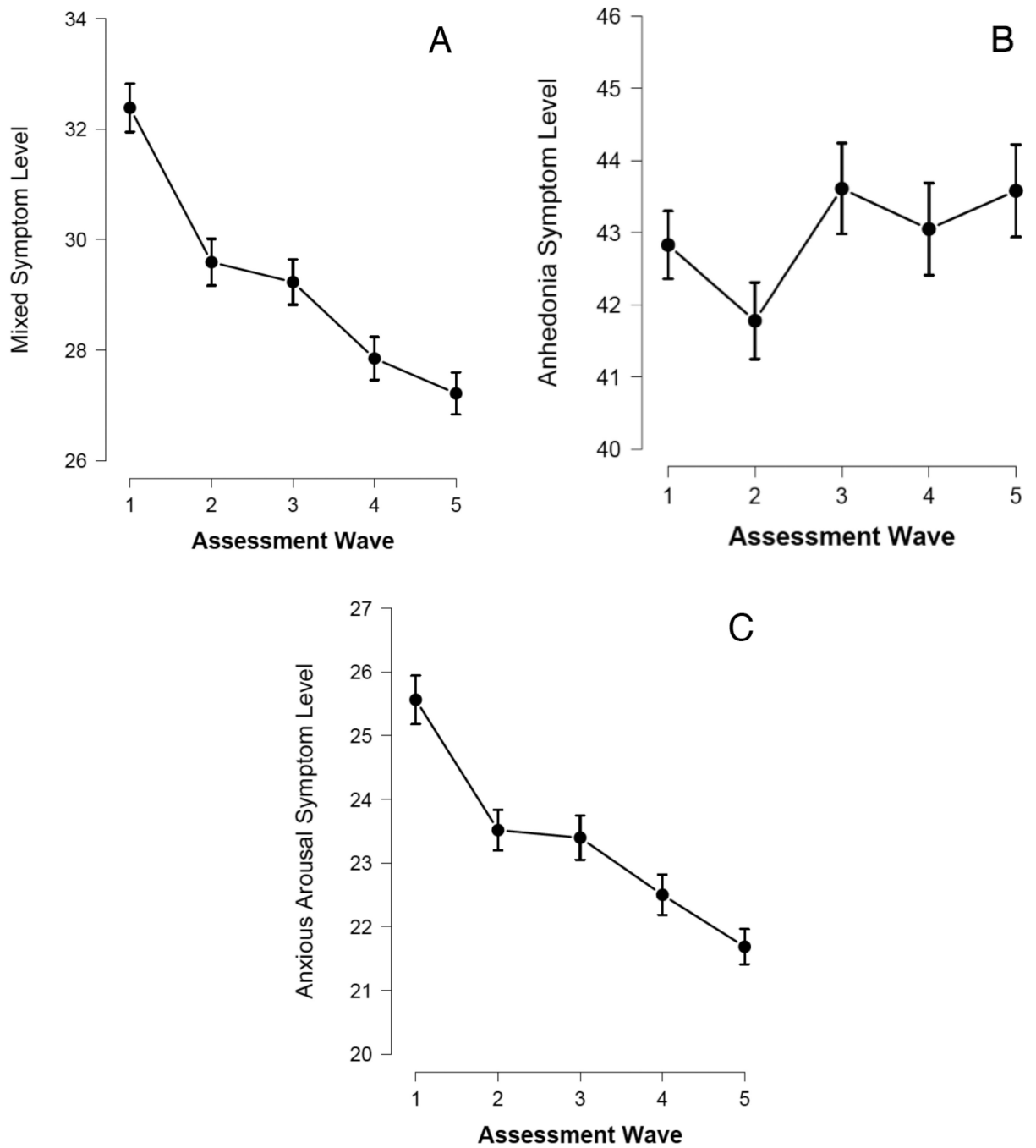


Figure 2. Repeated measures of Mood and Anxiety Symptom Questionnaire scales. (A) General Distress: Mixed Symptoms; (B) Anhedonic Depression; (C) Anxious Arousal.

Table 1

Means and Standard Deviations of Tripartite Model Symptom Dimensions.

Assessment Wave	N	Mixed Symptoms		Anxious Arousal		Anhedonic Depression	
		Mean	SD	Mean	SD	Mean	SD
Time 1	604	32.39	10.97	25.56	9.55	42.83	11.57
Time 2	443	29.59	10.59	23.52	7.92	41.78	11.23
Time 3	326	29.23	10.22	23.40	8.67	43.61	11.36
Time 4	340	27.85	9.75	22.50	7.90	43.05	11.92
Time 5	360	27.22	9.50	21.69	6.95	43.58	12.19

Note. N = sample size; SD = standard deviation. The Anhedonic Depression dimensions included only those 14 Mood and Anxiety Symptom Questionnaire items assessing high positive affect (see main text).

Table 2

Unstandardized Parameter Estimates from Unconditional Univariate Latent Growth Models for Mixed Symptoms, Anxious Arousal, and Anhedonic Depression

Parameter	Mixed Symptoms ^a	Anxious Arousal ^b	Anhedonic Depression ^c
Intercept variance	62.76 (7.54) ***	47.09 (7.32) ***	73.62 (7.65) ***
Linear trend mean	-1.13 (0.13) ***	-0.82 (0.10) ***	0.28 (0.16) †
Linear trend variance	2.45 (0.64) ***	2.05 (0.61) ***	5.27 (0.93) ***
Linear trend-intercept <i>r</i>	-.15 (.12)	-.09 (.12)	.01 (.10)
Quadratic trend mean	0.28 (0.11) **	0.13 (0.10)	0.13 (0.12)
Quadratic trend variance	1.30 (0.46) **	1.66 (0.41) ***	1.73 (0.57) **
Quadratic trend-intercept <i>r</i>	-.44 (.10) ***	-.52 (.10) ***	-.45 (.08) ***
Quadratic trend-linear trend <i>r</i>	-.20 (.20)	-.50 (.20) *	.13 (.17)

Note. *N* = 606. Standard errors appear in parentheses.

† *p* < .10,

* *p* < .05,

** *p* < .01,

*** *p* < .001.

^a Model fit: $\chi^2(10) = 9.04$, *p* = .53; CFI = 1.00; TLI = 1.00; RMSEA = 0.00; SRMR = .03.

^b Model fit: $\chi^2(10) = 9.77$, *p* = .46; CFI = 1.00; TLI = 1.00; RMSEA = 0.00; SRMR = .05.

^c Model fit: $\chi^2(10) = 16.05$, *p* = .10; CFI = .98; TLI = 0.98; RMSEA = 0.03; SRMR = .06.

Table 3
Growth Factor Correlations from a Trivariate Unconditional Latent Curve Model

	Intercepts			Trends		
	MIX	ARO	ANH	MIX _Q	ARO _L	ANH _L
Intercepts						
ARO	.73***					
ANH	.36***	.20**				
Trends						
MIX _L	-.15	-.02	-.02			
MIX _Q	-.44***	-.34**	-.23*	-.19		
ARO _L	-.27*	-.09	-.03	.59***	-.23	
ARO _Q	-.30**	-.52***	-.10	-.25	.75***	-.50*
ANH _L	.09	.07	.01	.27	-.14	-.02
ANH _Q	-.23	-.13	-.45***	.03	-.09	.15
						.13

Note. $N = 606$. Model fit: $\chi^2(75) = 73.50, p = .53$; CFI = 1.00; RMSEA = 0.00; SRMR = .04. MIX = Mixed Symptoms; ARO = Anxious Arousal; ANH = Anhedonic Depression; *L* subscript denotes linear factor; *Q* subscript denotes quadratic factor.