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The Impact of Environmental Experiences on Symptoms of Anxiety and Depression Across the Life Span

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Abstract

Symptoms of anxiety and depression are relatively stable over time. Can this stability be explained by genetic influences, or is it caused by the long-lasting effects of accumulating environmental experiences? To address this question, we analyzed longitudinally assessed symptoms of anxiety and depression in eight samples of monozygotic twins of widely varying ages. These samples were drawn from American and European population-based registries. Using hierarchical linear modeling, we examined individual differences and individual changes in the level of symptoms over time. This method enabled us to decompose the variance into the predictable variance shared by both members of each pair of twins, the differences between individuals within pairs, and the residual variance. We then modeled how these components of individual variation changed over time. Within pairs, the twins' predicted levels of symptoms increasingly diverged from childhood until late adulthood, at which point the divergence ceased. By middle adulthood, environmental experiences contributed substantially to stable and predictable interindividual differences in levels of anxiety and depression.

Keywords

anxiety, depression, adult development, behavior genetics, emotional development

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Individuals' levels of symptoms of depression and anxiety are relatively stable over time (Foley, Neale, & Kendler, 2001; Lovibond, 1998). What is responsible for this stability? The *genetic-set-point* hypothesis offers one plausible explanation: Genetic factors determine the stable set points to which an individual will eventually return after the anxiety and depression produced by environmental experiences have subsided (Kandler et al., 2010).

Studies of twins have provided consistent empirical support for this hypothesis. In such studies, researchers have found that across the life span, genetic factors both substantially influence levels of anxiety and depression (Boomsma, van Beijsterveldt, & Hudziak, 2005; Kendler, Gardner, & Lichtenstein, 2008; McGue & Christensen, 1997; Silberg et al., 1990) and are largely responsible for their temporal stability (Gillespie et al., 2004; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998).

The genetic-set-point hypothesis can be contrasted with an *environmentally-influenced-set-point* hypothesis, which assumes that environmental experiences produce enduring effects on an individual's set points for anxiety and depression. According to this alternative hypothesis, strong negative experiences, whether caused by acute events or by chronic difficulties, could elevate symptoms of anxiety and depression for decades, whereas powerful positive experiences could lessen symptoms. At least two mechanisms might underlie these processes. Life experiences could produce enduring effects on symptoms through biological changes in levels of DNA methylation that result in the altered structure and function of neural systems.

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Alternatively, changes in the intensity of symptoms might occur via psychological and social routes. For example, early environmental adversity could lead individuals to enter into poor interpersonal relationships (Daley & Hammen, 2002) and other kinds of high-risk environments that further exacerbate symptoms (Kendler, Gardner, & Prescott, 2011).

Several studies have offered empirical support for an environmentally influenced set point for anxiety and depression symptoms. Severe environmental stressors, such as childhood sexual abuse, natural disasters, and combat exposure, produce enduring effects on symptoms of anxiety and depression (Fergusson & Mullen, 1999; Goenjian et al., 2005; Kendler et al., 2000; Koenen et al., 2003). In the German Socio-Economic Panel Study (Headey, 2010), a representative longitudinal study that followed members of German households for 20 years, 14% to 30% of subjects experienced a large and stable change to their set point for well-being, a construct strongly correlated with levels of anxiety and depression (Keyes, 2002, 2005). However, most studies suggest that commonly experienced environmental adversities, particularly stressful life events, have only a temporary impact on depression symptoms, increasing risk for episodes of major depression for only a few months (Brown & Harris, 1978; Kendler, Karkowski, & Prescott, 1998; Surtees et al., 1986).

A cohort of monozygotic twins constitutes an ideal sample for assessing the sources of stability in symptoms of anxiety and depression. Because monozygotic twins are born with identical DNA sequences, differences that arise between monozygotic twins during the life course are mainly the result of nongenetic influences. Some of these differences, however, may reflect epigenetic effects, structural variation (Bruder et al., 2008), or random developmental processes (Molenaar, Boomsma, & Dolan, 1993). In this report, we refer to all such differences as "environmental."

Following the approach suggested by Dickens, Turkheimer, and Beam (2011) for studying intelligence across development, we analyzed symptoms of anxiety and depression in eight samples of longitudinally assessed monozygotic twins derived from population-based registries. Our first goal was to determine whether the data supported a genetically determined or environmentally influenced set point for such symptoms. According to the genetic-set-point hypothesis, the differences in symptoms of anxiety and depression within pairs of monozygotic twins should be stable over the life course. According to the standard environmentally-influenced-set-point hypothesis, levels of anxiety and depression should gradually diverge within monozygotic twin pairs as they age, as a result of the gradual accumulation of different environmental experiences (see Fig. 1).

Our second goal was to determine whether the differences in symptoms of anxiety and depression within monozygotic twin pairs continue to diverge over the entire life course or stop accumulating at some developmental stage. According to a modified environmentally-influenced-set-point hypothesis (see Fig. 1), differences within twin pairs should stabilize in early adulthood because most severe psychological traumas

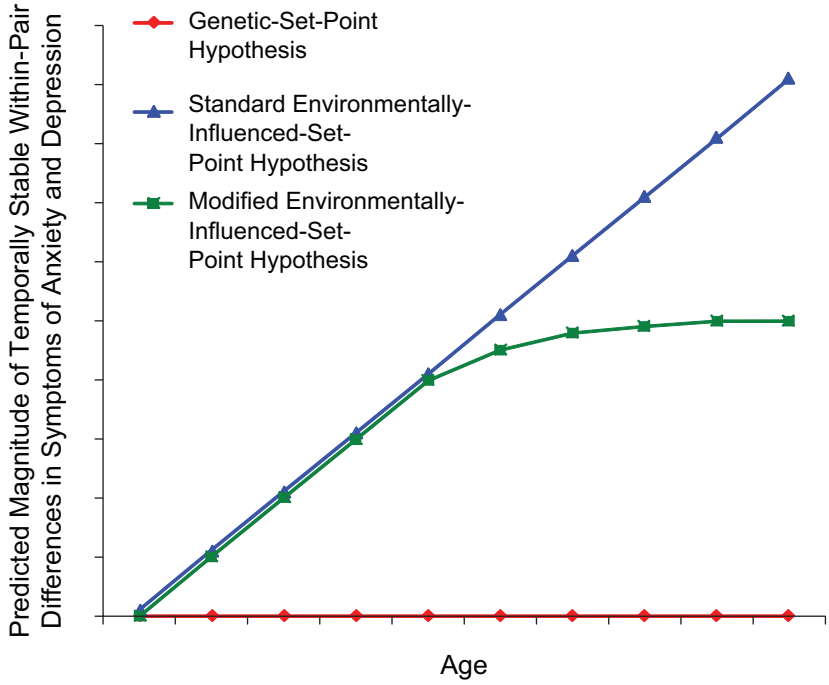

Fig. 1. The predicted relationship between age and the magnitude of predictable within-pair difference in symptoms of anxiety and depression in monozygotic twin pairs. Predictions are shown for the genetic-set-point hypothesis, the standard environmentally-influenced-set-point hypothesis, and the modified environmentally-influenced-set-point hypothesis.

(e.g., sexual or physical abuse) occur between early childhood and late adolescence. However, changes in emotional processing are especially prominent in older age groups (Mather & Carstensen, 2005). If emotional processing is central to the influence of environmental experiences on symptoms of anxiety and depression, differences in symptoms within monozygotic pairs might accumulate throughout early and middle adulthood and stabilize in late adulthood.

Method

Subjects

We examined seven samples of monozygotic twins with symptoms of depression or anxiety as measured by self-reports on at least three occasions and one sample of monozygotic twins who had reported on such symptoms on two occasions (Table 1). The combined sample contained 12,148 twins, including 8,470 twins in pairs and 3,678 unpaired twins (the data from unpaired twins provided useful information on the temporal stability of symptoms). The mean age of twins in these samples at each evaluation ranged from 10.6 to 66.8 years.

Statistical analyses

We analyzed the longitudinal data using a random-coefficients approach involving three steps. In the first step, we used a basic regression model with assessment wave, sex, birth cohort, and age entered as fixed effects. The fixed-effects regression equation provided an estimate for individual subjects' mean level of anxiety and depression, controlling for sex, birth cohort, and age at interview. Each observation for each individual had a residual representing his or her departure from this mean response. This residual, which is assumed to result from individual characteristics, measurement error, and other random error, was then used to estimate individual growth trajectories. A minimum of three measurements from different waves in a given study was needed to estimate a linear growth trajectory. These individual growth trajectories were characterized by their slopes and intercepts, which were the random coefficients.

Finally, we used the regression equation for the mean symptom levels (the fixed effects) and the individual trajectories to estimate the variance components of interest. Because these trajectories included both intercepts and slopes, the variance included mean between-subjects differences as well as within-subjects change over time. The final model yielded a *marginal residual* (the deviation of the individual measurement from the value predicted by the fixed-effects regression equation), which was made up of a *conditional residual* (the deviation of the individual measurement from that predicted by the fixed-effects regression equation plus the individual's linear growth trajectory) plus the *error residual*. Thus, the outcome measure was the sum of the mean response, the conditional residual, and the error residual, with a sampling variance equal to the sum of the variance of the conditional residual plus the variance of the error residual.

Because of clustering within monozygotic twin pairs, we were able to further decompose the variance predicted by individual growth trajectories into the variance in symptom levels shared by members of twin pairs and the variance in symptom levels not shared within pairs. We obtained the estimates of the variance components at a particular wave of a particular longitudinal study from the intercept measure by centering the age measure at the individual's age at that assessment (see Biesanz, Deeb-Sossa, Papadakis, Bollen, & Curran, 2004).

Longitudinal modeling of symptom scores was carried out using the SAS PROC MIXED procedure with a random-coefficients approach. The components of the model were entered as fixed effects, the individual linear growth trajectories were entered as random effects, and individuals were clustered hierarchically into monozygotic twin pairs.

The fixed-effects regression included the following demographic variables: sex, interview wave (to adjust for mean differences across waves), birth year (to adjust for cohort effects), and age centered at an individual's age in a particular study (in cases in which age was not confounded with birth year). Most of the data sets supplied subjects' exact age at time of interview. For example, an individual born in 1960 and interviewed at ages 36.36, 39.23, and 40.45 would have a birth year of 1960 at each interview, but the age vectors would be (0.00, 2.87, 4.09), (–2.87, 0.00, 1.22), and (–4.09, –1.22, 0.00) for the purpose of estimating parameters for Waves 1, 2, and 3, respectively. In some cases, however, the dates given for interview waves specified only the year.

All two-way interactions were considered, and those with p values less than .1 were retained. The fixed-effects equation gave the mean score for an individual of a particular sex, birth cohort, centered age, and interview wave. The scales used to measure anxiety and depression were all skewed rightward. Using the square root of the score produced reasonably homoscedastic residuals.

For each wave at which a subject was interviewed, the subject had a residual that represented his or her individual deviation from the mean score. Using these deviations, we constructed individual growth trajectories characterized by their slope and intercept (the value when age = 0, i.e., the age at the specific wave of a particular study for which we were estimating parameters). Individual deviations were then decomposed into two components, one predicted by individual growth trajectories and one due to residual variation (which included measurement error). Because the subjects within twin pairs were hierarchically clustered, we decomposed the variation due to individual growth trajectories into variation shared by both members of a twin pair and variation not shared by both members of the pair. The algorithm alternated between weighted least squares solutions for the fixed-effects regression and residual maximum likelihood estimation of the variance components (intercept, slope, and intercept-slope covariance) attributable to individual growth trajectories. Thus, for each wave of a particular study,

Table 1. Twin Samples Included in This Study and Instruments Used to Assess Their Symptoms of Depression and Anxiety

Study, instruments, and wave	Mean age (years)	*n*
Virginia Twin Study of Adolescent Behavioral Development (What I Think and Feel Anxiety Scale; Mood and Feeling Depression Scale); R_Λ = .78		
Wave 1	10.6 (1.7)	643
Wave 2	12.1 (1.8)	639
Wave 3	15.4 (1.7)	602
Young Netherlands Twin Register (Youth Self-Report Anxiety and Depression scales); R_Λ = .82		
Wave 1	12.0 (0.5)	464
Wave 2	14.6 (0.6)	490
Wave 3	16.8 (0.4)	555
Wave 4	18.5 (0.5)	270
Swedish Twin Study of Child and Adolescent Development[a] (Child Behavior Checklist Anxiety and Depression scales; Youth Self-Report Anxiety and Depression scales); R_Λ = .75		
Wave 1	13–14	800
Wave 2	16–17	830
Wave 3	19–20	636
Adult Netherlands Twin Register (Youth Self-Report Anxiety and Depression scales); R_Λ = .89		
Wave 1	19.7 (1.1)	399
Wave 2	21.6 (2.2)	796
Wave 3	23.5 (2.2)	533
Wave 4	27.3 (2.5)	727
Virginia Adult Twin Study of Psychiatric and Substance Use Disorders—females (Symptom Checklist-90 Anxiety, Phobic Anxiety, and Depression scales); R_Λ = .79		
Wave 1	29.4 (7.4)	1,086
Wave 2	34.5 (7.4)	1,078
Wave 3	37.2 (7.4)	965
Virginia Adult Twin Study of Psychiatric and Substance Use Disorders—males (Symptom Checklist-90 Anxiety, Phobic Anxiety, and Depression scales); R_Λ = .77		
Wave 1	34.5 (9.1)	1,422
Wave 2	36.0 (9.1)	1,422
Swedish Adoptive Twin Study of Aging (Spielberger State Anxiety Scale); R_Λ = .88		
Wave 1	57.0 (13.6)	454
Wave 2	60.0 (13.6)	483
Wave 3	62.1 (13.2)	457
Wave 4	63.4 (13.1)	409
American Association of Retired Persons twin sample (Symptom Checklist-90 Anxiety, Phobic Anxiety, and Depression scales); R_Λ = .91		
Wave 1	63.1 (7.5)	1,603
Wave 2	64.2 (7.5)	1,623
Wave 3	66.8 (7.5)	1,623

Note: Standard deviations are given in parentheses. R_Λ represents the reliability of an entire series of longitudinal observations (i.e., how reliably we captured the underlying latent measure by including scores from multiple observations of each individual in our random-coefficients model). Data from the Virginia Twin Study of Adolescent Behavioral Development were drawn from Simonoff et al. (1997). The What I Think and Feel Anxiety Scale was taken from Reynolds and Richmond (1978). The Mood and Feeling Depression Scale was taken from Costello and Angold (1988). Data from the Young Netherlands Twin Register were drawn from Bartels et al. (2007). The Youth Self-Report scales were taken from Achenbach (1991). Data from the Swedish Twin Study of Child and Adolescent Development were drawn from Lichtenstein, Tuvblad, Larsson, and Carlstrom (2007). The Child Behavior Checklist scales were taken from Achenbach and Edelbrock (1983). Data from the Adult Netherlands Twin Register were drawn from Boomsma et al. (2006). Data from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders for both males and females were drawn from Kendler and Prescott (2006). The Symptom Checklist-90 scales were taken from Derogatis, Lipman, and Covi (1973). Data from the Swedish Adoptive Twin Study of Aging were drawn from Pedersen et al. (1991). The Spielberger State Anxiety Scale was taken from Spielberger, Gorsuch, and Luchene (1970). Data from the American Association of Retired Persons twin sample were drawn from Prescott et al. (1994).

[a]The Child Behavior Checklist scales were used for Waves 1 and 2 of the Swedish Twin Study of Child and Adolescent Development, and the Youth Self-Report scales were used for Wave 3. Both of these instruments included a 12-item Anxiety and Depression scale, which we examined in our analysis. Exact ages were not reported for this study.

we decomposed individual deviation from the mean into three components: a portion due to individual growth trajectories shared by members of a twin pair, a portion due to individual growth trajectories not shared by the members of the pair, and the residual variation.

Our methodology, which followed that used by Laenen, Alonso, and Molenberghs (2007) and Laenen, Alonso, Molenberghs, and Vangeneugden (2009), allowed us to calculate two reliabilities for longitudinal data: $R(T)$, an estimate of cross-sectional reliability (the reliability at a particular wave), and R_{Λ}, an estimate of the reliability of a series of longitudinal observations. For our purposes, "reliability" refers to the proportion of deviation predicted by individual growth trajectories, or the sum of the proportion shared by twins within a twin pair and the proportion not shared by twins within a pair.

Analysis of variance component proportion

For each wave of each study, we decomposed the variance due to deviation from the mean response into the variance due to growth trajectories shared by both members of a monozygotic twin pair (between-pair variance), the variance due to growth trajectories not shared between twins (within-pair variance), and the residual variance (a mixture of measurement error and other variation not explained by the model). We focused on the proportion of the total variance attributable to longitudinal trajectories not shared by twins within a pair (within-pair variance divided by the sum of between-pair variance, within-pair variance, and residual variance). As a statistic, this proportion bears a resemblance to heritability, which also reflects a proportion of variance associated with a variance component. In order to estimate the standard error for this proportion, we used the parameter estimates for the variance components and the variance-covariance matrix of these estimates to generate a population of 10,000 pseudosamples, assuming the parameters are drawn from a multivariate normal distribution. The standard deviation of the proportion in these 10,000 pseudosamples provided an approximate standard error for the proportion. The standard errors calculated by this method were very close to those based on approximate methods (Osborne & Paterson, 1952).

When the ratio of within-pair variance to total variance was plotted against mean age at each sampling point, we observed a curvilinear relationship approaching a maximum divergence asymptotically. Given the observed plots and assuming that monozygotic twins begin life in identical states, we fit several nonlinear growth functions used to describe biological growth and compared these fits with that of a linear growth function using the PROC NLIN procedure in SAS software. All analyses were performed with parameterizations constrained to go through the origin. We tested the following functions: linear ($y = b \times \text{age}$), quadratic with a plateau ($y = b \times \text{age} + g \times \text{age}^2$), quadratic with a plateau at age $= -b \div (2 \times g)$, von Bertalanffy ($y = c \times (1 - exp(-k \times \text{age}))$), and Gompertz ($y = c \times (exp(-exp(-k \times (\text{age} - m))))$). Both the quadratic function with a plateau and the von Bertalanffy function (two parameters each) are curved convex functions with decreasing slopes that approach an asymptotic limit, whereas the Gompertz function (three parameters) is an asymmetric sigmoidal curve that also approaches an asymptotic limit. At each point, the estimate of the derived ratio was weighted by the inverse of the sampling variance of that estimate ($1 \div SE^2$) to account for the fact that different sampling points are estimated with different precision. Model fit was assessed using the root-mean-square error of approximation (RMSEA); RMSEA values under .05 indicate good model fit (Steiger, 1990).

Because statistics derived at different points in a given study applied to the same sample, the linear and quadratic models were fit with a generalized-estimating-equation analysis using PROC GENMOD. This method allowed for the nonindependence of parameter estimates from the same study.

Results

We first tested the genetic-set-point hypothesis, according to which monozygotic twins will not differ in their set-point levels of symptoms of anxiety and depression across the life span. Our data fit this model quite poorly, RMSEA = .22. Next, we tested the standard environmentally-influenced-set-point hypothesis, which predicts a linear increase in differences in symptoms between members of monozygotic twin pairs over time. The fit of our data to this model was markedly better, $F(1, 24) = 367.5$, $p < .0001$, RMSEA = .056. Finally, we modified the model testing the environmentally-influenced-set-point hypothesis by adding a quadratic growth function with a plateau, so as to capture the idea that the accumulation of differences between monozygotic twins reaches an asymptote at some developmental point. Our data fit this model significantly better than they fit the linear model corresponding to the standard environmentally-influenced-set-point hypothesis, $F(1, 23) = 38.2$, $p < .0001$, RMSEA = .035.

The fit of this best-fitting model is illustrated in Figure 2. As predicted by the modified environmentally-influenced-set-point hypothesis, the data showed a rapid and roughly linear growth in differences within monozygotic pairs in the temporally stable component of their self-reported symptoms through adolescence and into middle adulthood. However, the accumulation of these differences slowed down in late adulthood. The asymptote was estimated as equal to 69.4 years, but this estimate was imprecise (95% confidence interval = [60.1, 89.6]).

Analysis of our data using generalized estimating equations, and thereby treating estimates at different time points within a study as clustered, also contradicted the standard environmentally-influenced-set-point hypothesis. That is, the fit of the linear-plus-quadratic function was much better than that of the linear term alone (Wald $z = 4.49$, $p < .0001$). We tested two other growth functions, the von Bertalanffy function ($df = 2$) and the Gompertz function ($df = 3$), neither of which provided a fit superior to that observed with the linear-plus-quadratic model.

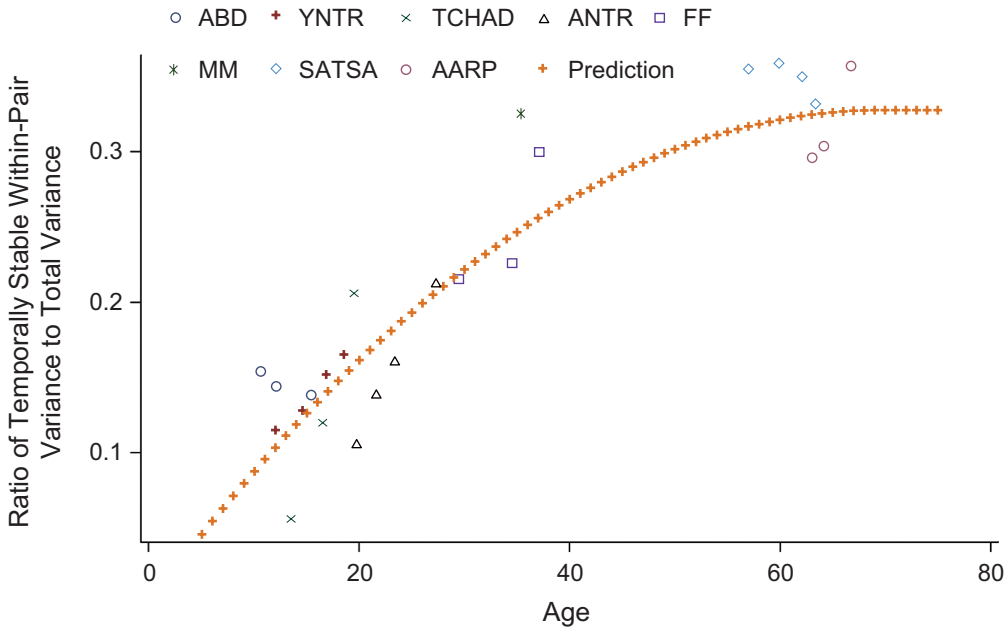

Fig. 2. Actual and predicted ratios of within-pair variance to total variance of the temporally stable component of symptoms of anxiety and depression as a function of the mean age of the tested cohort. The predicted values of the parameter estimates come from the best-fitting model, which had a linear component and a quadratic growth function with a plateau. ABD = Virginia Twin Study of Adolescent Behavioral Development (Simonoff et al., 1997); YNTR = Young Netherlands Twin Register (Bartels et al., 2007); TCHAD = Swedish Twin Study of Child and Adolescent Development (Lichtenstein, Tuvblad, Larsson, & Carlstrom, 2007); ANTR = Adult Netherlands Twin Register (Boomsma et al., 2006); FF = Virginia Adult Twin Study of Psychiatric and Substance Use Disorders—females (Kendler & Prescott, 2006); MM = Virginia Adult Twin Study of Psychiatric and Substance Use Disorders—males (Kendler & Prescott, 2006); SATSA = Swedish Adoptive Twin Study of Aging (Pedersen et al., 1991); AARP = American Association of Retired Persons twin sample (Prescott et al., 1994).

We examined the clinical significance of the differences within the monozygotic twin pairs by examining female-female twin pairs from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (Kendler & Prescott, 2006). For each month when symptoms of depression and anxiety were assessed, the authors reported whether the twins met the criteria outlined in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM–IV; American Psychiatric Association, 1994) for major depression (MD) or generalized anxiety disorder (GAD) with a 1-month, rather than 6-month, minimal duration. We generated a score for each subject's individual trajectory by obtaining the marginal residual (after the removal of the demographic fixed effects) and the conditional residual (based on fixed effects and individual trajectories). The difference between these two residuals was the individual trajectory deviation for that wave. In pairs of twins in which only one twin experienced an episode of MD or GAD at interview, the affected twin was much more likely than the unaffected twin to have the higher trajectory score (odds ratio = 16.0), $\chi^2(1) = 28.4$, $p < .0001$.

Finally, we examined sex differences in the linear and quadratic effects. Using an autoregressive working correlation matrix, we fit four models, comparing the fit using the quasi-likelihood information criterion (QIC), for which lower values indicate better fit (Pan, 2001). For the linear model with no sex interaction, the QIC was 61.69; for the linear model with a sex interaction, the QIC was 60.85; for the quadratic model with no sex interaction, the QIC was 54.35; and for the quadratic model with a sex interaction, the QIC was 58.72. Thus, the best-fitting model included linear and quadratic components but no sex interactions.

Discussion

On the basis of our findings, we reject the genetic-set-point hypothesis, according to which genetic factors alone are responsible for the temporal stability of symptoms of anxiety and depression. Our results are much more consistent with the environmentally-influenced-set-point hypothesis. However, we also reject the standard version of this hypothesis, according to which environmentally driven differences in stable levels of anxiety and depression continue to increase throughout the life cycle. To the contrary, we found that these differences continued to accumulate only until late adulthood. Our results are consistent with studies that have demonstrated an effect of aging on the emotional processing of adverse experiences. For example, Mather and Carstensen (2005) found that individuals show an increasingly selective retention of emotionally

positive memories relative to negative memories as they age. It is important to emphasize that in all the analyses presented in this report, the environment, including all influences that contributed to differences between monozygotic twins, was defined as a latent variable.

Our results are consistent with findings obtained in many community studies showing that severe environmental exposures can have enduring effects on levels of depression and anxiety (Fergusson & Mullen, 1999; Goenjian et al., 2005; Kendler et al., 2000; Koenen et al., 2003). The differences in levels of symptoms that developed within the monozygotic twin pairs in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (Kendler & Prescott, 2006) were strongly related to differences in risk for DSM–IV MD or GAD. We found no significant differences between males and females in these results.

To our knowledge, there are no other multiwave studies that have used genetically informative designs to measure the stability of environmental influences on symptoms of anxiety and depression and with which our study might be compared. However, a number of twin studies have longitudinally examined personality traits such as neuroticism and negative emotionality, which are closely related to symptoms of anxiety and depression (Jardine, Martin, & Henderson, 1984).

The studies that could most relevantly be compared with ours are those that have calculated within a genetically informative sample the long-term stability of personality traits that results from environmental experiences unique to the individuals. If such environmental experiences produce a cumulative effect on symptoms of anxiety and depression over time, then the environmental correlations for personality traits (i.e., cross-time correlations in personality due to environmental influences) related to anxiety and depression should increase with age. This possibility was first investigated by Viken, Rose, Kaprio, and Koskenvuo (1994) in a study of 15,000 Finnish twins, ages 18 to 53 at baseline, who were tested on two occasions 6 years apart. Across the two waves, environmental correlations for neuroticism, as measured by the short form of the Eysenck Personality Questionnaire (Eysenck, Eysenck, & Barrett, 1985), increased with age. Pedersen and Reynolds (1998) examined neuroticism in a sample of 2,209 twins, ages 26 to over 90, assessed at up to four waves in the Swedish Adoption/Twin Study of Aging. Although Pedersen and Reynolds did not formally calculate environmental correlations across time, their longitudinal model included a stable nonshared environmental component. They found that approximately half of the nonshared environmental variance was transmitted from occasion to occasion, and this transmission contributed substantially to the observed phenotypic stability of neuroticism over time.

More recently, Kandler et al. (2010) used self- and peer-report data from 696 monozygotic and 387 dizygotic twin pairs between the ages of 23 and 55 to investigate stability and change in personality over time. They found a strong trend for increasing environmental correlations for neuroticism with age and concluded that the "phenotypic continuity [of personality] increased as a function of cumulative environmental effects, which became manifest in stable trait variance and decreasing occasion-specific effects with age" (p. 995). Hopwood et al. (2011) studied 626 complete twin pairs, ages 17 to 29, over three waves using the Multidimensional Personality Questionnaire. The researchers found substantially higher environmental correlations for negative emotionality among twins who were 24 to 29 years old than among twins between 17 and 24 years old. Finally, Kupper, Boomsma, de Geus, Denollet, and Willemsen (2011) studied negative affectivity in 3,235 subjects from the Netherlands Twin Register and found that the environmental correlation increased dramatically from ages 17 to 25 to ages 25 to 30. Thus, prior twin studies provide considerable support for an environmentally influenced set point for personality traits, such as neuroticism, that are closely related to symptoms of anxiety and depression.

Two mechanisms might account for the ability of environmental experiences to stably alter levels of reported anxiety and depression. As recently reviewed by McCrory, De Brito, and Viding (2010), the most straightforward mechanism would be a direct effect on biological processes, from gene regulation and the modification of stress-response systems to anatomical changes in key brain structures, all of which could influence levels of depression and anxiety. Alternatively, the process could be psychological, driven by self-perpetuating interactions between symptomatic individuals and their social environment. For example, as shown in one integrated etiological model for depression (Kendler, Gardner, & Prescott, 2002), individuals who experience emotional trauma in childhood are prone to experience early-onset anxiety, which in turn leads to substance misuse, stressful life events, and low levels of social support, all of which predispose individuals to further symptoms. Much as has been described for individuals with normative personality traits (Hopwood et al., 2011), as individuals age, through self-selection they may enter into stability-promoting environments selected on the basis of the individuals' vulnerabilities. That is, individuals low in depression and anxiety symptoms tend to enter into mental-health-promoting environments, whereas anxious or depressed individuals tend to enter into stressful environments that further exacerbate their symptomatology.

In conclusion, our findings suggest that with respect to symptoms of anxiety and depression, individuals are what they have experienced, to an appreciable degree. Despite recent findings supporting the important influence of genetics on anxiety and depression, our findings contradict the idea that stability of symptoms of depression and anxiety results solely from genetic factors. Although many environmental stressors produce only short-term effects on mood, some aspects of environmental experience have long-term effects, through mechanisms that have not yet been identified, on levels of anxiety and depression symptoms.

Our results should be interpreted in the context of three methodological limitations. First, we combined studies of

twins of different ages and countries, and the studies we combined used a range of instruments to assess symptoms. Prior studies have shown that self-reported symptoms of anxiety and self-reported symptoms of depression are related forms of negative affect and are highly correlated in population samples (Clark & Watson, 1991). Second, our results provide no insight into the mechanisms by which environmental experiences alter the affective set point. Third, although we needed to study monozygotic twins to isolate the impact of environmental factors on the predictable component of symptom levels, it is probable that using monozygotic twins produced a downward bias on the magnitude of this effect. Monozygotic twins share much of their rearing environment, including many early environmental adversities. Furthermore, pairs of twins may share some stressful experiences in their adult lives. In addition, given the evidence that genetic factors influence the quality of many social relationships and individuals' entry into stressful situations (Kendler & Baker, 2007), the environmental exposures of monozygotic twins are likely to be more similar than those of individuals picked at random from a given population. Thus, our results probably underestimate the contribution of environmental exposures to stable individual differences in mood.

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