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Regular Article

Dispositional threat sensitivity as a liability for fear-related pathologies: Evidence from a child-aged twin sample

Chelsea K. Sawyers[1], Ashlee A. Moore[2], Christopher J. Patrick[3], James R. Yancey[4], Melissa A. Brotman[5], Ellen Leibenluft[5], Daniel S. Pine[5], Roxann Roberson-Nay[1], Mark D. Kramer[6] and John M. Hettema[7]

[1]Department of Psychiatry, Virginia Institute for Psychiatry and Behavioral Genetics, Virginia Commonwealth University, Richmond, VA, USA, [2]Department of Psychology, State University of New York, Oswego, NY, USA, [3]Department of Psychology, Florida State University, Tallahassee, FL, USA, [4]Department of Psychiatry, University of Utah, Salt Lake City, UT, USA, [5]Emotion and Development Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD, USA, [6]Department of Psychology, University of Minnesota–Twin Cities, Minneapolis, MN, USA and [7]Department of Psychiatry and Behavioral Sciences, Texas A&M Health Science Center, Bryan, TX, USA

Abstract

Threat sensitivity, an individual difference construct reflecting variation in responsiveness to threats of various types, predicts physiological reactivity to aversive stimuli and shares heritable variance with anxiety disorders in adults. However, no research has been conducted yet with youth to examine the heritability of threat sensitivity or evaluate the role of genetic versus environmental influences in its relations with mental health problems. The current study addressed this gap by evaluating the psychometric properties of a measure of this construct, the 20-item Trait Fear scale (TF-20), and examining its phenotypic and genotypic correlations with different forms of psychopathology in a sample of 346 twin pairs (121 monozygotic), aged 9–14 years. Analyses revealed high internal consistency and test-retest reliability for the TF-20. Evidence was also found for its convergent and discriminant validity in terms of phenotypic and genotypic correlations with measures of fear-related psychopathology. By contrast, the TF-20's associations with depressive conditions were largely attributable to environmental influences. Extending prior work with adults, current study findings provide support for threat sensitivity as a genetically-influenced liability for phobic fear disorders in youth.

Keywords: anxiety disorders; development; fear; threat sensitivity; twins

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Introduction

Anxiety disorders are common in the general population, with lifetime prevalence estimates reaching as high as 28% (Baxter et al., 2013). Many anxiety disorders have an average onset before age 15 (de Lijster et al., 2017). Thus, research aimed at understanding etiological factors that give rise to anxiety disorders in childhood and early adolescence has important implications for assessments and interventions for these conditions. In line with the idea of fearfulness and anxiety-proneness as distinct trait dispositions (Sylvers et al., 2011), there is evidence that anxiety disorders entailing excessive fear are distinguishable from those involving nonspecific distress and pervasive negative affect. The former are associated with hyperreactivity to threat cues and avoidance behavior, the latter more with dysphoric mood and depressive symptoms (Watson, 2005). The current work focuses on the construct of threat sensitivity as a specific liability for fear-related psychopathology. Specifically, we sought to extend prior twin research demonstrating a heritable basis for threat sensitivity's association with phobic-fear symptomatology in adulthood to younger-aged individuals (i.e., children aged 9 to 14).

An important referent for the current work is research demonstrating that anxiety disorders entailing excessive fear (specific phobia, social anxiety disorder, panic disorder, agoraphobia) are highly comorbid and can be organized along a coherent "*phobic fear*" dimension. This dimension is distinct from, though correlated with, a "*dysphoric distress*" dimension reflecting symptoms of low mood and pervasive negative affect and cognitions, associated with disorders including major depression, persistent depressive (dysthymic) disorder, generalized anxiety disorder (GAD), and post-traumatic stress disorder (Kotov et al., 2017, 2021). Twin-biometrical modeling studies utilizing both adult and youth twin samples have found that unique additive genetic influences contribute to variance in fear disorder symptomatology and related individual difference measures (Hettema et al., 2020; Kendler et al., 1995; Lahey et al., 2017). Moreover, research employing psychophysiological measures has shown that patients with fear disorders, but not dysphoric distress disorders, tend to exhibit enhanced potentiation of the startle reflex – a well-established measure of limbic threat reactivity in humans as well as animals (Lang and Davis, 2006) – during exposure to aversive cues (Vaidyanathan et al., 2009b; see also Vaidyanathan et al., 2009a).

Corresponding author: John Hettema; Email: hettema@tamu.edu

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This finding has been observed across fear-related traits and psychological disorders involving excessive fear (for a review, see (Vaidyanathan et al., 2009a), suggesting that these characteristics/conditions may have a basis in common underlying neurophysiological mechanisms along with shared genetic influences. Taken together, these lines of research converge on the notion that fear-related disorders share a common premorbid liability factor that confers specific risk for disorders of this type and is at least partially rooted in neurobiology.

Operating from this perspective, Kramer et al. (2012) applied structural equation and biometrical modeling to adult twin data for a number of well-established self-report scale measures of fear and fearlessness as a basis for conceptualizing this trait domain and characterizing it etiologically and neurobiologically. The scales selected for their analysis were ones that prior research had shown to be related to aversive startle potentiation, a physiological-reflex measure of threat sensitivity (Vaidyanathan et al., 2009b). Kramer et al.'s structural analysis revealed a broad bipolar dimension, termed "trait fear" by the authors, accounting for appreciable variance in all scales. A biometrical analysis revealed that additive genetic influences accounted for 51% of the variance in scores on this broad fear/fearlessness factor. Additionally, Kramer et al. presented evidence that scores on this trait fear dimension were positively related to aversive startle potentiation, establishing linkage with biobehavioral threat sensitivity.

Using data for another adult twin sample, Yancey et al. (2015) showed that increased aversive startle potentiation in persons with a history of one or more fear disorder diagnoses (specific phobia, social anxiety disorder, panic disorder, agoraphobia; cf. Vaidyanathan et al., 2009b), relative to those with no fear disorder history, was accounted for by elevated trait fear scores. Interestingly, the association between fear disorder history and startle potentiation, and trait fear's mediating role in this association, was evident only for participants with no history of major depression. The implication is that trait fear shares a component of biological threat sensitivity with fear-related psychopathology that is distinct from internalizing disorders characterized by dysphoric distress.

Subsequent to this, Yancey et al., (2016) demonstrated in the same sample that scores on a scale measure of trait fear covaried with other physiological indicators of threat sensitivity besides startle potentiation (i.e., cardiac, facial electromyographic) and that scores for these different physiological indicators could be combined together with fear-scale scores into a biobehavioral index of dispositional fear. Scores on this biobehavioral fear index were positively predictive of both clinician-assessed fear disorder symptoms and separate measures of threat sensitivity including electrocortical measures. A further study by Venables et al. (2017) applied twin-biometrical modeling to data for this twin sample and showed a very high genetic correlation (~ .8) for the biobehavioral fear index with fear disorder symptom scores, compared to moderate and negligible genetic correlations, respectively, with dysphoric and substance disorder symptoms.

The foregoing lines of evidence point to an individual difference dimension of threat sensitivity as a specific liability for fear-related psychopathology. However, research to date supporting this view has been limited to adult samples. Accordingly, there is a need to evaluate threat sensitivity's association with fear disorder symptomatology in youth and characterize the role of genetic versus environmental influences in this association. We know from prior research that precursors to certain adult personality traits, conceptualized as early temperament characteristics, appear to be prominently heritable and biologically based (Buss and Plomin, 1984). Sensitivity to fearful or threatening stimuli has been identified as one such characteristic in prominent theories of temperament (Buss and Plomin, 1986; Goldsmith and Campos, 1982; Kagan, 1997; Kochanska et al., 2007; Kochanska, 1997; Tellegen, 1985). Operating from this temperament-based conceptualization, the current study employed data for a child-aged twin sample to examine the diagnostic correlates of dispositional threat sensitivity and examine the role of genetic versus environmental influences in its relationship with fear disorders compared to other disorders. We first assessed the internal psychometric properties of an established measure of threat sensitivity, the 20-item Trait Fear scale (TF-20; Kramer et al., 2020), in terms of its item-level structure, internal consistency, and reliability in our child-aged study sample. Next, we evaluated the specificity of the TF-20's relations with fear disorder symptomatology, relative to dysphoric-distress and externalizing disorder symptomatology. Finally, we examined its underlying latent genetic and environmental structure in relation to internalizing disorders in youth, disaggregating the shared versus specific etiological influences across these two domains (i.e., trait scores and symptom dimensions) using twin-biometrical modeling.

Method

Participants

Data for the current study derived from the Virginia Commonwealth University Juvenile Anxiety Study, a twin study of anxiety and related phenotypes in White children aged 9–14 (692 individuals from 121 monozygotic [MZ] and 225 dizygotic [DZ] pairs). Twin pairs were recruited through the Mid-Atlantic Twin Registry (MATR; Lilley and Silberg, 2013) and enrolled at either the VCU site or the National Institute of Health (NIH) site. Zygosity was determined using parental responses to standard questions about physical appearance for all 346 twin pairs and DNA testing for a subset of 13 pairs, with results for the two methods converging perfectly in that subset of participants (for details, see Carney et al., 2016). A variety of self- and parent-report measures were collected via REDCap (Research Electronic Data Capture; Harris et al., 2009). A masters or doctoral level trained clinician administered the Kiddie Schedule for Affective Disorders and Schizophrenia-Present & Lifetime Version (KSADS-PL; Kaufman et al., 1997) in relation to each twin through a face-to-face interview with the parent(s).

Following the initial visit, a portion of participants ($n = 241$) were re-assessed to evaluate test-retest reliability. Six of these with intervals exceeding 90 days between visits were excluded from reliability analyses, resulting in retest data for 235 children, for whom the mean time between visits was 25.9 days ($SD = 10.19$; range = 11–70). The subset of participants who returned for visit 2 did not differ significantly from the overall visit 1 sample in terms of sex, age, or zygosity ($ps = .65$, .80, and .82, respectively).

The Institutional Review Boards at VCU and NIH approved the study, and all participants provided informed consent (parents) and assent (children) prior to testing. Twins and parents were monetarily compensated for their participation in the study.

Measures

20-Item trait fear inventory (TF-20)

The TF-20 (Kramer et al., 2020) is an abbreviated version of a 44-item self-report scale developed to index the broad

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fear/fearlessness (dispositional threat sensitivity) dimension identified by Kramer et al. (2012). Each item is rated on a 4-point Likert scale, from 0 (false) to 3 (true). Items indexing fearlessness were reversed scored, and a sum score was computed for the scale as a whole – with possible total scores ranging from 0–60 and higher scores representing greater fearfulness. Kramer et al. (2020) reported that scores on this scale correlated robustly with fear disorder symptomatology, at a level ($r = .44$) that exceeded its associations with dysphoric/distress and substance use symptomatology ($rs = .29$ and $-.16$).

Other measures

Other child self-report measures administered to participants included: the Revised Fear Survey Schedule for Children (FSSC-R; Ollendick, 1983), the Screen for Child Anxiety Related Disorders (SCARED; Birmaher et al., 1999), Carver and White's (1994) Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) scales, the Affective Reactivity Index (ARI; Stringaris et al., 2012), the Short Mood and Feelings Questionnaire (SMFQ; Angold et al., 1995), the Junior Eysenck Personality Questionnaire (JEPQ; Eysenck, 1965), and the Child Anxiety Sensitivity Index (CASI; Silverman et al., 1991). Parent-report measures included: the Behavioral Inhibition Questionnaire (BIQ; Gensthaler et al., 2013), the Inventory of Callous-Unemotional Traits (ICU; Kimonis et al., 2008), and the Child Behavior Checklist (CBCL; Achenbach and Rescorla, 2001), along with parent-adapted versions of the SCARED, SMFQ, and ARI (see "Descriptions of Study Measures" section of the online Supplement for information regarding these measures, and Supplemental Table A for *M*s/*SD*s and reliability coefficients [α, test-retest] for each).

Statistical analyses

Exploratory structural equation modeling (ESEM), implemented via MPlus version 7.31 (Muthén and Muthén, 1998–2015) with adjustment for potential clustering of responses within families to account for non-independence of twin observations, was used to characterize the factor structure of the TF-20 (Kramer et al., 2020). We also conducted reliability and validity analyses of the TF-20 using the R software package (R Core Team, 2015). Test-retest reliability of TF-20 sum scores between visit 1 and visit 2 was evaluated using Pearson's product-moment correlation. In addition, we estimated internal consistency reliability of scores for each assessment visit via Cronbach's α, using the *psych* package in R. To assess convergent and discriminant validity, we computed correlations between sum scores for the TF-20 at visit 1 and scores for the various other construct-relevant measures administered at that initial visit. We also examined criterion-related validity in terms of polyserial correlations between TF-20 sum scores and current and past psychiatric diagnoses from the KSADS. To verify that findings from these reliability and validity analyses were not overly affected by twin dependency, we compared results for all participants with results for a subsample consisting of one twin randomly selected from each pair.

To further evaluate the criterion-related validity of the TF-20, we examined its relations with correlated factors corresponding to phobic fear and dysphoric distress, derived from a confirmatory factor analysis (CFA; conducted in Mplus version 8.6) of child-report scores for the following scales: FSSC-R, SCARED, and BIS as indicators of phobic fear, and ARI, SMFQ, and JEPN as indicators of dysphoric distress. Regression analysis was used to quantify relations of TF-20 scores with each factor of this model when controlling for its overlap with the other factor. Additionally, we added TF-20 sum scores to the model as a fourth indicator of the latent fear factor to evaluate its loading on this factor. Twin interdependency was accounted for in these analyses by adjusting for clustering of responses within families, and participant sex and age were included as covariates to account for any potential influence of these variables.[1]

Within twin pairs, we applied biometrical structural equation modeling (SEM; Neale and Cardon, 1992) to decompose the observed variance of TF-20 scores into additive genetic, common environmental, and unique environmental effects. Additive genetic effects (A) reflect the combined contributions of different gene alleles to an observed trait. Common environment effects (C) comprise nongenetic influences that make twins more similar to each other compared to the general population. Unique environmental effects (E) represent influences that contribute to observed differences between co-twins and include measurement error. Using this modeling approach, we estimated the heritability of the TF-20 (i.e., proportion of variance in scores attributable to additive genetic influences) and the extent to which its observed covariance with child-reported fear measures (FSSC-R, SCARED, BIS) could be accounted for by overlapping genetic versus environmental influences. A common pathway model was then used to estimate the etiologic overlap of the TF-20 with the aforementioned fear and dysphoria latent factors.[2]

We conducted these twin biometric modeling analyses using the OpenMx package (Neale et al., 2016) in R. Customary alternative models consisting of ACE, AE, CE, and E were specified. Variance components for each of these twin models were estimated using the direct symmetric approach recommended by Verhulst et al. (2019), in which variance-covariance matrices for variance components are estimated directly, without constraints. Participant sex and age were included as covariates to account for any potential influence of these variables. Model parameters reflecting genetic and environmental sources of variance were sequentially constrained to zero to determine the best-fitting, most parsimonious model. Comparative fit was evaluated using the likelihood ratio χ^2 and Akaike information criterion (AIC) statistics.[3]

Results

Sample characteristics and basic statistics for TF-20 scores

The mean age of participants in the analytic sample at visit 1 was 11.23 ($SD = 1.43$; range = 9–14.5), with 52.4% being female. The mean TF-20 scores at visit 1 and visit 2 were 29.8 ($SD = 10.5$; range = 2–59) and 28.6 ($SD = 12.1$; range = 0–59), respectively. Figure 1 displays the distribution of TF-20 sum scores in the visit 1 sample. The distribution of scores for the sample as a whole approximated normality (skew = −.11; kurtosis = −.36).

[1]We conducted alternative versions of this analysis, and the twin biometric analyses described next, without including sex and age as covariates, and found results highly similar to those reported here.

[2]A common pathway modeling approach was used because we sought to specify two *a priori* latent factors corresponding to fear and dysphoric symptomatology. A Cholesky decomposition approach would not have served our aims because it focuses on observed measures rather than latent factors.

[3]Code and output for our modeling analyses, run in Mplus and OpenMx, can be accessed online at the following Open Science Framework (OSF) location: https://osf.io/5yjvh/. Shareable data for families who granted permission can be accessed at: https://nda.nih.gov/edit_collection.html?id=2131

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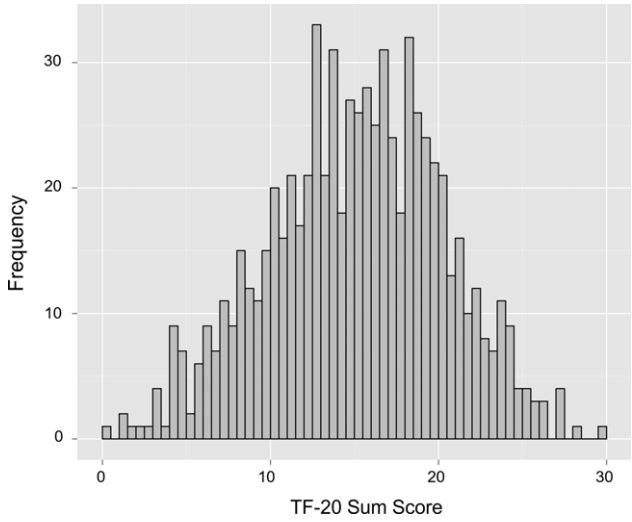


Figure 1. Distribution of TF-20 sum scores in the overall sample (Visit 1).

Factor structure of TF-20 items

We conducted an ESEM corrected for non-independence of twins using TF-20 data from the visit 1 assessment. Based on an initial exploratory factor analysis revealing the presence of one large factor and two much smaller factors underlying the TF-20 item set, we specified a bifactor ESEM with geomin rotation in which items loaded onto a general factor (corresponding to dispositional threat sensitivity; Kramer et al., 2020) and two orthogonal subfactors. The model fit well (CFI = .962, TLI = .946, RMSEA = .035 [90% CI: .028–.041]), with all but one item loading above .3 on the general factor. Table 1 lists the wording of the items and their loadings on the general factor of this model.

Test-retest reliability and internal consistency

The correlation between TF-20 total scores across visits 1 and 2 was $r = .83$ (95% CI: .79–.87), indicating good test-retest reliability for the TF-20 total score.[4] Internal consistencies for the TF-20 total at each visit were similarly high: $\alpha = .86$ (95% CI: .85–.88) for visit 1, and .90 (95% CI: .89–.92) for visit 2.[5]

Convergent & discriminant validity

Table 2 lists between-subject correlations between TF-20 total scores and the various continuous-score criterion measures at the initial (T1) assessment point ($N = 692$). Providing evidence for convergent validity, the TF-20 sum score was strongly and significantly correlated with child-report scales that tap fear symptomatology and associated processes: FSSC-R (.63), BIS (.56), SCARED (.52), and CASI (.44). Indicative of discriminant validity, correlations with child-report scales assessing dysphoric/depressive symptoms (SMFQ, ARI) were significantly lower ($rs = .27$ and .23, respectively) than those for fear symptom scales (i.e., Steiger's Z-test for dependent correlations was significant at $p < .001$ for all comparisons).

The TF-20's correlations with parent-report versions of the BIS and SCARED (i.e., BIQ and parent-SCARED) were also significant, but expectably smaller ($rs = .23$ and .27, respectively) given that TF-20 scores were available only in child self-report form. Corresponding correlations with parent-report versions of the SMFQ and ARI were weak and nonsignificant ($rs = .07$ and −.01). The TF-20's correlation with another measure of dysphoric symptomatology collected from parents only, the Withdrawn/Depressed scale of the CBCL, was .13, $p < .005$.

Among clinical diagnoses (Table 3), TF-20 scores were positively correlated with the following current or past diagnoses: specific phobia, social phobia, separation anxiety, and GAD ($rs = .16$–$.32$, all $ps < .05$). The TF-20 was not associated significantly with diagnoses of major depression or post-traumatic stress disorder, or with any externalizing disorder diagnosis.

Additional support for convergent validity was provided by results of the CFA we conducted using the FSSC-R, SCARED, and BIS as indicators of a phobic fear symptom dimension and the SMFQ, ARI, and JEPQ-N as indicators of a dysphoria/distress symptom dimension. The 2-factor model specifying these two symptom dimensions as correlated latent factors provided acceptable fit to the data (CFI = .980, TLI = .931, RMSEA = .079 [90% CI: .058–.101]). Regression analyses revealed a very strong positive relationship for the fear factor of this model with TF-20 scores ($B = .96$, $p < .001$) when accounting for its overlap (covariation) with the dysphoria factor, in contrast with a modest *negative* relationship for the dysphoria factor when accounting for its overlap with the fear factor ($B = -.33$, $p < .001$). Additionally, when added to the model as an indicator of the latent fear factor, the TF-20 sum-score measure evidenced a very strong loading on this factor (.65). The factor loadings for this CFA model with and without the TF-20 are listed in Supplemental Table B.

As shown in Table 2, discriminant validity was evidenced by small negative correlations for the TF-20 with measures of externalizing symptoms (i.e., parent-rated CBCL rule-breaking and aggressive behavior scales, $rs = -.11$ and −.09, respectively, and externalizing composite score $r = -.10$) and associated traits of impulsive reward seeking (i.e., child-report BAS scale, $r = -.17$) and callous-unemotionality (i.e., parent-rated ICU, $r = -.12$).[6]

Twin biometric modeling

To disaggregate genetic and environmental influences on the TF-20, we specified full ACE univariate and multivariate twin models. Individual parameters in the univariate model were constrained to zero to test their significance, and AIC values were then compared (Table 4). The best-fitting model was model II (AE model), as indicated by the lowest AIC and a nonsignificant decrement in fit relative to model I ($p = 1.0$). Additive genetic influences accounted for approximately 43% (95% CI = 33–53%) of variance in TF-20 scores, with the remaining 57% of variance (95% CI = 46–66%) attributable to unique environmental factors.

We investigated genetic and environmental overlap between the TF-20 and other internalizing measures that showed at least moderate phenotypic overlap. This was accomplished with two models: a multivariate correlated-factors model that examined the

[4]Test-retest reliability computed using twin 1 data only yielded a point estimate ($r = .84$) consistent with that for the full sample.

[5]Internal consistency reliability computed using twin 1 data only also yielded estimates consistent with those for the full sample: αs for visits 1 and 2 = .87 and .90, respectively.

[6]Validity was also computed using data for twin 1 only, and although point estimates were generally consistent (all correlation differences < .15), the smaller sample size resulted in several associations becoming non-significant (i.e., with parent report SCARED separation anxiety, SCARED panic, SCARED GAD, CBCL withdrawn/depressed, and CBCL externalizing, as well as past phobia diagnosis and current/past GAD diagnoses).

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Table 1. TF-20 item wording and factor loadings for exploratory structural equation model

Item #	Wording	Factor loading *(SE)*
1	I tend to be unsure of myself in tough situations	.319 (.046)
2	I like doing physically dangerous things (R)	.583 (.043)
3	I'm always willing to rush in where others fear to tread (R)	.545 (.039)
4	I am afraid of a lot of things	.494 (.039)
5	I find it frightening to be in a strange new place on my own	.398 (.042)
6	I have a great deal of courage (R)	.443 (.048)
7	I stay calm, cool, and collected in scary situations (R)	.557 (.039)
8	I do not like walking into new situations, even when there is nothing to fear	.391 (.039)
9	I am very easily frightened	.530 (.039)
10	I gladly do things I've never done before, even if they might be dangerous (R)	.672 (.032)
11	I sometimes shy away from crowds of people	.281 (.041)
12	I am fearless (R)	.517 (.040)
13	Major tasks or challenges can seem overwhelming to me	.380 (.044)
14	I'm afraid of far fewer things than most people (R)	.419 (.048)
15	It does not disturb me when I have to do something novel and unfamiliar (R)	.496 (.037)
16	I stay away from physical danger as much as I can	.529 (.047)
17	I am never as afraid as most other people (R)	.512 (.047)
18	It bothers me to be in new situations where things are uncertain	.513 (.042)
19	In challenging situations, I love to be in the "driver's seat" (R)	.500 (.035)
20	I enjoy doing new things that other people are afraid to do (R)	.680 (.027)

Note. (R) indicates item is reverse coded. Item responses range from 0–3. SE = standard error. All loadings are significant at $p < .001$.

role of genetic versus environmental influences in observed relations among the four fear/anxiety measures (TF-20, FSSC-R, SCARED, BIS), and a correlated common pathway model that estimated the overlap in these sources of influence between TF-20 and latent factors corresponding to phobic fear and dysphoric distress symptomatology. A full ACE model was initially fit to the four fear-related measures; however, negative variances were estimated for the common environment (C) factors for TF-20, FSSC-R, and BIS. Based on the best-fitting univariate models of TF-20, FSSC-R, and BIS, we constrained C to be zero in the multivariate correlated factors model (estimated parameters = 32, −2LL = 19,008.25, DF = 2642, AIC = 13,724.249). Proportions of variance in each measure accounted for by genetic and environmental factors are presented in Table 5, and genetic/environmental correlations between the different measures are shown in Table 6. The etiological correlation estimates between scores for the TF-20 and the other internalizing measures (r_gs = .71, .56, and .54 for FCCS-R, SCARED, and BIS, respectively) indicate a moderate degree of genetic overlap between the TF-20 and these fear/anxiety measures.

Lastly, we fit a correlated common pathway twin model specifying latent factors of fear and dysphoric symptomatology based on previous work by our group (Hettema et al., 2020) (estimated parameters = 45, −2LL = 28,186.14, DF = 4467, AIC = 19,252.14). The proportions of variance and genetic/environmental correlations are displayed in Figure 2. Overall, strong overlap was found between the TF-20 and the latent fear factor in terms of both genetic (.68) and environmental correlations (.69). While the TF-20 showed an environmentally based association with the dysphoria factor (.62), there was essentially no genetic overlap ($r = .01$).

Discussion

The current study used data from a large community sample of child-aged twins to evaluate the psychometric properties of an established measure of dispositional threat sensitivity, the 20-item Trait Fear scale (TF-20; Kramer et al., 2020), in youthful participants and examine its phenotypic relations with fear versus dysphoric-distress disorder symptoms and diagnoses and evaluate the role of overlapping genetic and environmental influences in these observed relations.

The TF-20 evidenced excellent psychometric properties in our 9 to 14 year old twins. Consistent with findings for young adults (Kramer et al., 2020), sum scores for the TF-20 were close to normally distributed, and its items as a whole loaded quite uniformly onto a general factor interpretable as dispositional threat sensitivity. The normality of the score distribution accords with the concept of threat sensitivity as a continuous dimensional trait, ranging from low to high – with moderate levels occurring most commonly in the population (Kramer et al., 2012; Patrick et al., 2012). High reliability was evident for overall TF-20 scores, in terms of both test-retest reliability from study visit 1 to visit 2 (*M* retest interval ~ 26 days) and internal consistencies for scores from the two assessment sessions.

As a measure constructed to specifically index variations in dispositional threat sensitivity, the TF-20 displayed expected patterns of convergent and discriminant associations with

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Table 2. Correlations between self-report TF-20 and other self/parent-report measures

	Self-report		Parent-report	
Variable	*r*	*p*	*r*	*p*
FSSC-R	.625	< .0001	–	–
SCARED - Total	.520	< .0001	.270	< .0001
SCARED – Social anxiety scale	.480	< .0001	.264	< .0001
SCARED – Separation anxiety scale	.383	< .0001	.180	< .0001
SCARED – Panic scale	.344	< .0001	.159	< .0001
SCARED – Generalized anxiety scale	.417	< .0001	.179	< .0001
BIS/BAS – Inhibition scale	.564	< .0001	–	–
CASI	.435	< .0001	–	–
ARI	.225	< .0001	-.006	.894
SMFQ	.274	< .0001	.071	.094
JEPQ – Neuroticism scale	.442	< .0001	–	–
JEPQ – Extraversion scale	−.529	< .0001	–	–
BIS/BAS – Activation scale	−.170	< .0001	–	–
BIQ	–	–	.232	< .0001
ICU	–	–	−.117	.006
CBCL – Anxious/Depressed scale	–	–	.218	< .0001
CBCL – Withdrawn/Depressed scale	–	–	.126	.003
CBCL – Externalizing composite (Attention, aggression, rule-breaking)	–	–	−.097	.023
CBCL – Attention problems scale	–	–	−.059	.167
CBCL – Aggressive behavior scale	–	–	−.088	.039
CBCL – Rule-breaking behavior scale	–	–	−.106	.013

Note. Not all scales were administered to both parents and children, as indicated by empty cells. FSSC-R = Revised Fear Survey Schedule for Children; SCARED = Screen for Child Anxiety Related Disorders; BIS/BAS = Behavior Inhibition System/Behavioral Activation System scales; CASI = Child Anxiety Sensitivity Index; ARI = Affective Reactivity Index; SMFQ = Short Mood and Feelings Questionnaire; JEPQ = Junior Eysenck Personality Questionnaire; BIQ = Behavioral Inhibition Questionnaire; ICU = Inventory of Callous-Unemotional Traits; CBCL = Child Behavior Checklist.

Table 3. Polyserial correlations between self-report TF-20 and disorder diagnoses

Disorder	*N*	*r*	*p*
Specific phobia (past/current)	123/107	.180/.217	.002/ < .001
Social phobia (past/current)	34/26	.233/.315	.006/ < .001
Separation anxiety (past/current)	57/33	.164/.245	.031/.005
Panic (past/current)	7/4	.158/NA	.295/NA
Fear disorder composite (ever)	198	.255	< .001
Major depression (past/current)	12/8	.037/.164	.750/.214
Post-traumatic stress (past/current)	11/2	−.008/NA	.957/NA
Generalized anxiety (past/current)	56/55	.240/.257	.001/ < .001
Dysphoric disorder composite (ever)	78	.190	.003
Conduct disorder (past/current)	0/0	NA	NA
Oppositional defiant (past/current)	32/32	−.024/−.099	.787/ .262
Attention deficit hyperactivity (past/current)	112/118	−.062/−.045	.299/.443
Externalizing disorder composite (ever)	140	−.039	.48

Note. *N* = total number exhibiting disorder in past (left of slash) or currently (right of slash). NA indicates correlation was not computed due to small number of cases (< 5).

psychopathology-related criterion variables assessed via parent-report as well as child self-report. The TF-20 showed a particularly strong positive relationship with the FSSC-R, a child-adapted version of an inventory that assesses fear in relation to a range of specific situations and stimuli, the Fear Survey Schedule (FSS). This strong association was expected, given that the adult version of the FSS served as an indicator of the general factor in Kramer et al.'s (2012) fear/fearlessness model, which the TF-20 was designed to index. The TF-20 also showed moderately high correlations with the BIS, a self-report measure of sensitivity to aversive cues, and the SCARED, a youth-report measure of phobic fear and anxiety symptoms. Importantly, and in line with prior research (Kramer et al., 2020; Nelson et al., 2016), the TF-20's observed correlations with these fear-symptom measures ($M = .57$) were more than twice those for child-report measures of dysphoric/distress symptomatology (i.e., SMFQ and ARI, $M = .25$).

The TF-20 also showed a robust association with anxiety sensitivity as measured by the CASI, a construct reflecting the tendency to fear bodily sensations that has been established as a risk factor for panic disorder (Silverman et al., 1991). Notably, the TF's association with the Panic symptom scale of the SCARED was lower, and weaker than its correlation with all other subscales of the SCARED, including Generalized Anxiety. A potential explanation for the TF's comparatively modest association with the SCARED Panic scale, given our sample's very low rate of diagnosable panic disorder (see Table 3), is that general threat sensitivity relates more to latent *risk* for this disorder at younger ages, prior to when panic symptoms emerge. Regarding the TF's stronger than expected association with the SCARED's Generalized Anxiety scale, anxiety of this type in younger aged individuals tends to covary as much or more with fear symptomatology as depressive symptomatology (Lee et al., 2017; Waszczuk et al., 2014), whereas in adults it connects more to dysphoric-distress disorders than phobic fear conditions (e.g., Krueger, 1999; Watson, 2005). The difference may lie in the fact that in younger aged individuals, the cardinal feature of generalized anxiety, worry, entails concerns about specific types of things—in particular, concerns about social standing and performance—rather than broad, non-circumscribed concerns. This more context-related form of worry is in fact reflected in a number of items comprising the Generalized Anxiety subscale of the SCARED (e.g., "I worry about other people liking me."; "I worry about being as good as other kids."; "I worry about things working out for me."; "I worry about how well I do things.")

The correlations of the TF-20 with parent-report measures of phobic fear (SCARED total and subscales, BIQ) and dysphoric/distress symptoms (SMFQ, ARI, CBCL Withdrawn/ Depressed scale) were uniformly lower than those for counterpart child-report measures, as expected given report-modality mismatch. However, paralleling findings for the child-report measures, *r*s with dysphoric symptoms were less than half those with fear symptoms. Further indicative of associational specificity

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Table 4. Twin model fit statistics for TF-20

Model	Domains	Path constraints	Estimated parameters	-2LL	DF	AIC	*p*
I	ACE	–	6	10,869.14	1449	10,857.14	–
II	**AE**	***C* = 0**	**5**	**10,845.14**	**1450**	**10,855.14**	**1.00**
III	CE	*A* = 0	5	10,868.93	1450	10,878.93	<0.01
IV	E	*A* = 0, *C* = 0	4	10,907.70	1451	10,915.70	<0.01

Note. −2LL = negative two log likelihood; DF = degrees of freedom; AIC = Akaike information criterion; ACE = full model with genetic (A), common environmental (C), and nonshared environmental (E) domains included; AE = submodel with only genetic and unique environment included; CE = submodel with only common and unique environment included; *E* = submodel with only unique environment included. Bolding denotes the best-fitting model (i.e., AE).

Table 5. Multivariate correlated factors model: proportions of variance in each scale measure accounted for by genetic and environmental factors

Measure	Genetic factor	Environmental factor
TF-20	.38 (.22–.51)	.62 (.48–.78)
FSSC-R	.40 (.26–.51)	.60 (.48–.74)
SCARED	.50 (.38–.60)	.50 (.40–.61)
BIS	.36 (.20–.50)	.64 (.50–.85)

(discriminant validity), the TF-20's correlations with scale measures of externalizing symptomatology (attentional, aggressive, and rule-breaking), available only in parent-report (CBCL) form, were nonsignificantly negative.

Turning to child-report measures of personality, TF-20 scores showed a positive association with broad negative affectivity as assessed by the JEPQ Neuroticism scale, and opposing negative relations with the Extraversion scale of the JEPQ, encompassing traits of social gregariousness, activity, and excitement seeking, and Carver and White's (1994) BAS, which indexes impulsive reward seeking. The TF-20 also showed a modest negative correlation with the parent-rated ICU scale, which assesses observable propensities toward callousness and emotional insensitivity.

Additionally, the TF-20 evidenced convergent and discriminant validity in relation to psychiatric disorder diagnoses. It related significantly to the three fear disorders that occurred with sufficient prevalence to permit analysis (specific phobia, social phobia, separation anxiety) as well as to the fear disorder composite. Of note, the TF's correlations with fear disorder diagnoses were lower than with self-reported fear symptoms, likely due to modality mismatch (i.e., parent interview vs. child questionnaire) and to expectably lower rates of diagnosable expression of risk for psychopathology at younger versus older ages. The TF-20 also correlated significantly with generalized anxiety which, as noted earlier, aligns more with the fear disorder spectrum than the dysphoric disorder spectrum in younger-aged samples. Predictive specificity of the TF-20 in relation to fear disorder diagnoses was evidenced by a lack of significant associations with major depression, post-traumatic stress disorder, or the dysphoric disorder composite, and negligible relations with externalizing disorder diagnoses (oppositional defiant, attention deficit hyperactivity, externalizing composite).

Univariate twin modeling revealed that additive genetic influences accounted for a substantial portion (43%) of the variance in TF-20 scores in the current youth sample, with the remainder (57%) attributable to environmental factors not shared between twins. These estimates are quite similar to those reported by Kramer et al. (2012) for the general factor of their fear/fearlessness model in a much larger adult twin sample ($N = 2,511$). Of note, measures of various normal-range personality traits also show similar proportions of genetic versus environmental determinants in younger- versus older-aged individuals. For example, the estimated heritability of Eysenck's broad trait of neuroticism is 0.4–0.5 (van den Berg et al., 2014; Vukasović and Bratko, 2015). Also notable, similar to normative personality traits, common (shared family) environment did not contribute significantly to individual differences in dispositional fear/fearlessness in youth. By contrast, common environment has been found to account for 10–20% of variance in many forms of childhood psychopathology (Burt, 2009).

A multivariate correlated-factors twin model was used to clarify the basis of the TF-20's observed (phenotypic) associations with other fear-related constructs assessed via child-report. Genetic correlations of .54 to .71 were evident between the TF-20 and the three other fear-specific measures (FSSC-R, SCARED, BIS), indicating a moderate contribution of additive genetic influences to the observed covariance among these different fear measures. The remainder of their covariance was attributable to overlap in environmental influences among them.

In addition, we used a correlated common pathway twin model to further clarify the role of genetic versus environmental influences in the TF-20's observed relations with latent fear and dysphoria factors of internalizing psychopathology defined using pertinent child-report measures of each. Consistent with hypothesis, we found substantial overlap between genetic influences accounting for variance in TF-20 scores and those accounting for variance specific to the latent fear factor, as evidenced by a strong genetic association between the two. By contrast, there was negligible overlap between the variance in TF-20 scores attributable to genes and genetic influences contributing specifically to the latent dysphoria factor. Of note, the variance in TF-20 scores attributable to environmental influences showed very similar associations with the environment-related variance in latent fear and latent dysphoria. Taken together, these findings suggest that, whereas most of the phenotypic relationship between TF-20 scores and dysphoric disorders reflects overlapping environmental influences, genetic influences unique to the construct assessed by the TF-20 intersect selectively with heritable risk for fear-related disorders.

Implications, limitations, and future directions

Considered in the light of previous research on the threat sensitivity dimension indexed by the TF-20, current study results have important implications for conceptualizing and assessing dispositional risk for fear-related pathologies. Corroborating results from prior work with adults and children (Nelson et al., 2016; Palumbo et al., 2021), current study analyses revealed

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Table 6. Multivariate correlated factors model: genetic and environmental correlations. Between fear-related scale measures

	Genetic correlations			Environmental correlations		
Measure	TF20	FSSC-R	SCARED	TF20	FSSC-R	SCARED
FSSC-R	.71 (.51–.85)	–	–	.56 (.43–.65)	–	–
SCARED	.56 (.36–.72)	.79 (.64–.90)	–	.50 (.37–.60)	.47 (.35–.58)	–
BIS	.54 (.26–.72)	.74 (.54–.89)	.72 (.56–.84)	.52 (.40–63)	.46 (.34–.57)	.60 (.50–69)

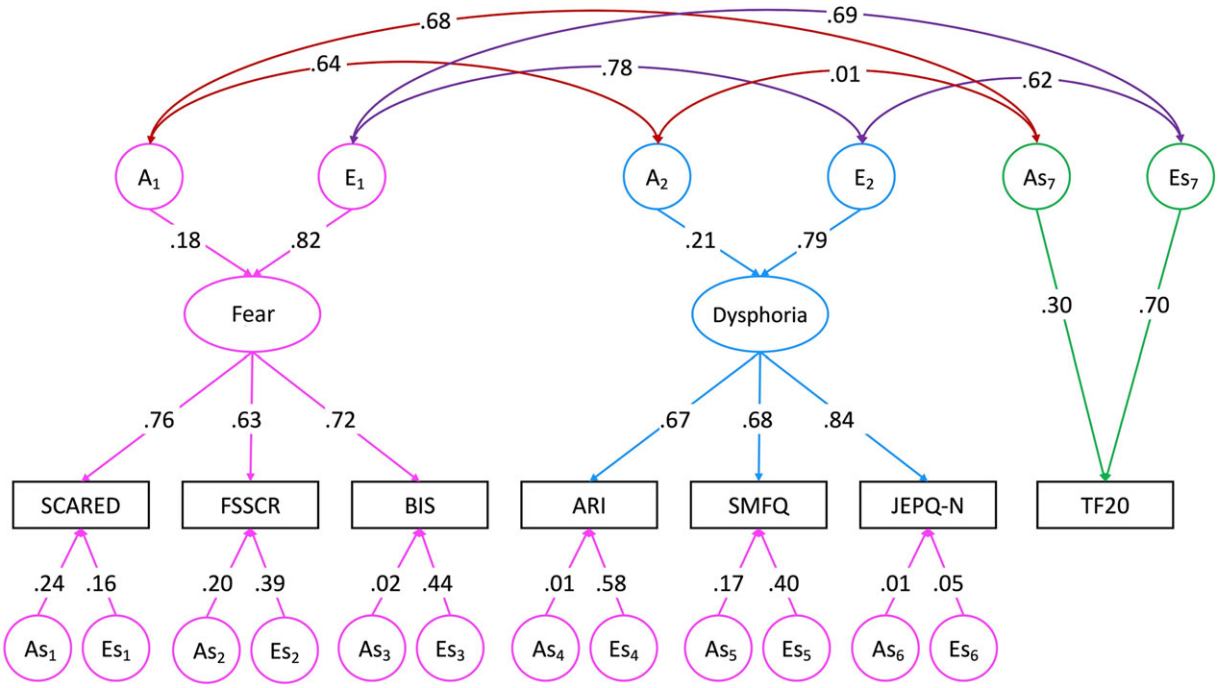


Figure 2. Common pathway model examining the etiological overlap between the TF-20 and latent factors defined by measures of fear and dysphoria. Single-headed arrows denote proportions of variance due to genetic (A) or environmental (E) factors. Double-headed arrows denote correlations between those sources of variance. Subscripts F and D on common A and E factors (top level) denote fear and dysphoria, respectively. Subscripts S_i ($i = 1–6$) on A and E factors (bottom level) denote sources of variance specific to each of the six measured phenotypes: SCARED = Screen for Child Anxiety Related Disorders; FSSC-R = Revised Fear Survey Schedule for Children; BIS = Bbehavior Inhibition System; ARI = Affective Reactivity Index; SMFQ = Short Mood and Feelings Questionnaire; JEPQ-N = Junior Eysenck Personality Questionnaire – Neuroticism scale.

preferential relations for dispositional threat sensitivity with phobic fear disorders relative to dysphoric distress disorders. Extending twin-modeling findings for adults (Kramer et al., 2012), our study also demonstrated moderate-level heritability for scores on this trait dimension in children aged 9-14. Additionally, the current study demonstrated specificity in the contribution of common genetic influences to threat sensitivity's relationship with phobic fear symptoms: whereas TF-20 scores showed a moderately high genetic correlation with a latent fear-symptom factor, its genetic correlation with a counterpart dysphoric-symptom factor was negligible.

These findings indicate that the TF-20 assesses an underlying dispositional risk factor for fear-related pathologies. What is the nature of this underlying dispositional risk factor? In line with prior published work (Patrick et al., 2012), we posit that the TF-20 – and other scale measures designed to harmonize with it (for a review, see Patrick, 2022) – index biobehavioral threat sensitivity in the modality of self-report. As a biobehavioral construct, threat sensitivity can also be indexed using physiological response (e.g., Kramer et al., 2012; Yancey et al., 2016) or task behavioral measures (e.g., Yancey et al., 2019). Indeed, Venables et al. (2017) found that when dispositional threat sensitivity was operationalized using aversive-reactivity measures together with scale-assessed trait fear, it evidenced an even stronger genetic correlation (.80) with phobic fear symptomatology than we found for the TF-20 alone in the current study (.68). The implication is that assessments of genetic risk for fear-related pathologies could be optimized by combining indicators of threat sensitivity from different response modalities. Additionally, measures of threat sensitivity from non-report modalities could be used to assess fear-disorder liability in nonverbal individuals (e.g., very young children).

Some limitations of the current study warrant mention. One is that the data were cross-sectional in nature. Although specificity was evident in threat sensitivity's phenotypic and genotypic associations with phobic fear relative to dysphoric distress symptomatology, longitudinal research demonstrating prospective prediction of phobic fear disorders from earlier-assessed threat sensitivity is needed to firmly establish it as a dispositional liability factor. Further research is also needed to examine threat sensitivity's association with GAD symptoms at younger versus older ages, in order to clarify whether this disorder aligns more with the phobic fear or the dysphoric distress spectrum. Another study limitation is that the youthful age of participants limited the opportunity for dispositional risk to be expressed in the form of

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psychopathology symptoms. In this regard, we posit that TF-20 scores related more to CASI-assessed panic disorder risk in our study than to SCARED-assessed panic symptoms because most participants were below the age of panic disorder expression, and that their youthful age may also have constrained the TF-20's relations with full fear-disorder diagnoses. Longitudinal research can provide a means to test these hypotheses. A further limitation is that the current study included only report-based measures (i.e., child questionnaires, parent ratings, and interview-based assessments). Given the potential value of physiological and behavioral response measures for assessing risk in individuals of various ages, further research using measures of these types is needed. One potential avenue for work of this kind is to utilize data from already existing multi-modal assessment studies in which effective proxy measures of trait fear have been developed (e.g., Bertoldi et al., 2022; Brislin & Patrick, 2019; Palumbo et al., 2021).

Notwithstanding these limitations, the current study importantly extends previous research on dispositional risk for internalizing psychopathology. It adds to growing evidence for a dispositional construct of threat sensitivity, quantifiable through measures in different response modalities, as a specific liability for disorders marked by excessive, irrational fear. Our findings encourage further research utilizing longitudinal collection of multi-modal data from general community, twin, and clinical samples to further understand the nature of this dispositional liability and experiential factors that moderate outcomes associated with it.

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Competing interests. None.

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