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Prospective Associations of Low Positive Emotionality with First Onsets of Depressive and Anxiety Disorders: Results from a 10-Wave Latent Trait-State Modeling Study

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Abstract

Unipolar depressive disorders (DDs) and anxiety disorders (ADs) co-occur at high rates and can be difficult to distinguish from one another. Cross-sectional evidence has demonstrated that whereas all these disorders are characterized by high negative emotion, low positive emotion shows specificity in its associations with DDs, social anxiety disorder (SAD), and possibly generalized anxiety disorder (GAD). However, it remains unknown whether low positive emotionality, a personality trait characterized by the tendency to experience low positive emotion over time, prospectively marks risk for the initial development of these disorders. We aimed to help address this gap. Each year for up to 10 waves, participants (n = 627, mean age = 17 years at baseline) completed self-report measures of mood and personality, and a structured clinical interview. A latent trait-state decomposition technique was used to model positive emotionality and related personality traits over the first three years of the study. Survival analyses were used to test the prospective associations of low positive emotionality with first onsets of disorders over the subsequent six-year follow-up among participants with no relevant disorder history. The results showed that low positive emotionality was a risk marker for DDs, SAD, and GAD, although evidence for its specificity to these disorders versus the remaining ADs was inconclusive.

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Additional analyses revealed that the risk effects were largely accounted for by the overlap of low positive emotionality with neuroticism. The implications for understanding the role of positive emotionality in DDs and ADs are discussed.

Keywords

Positive emotionality; neuroticism; stability; risk; emotional disorders

Although psychopathology research has traditionally focused on negative emotion in the emotional (i.e., unipolar depressive and anxiety) disorders, recent years have also seen strong interest in the role of positive emotion. This interest has been fueled in part by cross-sectional findings that whereas high negative emotion relates broadly to all depressive disorders (DDs) and anxiety disorders (ADs) (e.g., Clark & Watson, 1991), low positive emotion shows specificity to some disorders relative to others (see Watson & Naragon-Gainey, 2010). Such findings marked a breakthrough in describing the structure of depression and anxiety, which co-occur at high rates on both the symptom and diagnostic levels (e.g., Clark & Watson, 1991; Mineka, Watson, & Clark, 1998) and can be difficult to distinguish from one another. Virtually all the available data in this area have been cross-sectional, however, leaving unanswered the question of whether low positive emotionality prospectively marks risk for the initial development of emotional disorders. We sought to address this gap. Examining the prospective associations of low positive emotionality with first onsets of emotional disorders may help clarify if temporary experiences of low positive emotion are merely a symptom of some disorders, or if the enduring tendency to experience low positive emotionality is also a premorbid risk marker. Evidence that low positive emotionality marks risk for first disorder onsets could also inform prevention efforts.

Throughout our paper, the term *emotionality* describes *trait* phenomena, which are more time-invariant/stable. *Emotion* refers to *state* phenomena that are time-variant/unstable. In past research assessing emotionality, trait instructions (e.g., “Describe yourself as you are typically”) have often been employed, whereas state instructions (e.g., “Describe how you are right now”) have generally been used to assess emotion. As explained below, a contribution of the present study is that it instead used a latent trait-state decomposition technique to statistically separate emotionality from emotion.

Theoretical Background

Many of the dominant structural models of emotional disorders converge in suggesting low positive emotion distinguishes DDs from most forms of ADs. Facets of positive emotion—particularly high-approach/anticipatory states such as *wanting* and low-approach/consummatory states such as *liking* (e.g., Berridge & Robinson, 2003)—have received increased attention in the positive emotion and psychopathology literature. The structural models, however, have typically emphasized global, rather than facet-level, emotion. For example, the authors of the integrative hierarchical model (Mineka et al., 1998) showed that symptoms associated with high negative emotion, such as irritability, were common across DDs and ADs, helping account for their overlap. Symptoms associated with low positive emotion, such as anhedonia, the central feature of which is the loss of ability to experience

pleasure, were shown to be relatively specific to DDs and, to a lesser extent, social anxiety disorder (SAD)[1]. The integrative hierarchical model did not directly address etiology. However, it might be inferred that high negative emotionality should be a common risk marker of all emotional disorders, whereas low positive emotionality should be a relatively specific risk marker for DDs and perhaps SAD.

Developmental models of depression (e.g., Hankin & Abramson, 2001) provide a framework for considering how low positive emotionality might confer disorder risk. These models have generally focused on adolescence, when there is a sharp increase in DD onset, and have pointed to stressful events—which increase markedly during this period—as an important contributing factor. Empirical studies have linked positive emotions to resources (e.g., cognitive flexibility) that might buffer against the effects of stress (see Fredrickson, 1998, 2001). We speculate that people low on positive emotionality might have fewer resources to offset the effects of stress as they navigate adolescence and young adulthood, increasing vulnerability to DDs (see Gilbert, 2012).

Cross-Sectional Evidence

Cross-sectional studies of positive emotion and emotional disorders have generally supported the integrative hierarchical model (see Watson & Naragon-Gainey, 2010). One investigation showed that low positive emotion was also associated with symptoms of generalized anxiety disorder (GAD) (Prenoveau et al., 2010). Although preliminary, this work is relevant as it used the same data as did our study.

On the personality level, a meta-analysis (Kotov, Gamez, Schmidt, & Watson, 2010) showed that extraversion, a higher-order trait including positive emotionality and interpersonal facets (e.g., Watson & Clark, 1997; but see Smillie, Cooper, Wilt, & Revelle, 2012) was uniformly negatively associated with emotional disorders. However, its strongest links were with dysthymic disorder and SAD. Studies of positive emotionality in particular similarly found the strongest negative associations with DD diagnoses and symptoms, and moderate to strong negative links with SAD diagnoses and symptoms (e.g., Naragon-Gainey, Watson, & Markon, 2009; Watson, Stasik, Ellickson-Larew, & Stanton, in press). These findings persisted even after adjusting for other facets of extraversion and for neuroticism, a trait defined by high negative emotionality.

Prospective Evidence

Longitudinal studies have examined if low extraversion, measured with trait instructions, predicts first DD onsets, but have generally not provided support (e.g. Fanous, Neale, Aggen, & Kendler, 2007; Kendler, Neale, Kesler, Heath, & Eaves, 1993). One exception used a measure of extraversion *not* tapping positive emotionality (Kendler, Gatz, Gardner, & Pederson, 2006). The prospective effect of extraversion was accounted for by its shared variance with neuroticism.

[1]Although of less relevance to our study, the integrative hierarchical model also showed high levels of anxious arousal were relatively specific to ADs, helping distinguish them from DDs.

Other studies have prospectively related measures of low extraversion and/or positive emotionality to depressive symptoms or to any (not just first) DD onset (e.g., Dougherty, Klein, Durbin, Hayden, & Olino, 2010; Jorm et al., 2000; Lonigan, Phillips, & Hooe, 2003) using trait instructions. Conclusions specific to initial disorder development thus cannot be drawn. Although this work has generally not been supportive, one study showed low positive emotionality predicted higher depressive symptoms, even after accounting for neuroticism and past depressive symptoms (Dougherty et al., 2010). Some studies also included anxiety symptom outcomes (e.g., Jorm et al., 2000; Lonigan et al., 2003). No evidence was found for a significant relation between low extraversion and/or low positive emotionality and subsequent anxiety.

Methodological Considerations

In testing if low positive emotionality is a risk marker for the initial development of emotional disorders, two methodological issues should be addressed. These pertain to modeling positive emotionality, and accounting for its overlap with related personality constructs.

Stability and Change in "Trait" Constructs

Psychological constructs have historically been regarded as *either* traits or states (e.g., Spielberger, 1966). However, advances in longitudinal analysis have instead revealed that individuals' standing on psychological measures reflect *both* stable and unstable variance (Cole, Martin, & Steiger, 2005; Roberts & DelVecchio, 2000). Failure to decompose this stability and change may confound the two sources of variance. An additional benefit of decomposition is that it statistically adjusts for both occasion-specific fluctuations and measurement error (Cole & Maxwell, 2009).

These improvements in turn enhance the likelihood of identifying associations between a risk marker and an outcome. For instance, the trait—but not state—dimension of a construct could confer risk. If the stable and fluctuating variance in a measurement were not disentangled, the prospective association of the trait component with an outcome could be artificially attenuated. Indeed, longitudinal analyses indicated that removing the unstable variance from measurements of positive emotionality/extraversion and negative emotionality/neuroticism enhanced their prospective relations with subsequent depressive and social anxiety symptoms (Naragon-Gainey, Gallagher, & Brown, 2013).

Trait-state-occasion (TSO) latent variable modeling (Cole et al., 2005) is a state-of-theart method of identifying the stable and unstable variance in psychological constructs measured over time. The TSO model represents scores at a given time with a latent *state* variable (see Figure 1). Variance in each state is decomposed into three parts. First, variance that is completely stable across time and shared by all waves is represented by a single *trait*. Second, variance at each time that is unexplained by the trait and fluctuates is the *occasion* (i.e., residual of the trait). Third, occasions are autoregressive: an occasion can be partially predicted by the previous one through an autoregressive pathway (labeled β in Figure 1). The autoregressive pathways represent variance shared between two consecutive points that is not accounted for by the trait.

Related Personality Traits

Studies using trait instructions have established a robust correlation between positive emotionality and extraversion, typically r = .50-.70 (e.g., Watson & Clark, 1997). As noted, positive emotionality is often considered a lower-order facet of extraversion, along with facets such as sociability, assertiveness, and experience-seeking. Importantly, these facets may differentially relate to the symptoms and diagnoses of different emotional disorders (Naragon-Gainey et al., 2009; Watson et al., in press). On the disorder level, for instance, positive emotionality demonstrated the strongest and broadest negative links, followed by sociability (Watson et al., in press). Assertiveness and experience-seeking were weakly—and sometimes positively—associated with disorders. These findings underscore the need to separate positive emotionality from other facets of extraversion, perhaps most importantly sociability.

Positive emotionality and neuroticism, measured with trait instructions, typically show a weak negative or non-existent relation (e.g., Meyer & Shack, 1989). However, there is evidence that happiness, a facet of positive emotionality, is inversely related to neuroticism. A meta-analytic review (DeNeve & Cooper, 1998) related happiness to the Big Five dimensions of personality: extraversion, agreeableness, conscientiousness, neuroticism, and openness. Happiness was equally strongly associated with extraversion (average weighted r = .27) and neuroticism (average weighted r = −.25). An updated meta-analysis produced a similar pattern of results, with even stronger associations of extraversion (e.g., r = .49) and neuroticism (e.g., r = −.46) with happiness (Steel, Schmidt, & Shultz, 2008). An investigation applying the TSO model to extraversion/positive emotionality and neuroticism/negative emotionality showed a correlation of r = −.59 between trait components (Naragon-Gainey et al., 2013). These results, together with evidence that neuroticism marks risk for emotional disorders (e.g., Clark, Watson, & Mineka, 1994; Klein, Durbin, & Shankman, 2009), emphasize the importance of separating the influence of positive emotionality from that of neuroticism.

The Present Study

The present study sought to determine if low positive emotionality was a risk marker of first onsets of emotional disorders among adolescents and young adults with no relevant disorder history, using a 10-wave longitudinal design. We employed the TSO method to model positive emotionality over the first three years of the study. By incorporating assessments obtained at multiple waves over adolescence and young adulthood, rather than relying on a single assessment, the TSO model provided a more reliable estimate of positive emotionality over this developmental period (Cole & Maxwell, 2009). Survival analyses tested if low positive emotionality prospectively predicted first onsets of emotional disorders over the six-year follow-up. Survival analyses were selected over logistic regression because survival analyses would be sensitive to the timing until disorder onset. Furthermore, we tested the proportionality constraints imposed on our survival functions. This step, often overlooked in longitudinal research, allows for a more nuanced developmental perspective. For instance, it could show if the risk effects of low positive emotionality decay or strengthen over time. Finally, the shared variance of low positive emotionality with sociability and neuroticism,

also modeled over the first three years of the study using the TSO method, were accounted for in the prospective analyses.

We hypothesized that low positive emotionality would predict the subsequent development of DDs, SAD, and possibly GAD. We further expected the association of low positive emotionality with each of these disorders to be stronger than that with the remaining ADs. We tested this against the competing hypothesis that sociability and/or neuroticism would account for any prospective associations between low positive emotionality and disorders.

Method

Participants

High school juniors were recruited over three academic years for the Northwestern-UCLA Youth Emotion Project (YEP), a 10-wave longitudinal study of risk markers for emotional disorders (see Zinbarg et al., 2010). To capture the largest number of first onsets of different emotional disorders, youth were sampled starting around age 17 (e.g., Kessler et al., 2005). The two recruitment sites were racially and socioeconomically diverse public high schools in suburban Chicago and Los Angeles. To overcome possible statistical problems when predicting rare outcomes (e.g., Hauner, Zinbarg, & Revelle, 2014), students with high (i.e., top tertile) scores on a 22-item version of the neuroticism scale from the revised Eysenck Personality Questionnaire (EPQ-R-N; Eysenck & Eysenck, 1975) were oversampled.

At baseline, participants (n = 627, mean age = 16.9 years, SD = 0.43) were primarily female (69.2%) with EPQ-R-N scores in the mid (23%) or highest (59%) tertile. This gender imbalance reflected the higher levels of neuroticism that occur in females than males (Costa, Terracciano, & McCrae, 2001), and females' greater willingness to participate. The racial/ethnic composition was Caucasian (49%), Hispanic/Latin American (15%), African American (12%), Asian (5%), Pacific Islander (1%), mixed ethnicities (13%), and other (5%).

Measures

Data from self-report questionnaires and clinical interviews were collected annually over 10 waves: T1 (baseline) through T10. Each measure was selected in part because it had demonstrated good psychometric properties in youth (see Zinbarg et al., 2010). Participants in the first, second, and third cohorts could provide up to 10, nine, and eight waves of data, respectively.

Low Positive Emotion—The Anhedonic Depression scale of the Mood and Anxiety Symptom Questionnaire (MASQ-AD; Watson et al., 1995) measured low positive emotion over the last week (see Zinbarg et al., 2010). The MASQ-AD consists of 22 items, each on a five-point scale. Fourteen reverse-keyed items directly assess high positive emotion (e.g., "Felt really happy"); eight assess low positive emotion (e.g., "Felt like nothing was very enjoyable"). At baseline, alpha was .90.

Sociability—The Extraversion scale from the Big Five Mini Markers (Big 5-E; Saucier, 1994) measured sociability. The Big 5-E consists of eight sociability-related adjectives (e.g., “talkative”), each on a nine-point scale. Alpha at baseline was .80.

Neuroticism—Three scales measured neuroticism. The Neuroticism scale from the International Personality Item Pool-NEO-PI-R (IPIP-NEO-PI-R-N; Goldberg, 1999) consists of 60 items, each rated out of five points. It was designed to correspond with the neuroticism scale from the NEO-PI-R (Costa & McCrae, 1992). Baseline alpha for the IPIP-NEO-PI-R-N was .95. The Behavioral Inhibition Scale (BIS; Carver & White, 1994) consists of seven items measuring anxiety (often included as a facet of neuroticism in neuroticism scales, such as the IPIP-NEO-PI-R-N), each rated out of four points. BIS alpha was .75 at baseline. The Neuroticism scale from the Big Five Mini Markers (Big 5-N; Saucier, 1994) consists of eight adjectives (e.g., “moody”), each rated out of nine points. Baseline alpha was .80.

Emotional Disorders—Axis I diagnoses and clinical severity ratings (CSRs; DiNardo & Barlow, 1988) for unipolar DDs and ADs were assigned annually using the Structured Clinical Interview for *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 2002). Interviews were conducted by extensively-trained graduate and bachelor's-level research assistants (see Zinbarg et al., 2010). At baseline, the SCID assessed current and lifetime psychopathology; annual follow-up SCIDs assessed psychopathology since last interview. Interviewers were blind to past psychopathology. Only first onsets of clinically significant (CSR ≥ 4) emotional disorders occurring during the follow-up period (T5-T10) were analyzed. When predicting a given disorder outcome, cases were excluded for any history of the disorder prior to T5. The disorders were categorized as DDs (MDD, dysthymia, and/or adjustment disorder with depressed mood; n = 82 first onsets during T5-T10)[2], SAD (generalized or non-generalized type; n = 31), GAD (n = 24), and other ADs (obsessive-compulsive disorder, posttraumatic stress disorder, acute stress disorder, agoraphobia, panic disorder, specific phobia, and/or adjustment disorder with anxiety; n = 34). Audiotaped assessment of 69 cases showed SCID diagnoses had good inter-rater reliability (e.g., kappa at baseline = .82, .65, and .85 for MDD, SAD, and GAD, respectively) across sites.

Data Analyses

Data analyses were performed in Mplus version 6.11 (Muthen & Muthen, 1998-2011). Full information maximum likelihood accommodated missing data. To evaluate model goodness of fit, we used root mean square error of approximation (RMSEA; Steiger, 1989), standardized root mean square residual (SRMR), and comparative fit index (CFI; Bentler, 1990). To conclude good fit between observed data and hypothesized model, we considered the following cut offs after Hu and Bentler (1998) and Yu (2002): RMSEA ≤ 0.06, SRMR ≤ 0.08, and CFI ≥ 0.95. Given Marsh, Hau, and Wen's (2004) suggestion that these cut offs be used flexibly, we did not adhere rigidly to them. It should be noted that Hu and Bentler's

[2]Re-running our models predicting DD outcomes excluding adjustment disorders with depressed mood did not result in any meaningful changes to the pattern of results reported in our manuscript.

recommendations applied to interpreting pairs of fit indices (e.g., RMSEA and CFI), not triplets, as were used in our study.

The present analyses built on previous work applying the TSO model to the low positive emotion (Kendall et al., in press), sociability, and neuroticism (Prenoveau et al., 2011) data from T1-T4. The TSO model requires at least three waves of data, and at least two indicators of each latent construct. We used four waves of data to avoid model identification and convergence issues from fitting the TSO model to only three waves of low positive emotion data. The latent constructs in each TSO model were indicated by three continuous subscales. Continuous subscale indicators were used rather than item-level indicators to decrease the number of parameters that would otherwise have to be estimated.

Factor analyses of the baseline MASQ-AD data guided the decision to use three indicators to represent positive emotion (Kendall et al., in press). We selected a hierarchical structure consisting of a general Low Positive Emotion factor and three lower-order factors. We labeled the lower-order factors following the three subscales in the TSO model Happiness, Anhedonia, and Pride. We applied the TSO model to the general Low Positive Emotion factor indicated by the three subscales.

In prospective analyses using this TSO model, we confronted model identification and convergence problems. To resolve these issues, we removed seven items from the three-factor hierarchical model of the factor structure of the MASQ-AD, simplifying that model and improving TSO model fit. We attended to the concern that item removal could decrease model fit. If anything, item removal improved fit.[3] Our final Happiness factor consisted of eight items: “Felt like I was having a lot of fun,” “Felt really ‘up’ or lively,” “Felt really happy,” “Felt like I had a lot of energy,” “Looked forward to things with enjoyment,” “Felt optimistic,” “Felt like I had a lot to look forward to,” “Felt like I had a lot of interesting things to do.” Four items comprised the final Anhedonia factor: “Felt like nothing was very enjoyable,” “Felt withdrawn from other people,” “Felt really slowed down,” “Felt like it took extra effort to get started,” There were three items in the final Pride factor: “Was proud of myself,” “Felt like I had accomplished a lot,” “Felt really good about myself.” Subscale means over the four waves included in the TSO model were: happiness = 23.73-24.85 (standard deviation (*SD)* = 6.78-7.11), anhedonia = 6.74-7.72 (*SD* = 3.06-3.24), pride = 8.99-9.57 (*SD* = 2.87-3.07).

The anhedonia indicator had much lower loadings on the general Low Positive Emotion factor in the TSO model (standardized λ ranged from 0.33-0.36 across the four waves) than did either the happiness (standardized λ = 0.82-0.87) or pride (standardized λ = 0.88-0.95) indicators (see Table 1). We thus ran two full sets of analyses: (1) using the TSO model that included all three indicators, and (2) using the TSO model without the anhedonia indicator.

[3]We consulted the modification indices to determine which items had correlated residuals such that dropping the items would most improve fit, then examined item wording to confirm removal on a content level (i.e., redundancy with at least one item with which there was a correlated residual). Some items were removed if they did not have a strong loading on any factor in the model. Fit indices for the 15-item three-factor hierarchical model were χ^2 (75) = 225.76, $p < .001$; RMSEA = 0.058, 90% CI = [0.05-0.07]; SRMR = 0.03; CFI = 0.96. Comparing this model to one without a general factor using a chi-square difference test confirmed general factor inclusion, χ^2 (15) = 714.16, $p < .001$. A chi-square difference test showed the 15-item three-factor hierarchical model provided significantly better fit than did a 15-item one-factor solution, χ^2 (15) = 611.67, $p < .001$.

Following evidence that three versus two indicators would improve convergence rates of the TSO model of sociability (Ciesla, Cole, & Steiger, 2007), we selected three indicators. The indicators were created by dividing the Big 5-E items into three subscales, each consisting of two or three items (Prenoveau et al., 2011). The means for these subscales over T1-T4 were 5.79-5.90 (*SD* = 1.44-1.74), 5.22-5.52 (*SD* = 1.48-1.62), and 6.02-6.12 (*SD* = 1.32-1.47).

Finally, the NEO-PI-R, BIS, and Big 5-N each indicated a general Neuroticism factor to which the TSO model was applied (Prenoveau et al., 2011). The means over T1-T4 of these subscales, respectively, were 4.62-4.77 (*SD* = 1.32-1.42), 2.80-2.90 (*SD* = 0.57-0.61), and 2.61-2.75 (*SD* = 0.57-0.59). Confirmatory factor analyses of the three scales and the EPQ-R-N (used at screening) showed a single-factor model provided excellent fit to observed covariances among scales, confirming use of the three scales to indicate a general factor (Zinbarg et al., 2010).

Across-time equality constraints applied to the factor loadings of each measurement model tested metric invariance. Although important to test, the assumption of measurement equivalence across time often goes overlooked (Ciesla et al., 2007). With the 15-item positive emotion data[4], as well as sociability and neuroticism data (Prenoveau et al., 2011), imposing these constraints did not significantly decrease model fit. Temporal changes in the general Low Positive Emotion, Sociability, or Neuroticism factor were thus attributable to changes in the level of the respective latent construct.

The full TSO model provided good fit to and was the best representation of the 15-item positive emotion data with[5] or without [6] the anhedonia indicator. “Trait-only” models of the sociability and neuroticism data in which the autoregressive pathways were removed (and thus the trait factor was the only source of stable variance) were selected because including autoregressive pathways did not significantly improve model fit (Prenoveau et al., 2011). The variance attributable to the trait factor of the positive emotion data in the TSO models both with and without the anhedonia indicator was 42%. TSO models of the sociability and neuroticism data revealed that 82% and 84% of variance, respectively, was due to the trait factor.

[4]Across-time equality constraints were imposed on factor loadings in the model of the general Positive Emotion factor using subscales corresponding to the three group factors as indicators. Fit indices for the metric invariant model were χ^2 (9) = 7.42, p = .59; RMSEA = 0.000, 90% CI = [0.00-0.04]; SRMR = 0.03; CFI = 1.00. Those for the configural invariant model were χ^2 (6) = 5.41, p = 0.49; RMSEA = 0.000, 90% CI = [0.00-0.05]; SRMR = 0.01; CFI = 1.00. Results from the chi-square difference test were χ^2 (3) = 2.00, p > .05.

[5]Fit indices for the full TSO model with the anhedonia indicator were χ^2 (48) = 156.14, p < .001; RMSEA = 0.061, 90% CI = [0.05-0.07]; SRMR = 0.06; CFI = 0.96. Chi-square difference tests confirmed the full TSO model was a better representation of the data than was either a trait-only model without the auto-regressive pathways, χ^2 (1) = 12.72, p < .001, or an auto-regressive occasion-only model without the trait, χ^2 (3) = 48.55, p < .001.

[6]Fit indices for the full TSO model without the anhedonia indicator were χ^2 (14) = 49.22, p < .001; RMSEA = 0.064, 90% CI = [0.05-0.08]; SRMR = 0.03; CFI = 0.98.

Results

Prospective Associations of Low Trait Positive Emotionality with First Onsets of Emotional Disorders

The analyses involved three main steps, each run two ways: (1) using the three-indicator TSO model of the positive emotion data (i.e., including items tapping both high and low positive emotion), then (2) using the two-indicator TSO model excluding the anhedonia indicator (i.e., tapping only high positive emotion).

For Step 1, Cox proportional hazards modeling (Cox, 1972), a type of survival analysis (Singer & Willett, 2003), examined the prospective associations of low trait positive emotionality with first onsets of (a) DDs, (b) SAD, (c) GAD, and (d) the combined remaining ADs. We used a separate model to regress each disorder outcome variable onto the low positive emotion trait factor from the TSO model. In each case, a single model thus combined TSO and Cox proportional hazards models. The low trait positive emotionality hazard did not differ significantly across time for any disorder outcome, indicating its effects were consistent over time. In the three-indicator TSO model, low trait positive emotionality significantly predicted DDs, SAD, and GAD, but not other ADs (see Table 2). Comparing inferential confidence intervals (Tryon, 2001) revealed that the strength of the relations between low trait positive emotionality and each disorder category, including the other ADs, did not differ significantly from each other.

Removing the anhedonia indicator from the TSO model did not meaningfully change the predictions of SAD, GAD, or the remaining ADs (see Table 3). The direction and magnitude of the DD hazard ratio (HR) was roughly similar, but the effect was no longer significant. We again found no significant difference in the strength of relations between low trait positive emotionality and each of the disorder outcomes.

Follow-up analyses addressed DD-AD co-occurrence. We re-ran the Step 1 models predicting DD outcomes, eliminating cases with any AD during or before the time positive emotion was measured. Effects of low trait positive emotionality, modeled with all three indicators, remained at least as strong and significant: HR = 1.56, 95% CI = [1.07-2.27], $p < .05$. Without the anhedonia indicator, the effect was again at least as strong and was significant: HR = 1.46, 95% CI = [1.01-2.12], $p < .05$. Conversely, the effects of low trait positive emotionality, modeled with all three indicators, were reduced and non-significant for each AD outcome after adjusting for DDs during or before the time positive emotion was measured: SAD HR = 1.57, 95% CI = [0.70-3.52], $p = 0.28$; GAD HR = 1.94, 95% CI = [0.74-5.09], $p = 0.18$; other AD HR = 1.36, 95% CI = [0.71-2.59], $p = 0.35$. When low trait positive emotionality was modeled without anhedonia, SAD HR = 1.29, 95% CI = [0.56-2.93], $p = 0.55$; GAD HR = 1.75, 95% CI = [0.68-4.54], $p = 0.25$; other AD HR = 1.31, 95% CI = [0.68-2.51], $p = 0.41$.

Accounting for Trait Sociability and Neuroticism

Steps 2 and 3 determined whether low trait positive emotionality predicted first emotional disorder onsets, above and beyond trait sociability (Step 2) or neuroticism (Step 3). Low trait positive emotionality, modeled with three indicators, had significant zero-order

correlations with trait sociability ($r = -.65$, $p < .001$) and neuroticism ($r = .67$, $p < .001$). Results were similar for correlations of low trait positive emotionality, modeled without the anhedonia indicator, with trait sociability ($r = -.66$, $p < .001$) and neuroticism ($r = .65$, $p < .001$).

Each original model from Step 1 was re-run with low trait positive emotionality correlated with trait sociability, and with each disorder onset variable regressed onto both traits. The models were then run again with trait neuroticism replacing trait sociability. In interpreting the influence of the personality covariates on the prospective effects of low trait positive emotionality, we gave more consideration to effect sizes than *p*-values (e.g., Cohen, 1990).

At Step 2, accounting for trait sociability decreased the relation between low trait positive emotionality, modeled with all three indicators, and SAD to 41% and non-significance (see Table 2). Reduced to 97% of its original value, the relation between low trait positive emotionality and the other ADs category was no longer significant. The relations of low trait positive emotionality with DDs and GAD remained at least as large, although the respective significance values of $p = .05$ and .03 changed to $p = .07$ and .08. The associations between trait sociability and disorder onsets were non-significant in these models: DD HR = 1.28, 95% CI = [0.86-1.91], $p = .23$; SAD HR = 0.60, 95% CI = [0.23-1.56], $p = .30$; GAD HR = 1.35, 95% CI = [0.66-2.76], $p = .41$; other AD HR = 1.07, 95% CI = [0.54-2.09], $p = 0.85$.

Re-running the Step 2 models without the anhedonia indicator produced generally comparable results (see Table 3). Sociability continued to have non-significant prospective associations: DD HR = 1.20, 95% CI = [0.81-1.77], $p = .36$; SAD HR = 0.58, 95% CI = [0.21-1.61], $p = .29$; GAD HR = 1.27, 95% CI = [0.62-2.62], $p = .51$; other AD HR = 1.06, 95% CI = [0.54-2.07], $p = 0.87$.

At Step 3, entering trait neuroticism into the models largely accounted for the relations between low trait positive emotionality and all the emotional disorder onsets (see Table 2). Trait neuroticism predicted each disorder category in these models: DD HR = 2.08, 95% CI = [1.40-3.08], $p < .001$; SAD HR = 3.40, 95% CI = [1.34-8.65], $p = .01$; GAD HR = 2.88, 95% CI = [1.23-6.73], $p = .02$; other AD HR = 2.33, 95% CI = [1.16-4.67], $p = .02$.

Re-running the Step 3 models without the anhedonia indicator (see Table 3) did not meaningfully change the results. Trait neuroticism predicted each disorder category: DD HR = 2.09, 95% CI = [1.43-3.07], $p < .001$; SAD HR = 3.36, 95% CI = [1.35-8.34], $p < .01$; GAD HR = 2.68, 95% CI = [1.19-6.03], $p = .02$; other AD HR = 2.27, 95% CI = [1.16-4.43], $p = .02$.

Discussion

This study provided new evidence that low trait positive emotionality prospectively predicted the initial development of DDs, SAD, and GAD. Additional analyses revealed that the prospective associations were largely driven by the part of low trait positive emotionality that overlapped with trait neuroticism.

The initial results showing that low trait positive emotionality significantly predicted first onsets of DDs, SAD, and GAD, but not the remaining ADs, largely coincided with prior cross-sectional investigations (e.g., Mineka et al., 1998; Prenoveau et al., 2010). Evidence was mixed whether low trait positive emotionality operated as a specific risk factor. It did not significantly predict the remaining ADs category (or any of the AD categories in follow-up analyses that accounted for DD history). However, no significant differences arose in the magnitude of its relations with DDs, SAD, GAD, or the remaining ADs.

Entering trait sociability and neuroticism into the models revealed that it was the part of low trait positive emotionality overlapping with trait neuroticism that largely predicted disorder onsets. After accounting for trait sociability, the point estimate of the effect size was substantially reduced only for the relation between low trait positive emotionality and SAD, but not for any of the other disorder outcomes. Although the *p*-values for the other outcomes became non-significant, following Cohen (1990), we emphasized effect sizes over significance values. When trait neuroticism entered the models, the unique relation between low trait positive emotionality and each disorder category largely disappeared. This was consistent with earlier prospective work showing that low extraversion significantly predicted first DD onsets due to its overlap with neuroticism (Kendler et al., 2006).

Throughout the study, the pattern of results was not meaningfully changed by removal of the anhedonia indicator (i.e., the items explicitly tapping low positive emotion) from the positive emotion measure. For instance, the zero-order effects of low trait positive emotionality on SAD and GAD remained at least as large and significant after this removal. The zero-order effect of low trait positive emotionality on DDs was reduced by approximately 31% and became non-significant after the anheodnia indicator was removed. However, we are not inclined to emphasize this reduction given findings from our follow-up models addressing disorder co-occurrence, in which DDs were predicted after eliminating cases positive for any AD history. In these models, the prospective effect of low trait positive emotionality, either with or without the anhedonia indicator, was significant and at least as large as that from the original zero-order model of low trait positive emotionality, modeled with all three indicators, predicting DDs.

Ultimately, our findings did not provide support for our speculation that youth low on positive emotionality have fewer resources to buffer against the effects of stress during adolescence and young adulthood, increasing their risk for developing disorders in response to stress. However, we did not test if low positive emotionality interacted with high neuroticism to predict disorders (we attempted these analyses, but our interaction models did not terminate normally). Future research should examine this possibility.

Low trait positive emotionality and trait neuroticism were strongly related in our study, perhaps explaining why the strength of the associations between low trait positive emotionality and DDs, SAD, and GAD did not differ significantly from the association with the remaining ADs. Given that low positive emotion has been cross-sectionally associated with DDs, SAD, and symptoms of GAD after adjusting for neuroticism, our results could imply that low positive emotion is an effect of the disorders, rather than low trait levels

being a cause. However, firm conclusions cannot be drawn from our study design. This possibility merits future investigation.

It is interesting to consider our findings in light of Caspi et al. (2014), who introduced a General Psychopathology factor, or *p factor*. The authors examined the longitudinal structure of psychopathology from adolescence to midlife. They found evidence for three intermediate breadth factors—Internalizing (e.g., unipolar DDs), Externalizing (e.g., substance use disorders), and Thought Disorders (e.g., schizophrenia)—and for a general p factor. Caspi et al. speculated that it is difficult to find risk factors specific to disorders because most disorder variance is due primarily to p, leaving little specificity among disorders to be predicted in the first place. Low trait positive emotionality might not have uniquely related to specific disorder onsets in our study because of the strong associations of disorders with the underlying p factor, which was not measured.

Our findings diverged from those of Dougherty et al. (2010), who showed low positive emotionality prospectively predicted higher depressive symptoms in children, even after adjusting for neuroticism. We offer three possible explanations.

The first is rooted in differences in measures of positive emotionality. Dougherty et al. (2010) employed multiple observational measures, including maternal reports and laboratory and naturalistic observations. Those methods could have captured dimensions of the construct that predicted depression after accounting for neuroticism. On the other hand, their study did not use trait-state decomposition to isolate stable trait variance, leaving their measure contaminated by unknown amounts of state variance. It is possible that low levels of state positive emotion—rather than a premorbid personality risk marker—drove their relation with subsequent depression.

Second, the discrepant findings could have resulted from differences in outcomes. Dougherty et al. (2010) used dimensional symptom outcome measures in a non-clinical sample, whereas we employed categorical outcomes of clinically significant disorder onsets. As noted, neuroticism is a risk marker for emotional disorders. The relation between neuroticism and depression could be curvilinear and accelerated, such that neuroticism levels are lower among people with subclinical versus clinical depression. If true, there could have been more room in the Dougherty et al. sample for low positive emotionality to emerge as a risk marker of depression independent of neuroticism.

Finally, the difference could be attributable to the age groups sampled. Dougherty et al. (2010) examined children; we sampled late adolescents and young adults. Neuroticism typically increases during childhood and mid-adolescence, then decreases from late adolescence (Trzesniewski, Robins, Roberts, & Caspi, 2004). Dougherty et al.'s younger age range could have resulted in lower neuroticism among depressed youth than in our sample. Accordingly, the prospective relation of low positive emotionality with disorders could have emerged above and beyond neuroticism in their study, if the relation between neuroticism and depression weakens at lower neuroticism levels.

Turning to limitations, our having oversampled for neuroticism necessarily limits the generalizability of our findings. Oversampling could have inadvertently biased our results in

favor of neuroticism and against positive emotionality. Importantly, a simulation study directly addressing these issues showed that oversampling does not result in substantially biased effect sizes relative to correlated variables not directly oversampled (Hauner et al., 2014). In finding no bias for the directly oversampled variable, the study demonstrated that there was no differential bias for this variable compared to those indirectly oversampled.

Another limitation is that positive emotion was measured with state instructions in our study, but sociability and neuroticism with trait instructions. Indeed, in the TSO models of these constructs, the variance attributable to the trait factor of the sociability or neuroticism data was approximately twice that due to the trait factor of the positive emotion data. By isolating the stable variance in longitudinal data, however, TSO modeling produces a valid trait measure whether data were collected using trait or state directions. Critically, we did not relate the observed measures of positive emotion, sociability, and neuroticism to disorder onsets. Rather, we related the trait factors from TSO models of these constructs to disorders.

Next, low trait positive emotionality substantially overlapped with trait neuroticism, potentially impeding the former in showing incremental predictive power beyond the latter, limiting generalizability. Conversely, such overlap could also have impeded trait neuroticism from showing unique predictive power above and beyond low trait positive emotionality. This did not occur, demonstrating there was power left for unique effects.

It should be acknowledged that parceling (e.g., constructing multiple indicators from MASQ-AD items) is controversial (see Little, Rhemtulia, Gibson, & Schoemann, 2013). We did not, however, seek to test the factor analytic structure of our measures, as these structures were previously established. We recommend future research build on our work by using multiple methods to assess personality traits.

Our study would have also benefited from assessing a broader range of personality facets. The facets of low positive emotion tapped (happiness, pride, and anhedonia) have been identified as meaningful in the study of positive emotion and depression (e.g., Gruber, Oveis, Keltner, & Johnson, 2011; Joormann & Gotlib, 2006). However, they did not capture potentially important differences in the reward processing dimensions of positive emotionality—in particular, anticipation versus consumption. Similarly, we did not co-vary for facets of extraversion other than sociability because the larger study from which our data were derived did not include other extraversion measures. This is an important direction for future research, as illustrated by recent findings that correlations between different facets of extraversion and emotional disorders ranged from strongly negative to moderately positive (see Watson et al., in press).

A final limitation concerns the influence of past and current symptoms on our measure of trait positive emotionality. When predicting each disorder outcome, we excluded participants with a lifetime history of that disorder, or occurrence of the disorder during positive emotion measurement. We did this to rule out differences in trait measurement reflecting the effects of past or current disorders. However, it remains possible that low

levels of trait positive emotionality associated with past or current symptoms of subclinical disorders could have influenced trait measurement.

In conclusion, our initial models provided the first evidence of which we are aware that low positive emotionality prospectively predicted first onsets of DDs, SAD, and GAD. Additional analyses demonstrated the importance of accounting for related personality traits, particularly neuroticism, in longitudinal investigations of positive emotionality. In these ways, our work contributes to a larger literature describing the prospective effects of personality on the initial development of emotional disorders.

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General Scientific Summary

This study provided the first evidence of which we are aware that low positive emotionality prospectively predicted the initial development of depressive disorders, social anxiety disorder, and generalized anxiety disorder. Follow-up analyses revealed that the risk effects were largely accounted for by the overlap of low positive emotionality with neuroticism.

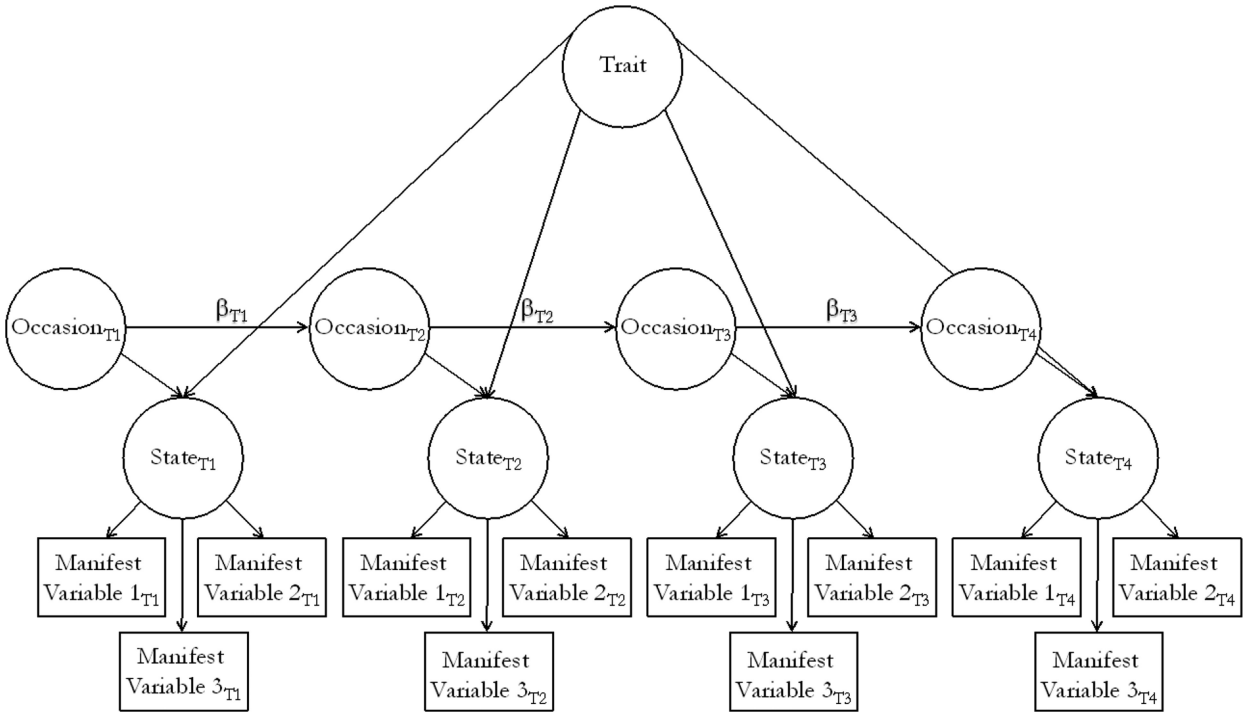

Figure 1.

Visual depiction of the trait-state-occasion (TSO) model for a construct measured over four time points (adapted from Cole et al., 2005). β = Autoregressive pathway coefficient. Subscripts indicate time points. Errors terms have been omitted for clarity.

Table 1

Standardized Path Estimates for Trait-State-Occasion (TSO) Models of Positive Emotion

Time (T)	Trait→State	$Occasion_T$→$Occasion_{T+1}$	Occasion→State	State→Happiness	State→Anhedonia	State→Pride
*Three-Indicator TSO (Includes Items Tapping **Both High and Low Positive Emotion**)*						
1	0.58	0.26	0.81	0.82	0.33	0.90
2	0.63	0.24	0.78	0.84	0.33	0.88
3	0.62	0.24	0.78	0.84	0.33	0.93
4	0.70	-	0.71	0.87	0.36	0.95
*Two-Indicator TSO (Includes Items Tapping **Only High Positive Emotion**)*						
1	0.59	0.27	0.81	0.82	-	0.90
2	0.62	0.26	0.78	0.84	-	0.89
3	0.61	0.25	0.79	0.84	-	0.93
4	0.70	-	0.72	0.86	-	0.96

Note. All paths were significant at $p < .05$. The Trait→State, State→Happiness, State→Anhedonia, and State→Pride paths represent factor loadings. In each TSO model, across-time equality constraints were imposed on the pathways between occasion factors, from occasion factors to state factors, and from each state factor to each subscale. The variance in the trait factor was set to 1, as was that in the first occasion factor; the remaining occasion variances were set equal to each other. The residual variance for each state factor was set to 0.

Table 2

Low Trait Positive Emotionality, Assessed with Items Tapping **Both High and Low Positive Emotion**, Prospectively Predicting First Onsets of the Emotional Disorders

Model	Hazard Ratio	95% Confidence Interval	*p*-value
Depressive Disorders	1.42	1.01-2.01	0.05
Social Anxiety Disorder	2.47	1.02-5.98	0.05
Generalized Anxiety Disorder	2.02	1.08-3.80	0.03
Other Anxiety Disorders	1.70	0.98-2.95	0.06
Accounting for Trait Sociability			
Depressive Disorders	1.57	0.96-2.56	0.07
Social Anxiety Disorder	1.60	0.44-5.80	0.48
Generalized Anxiety Disorder	2.18	0.92-5.20	0.08
Other Anxiety Disorders	1.68	0.76-3.69	0.20
Accounting for Trait Neuroticism			
Depressive Disorders	1.01	0.65-1.57	0.96
Social Anxiety Disorder	0.71	0.31-1.62	0.42
Generalized Anxiety Disorder	0.98	0.39-2.46	0.96
Other Anxiety Disorders	0.94	0.45-1.96	0.86

Table 3

Low Trait Positive Emotionality, Assessed with Items Tapping **Only High Positive Emotion**, Prospectively Predicting First Onsets of the Emotional Disorders

Model	Hazard Ratio	95% Confidence Interval	*p*-value
Depressive Disorders	1.29	0.91-1.82	0.15
Social Anxiety Disorder	2.56	1.04-6.28	0.04
Generalized Anxiety Disorder	2.09	1.10-3.95	0.02
Other Anxiety Disorders	1.69	0.97-2.93	0.06
Accounting for Trait Sociability			
Depressive Disorders	1.40	0.87-2.24	0.17
Social Anxiety Disorder	1.47	0.36-6.00	0.59
Generalized Anxiety Disorder	1.97	0.82-4.74	0.13
Other Anxiety Disorders	1.66	0.75-3.66	0.21
Accounting for Trait Neuroticism			
Depressive Disorders	0.99	0.65-1.52	0.98
Social Anxiety Disorder	0.71	0.32-1.55	0.39
Generalized Anxiety Disorder	1.09	0.44-2.68	0.85
Other Anxiety Disorders	0.97	0.48-1.99	0.94