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ORIGINAL ARTICLE

Delineating physiologic defensive reactivity in the domain of self-report: phenotypic and etiologic structure of dispositional fear

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Background. Individual differences in fear and fearlessness have been investigated at their extremes in relation to markedly different forms of psychopathology – anxiety disorders and psychopathy, respectively. A documented neural substrate of fear-related traits and disorders is defensive reactivity as reflected in aversive startle potentiation (ASP).

Method. The current study extended prior work by characterizing, in a sample of adult twins from the community ($n=2511$), the phenotypic and etiologic structure of self-report measures of fear and fearlessness known to be associated with ASP.

Results. Analyses revealed a hierarchical structure to the trait fear domain, with an overarching, bipolar fear/fearlessness dimension saturating each measure in this domain, and subfactors labeled 'distress,' 'stimulation seeking' and 'sociability' accounting for additional variance in particular measures. The structure of genetic and non-shared environmental associations among the measures closely mirrored the phenotypic structure of the domain.

Conclusions. The findings have implications for proposals to reconceptualize psychopathology in neurobiological terms.

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Key words: Aversive startle potentiation, fear, personality.

Introduction

Inspired by advances in measurement and understanding of brain circuits relevant to psychological processes, experts in the mental health field have called for efforts to systematically incorporate neurobiological findings into descriptive systems for mental disorders (Hyman, 2007; Insel *et al*. 2010; Sanislow *et al*. 2010). To accomplish this, it will be valuable to establish measures of individual difference constructs relevant to psychopathology that have direct ties to neurobiology (Patrick & Bernat, 2010). With this in mind, the current study examined the structure of various self-report measures of fear and fearlessness that have demonstrated relations in prior research with aversive startle potentiation (ASP), a physiological index of defensive reactivity. Our major aim was to elucidate the common construct indexed by these varying self-report scales. If this construct were to exhibit genotypic as well as phenotypic coherence, it could serve as a valuable referent for research on neural mechanisms underlying varying types of fear-related psychopathology.

Defensive reactivity and startle reflex potentiation

The emotional state of fear has been conceptualized as reflecting activation of the brain's defensive motivational system, which functions to prime evasive action in the presence of threat cues (Davis, 1992; Fanselow, 1994; Lang, 1995; LeDoux, 1995). Neuroscientific research has focused, in particular, on the amygdala as a key component of the defensive (fear) system in mammals (Davis, 1992; Fanselow, 1994; LeDoux, 1995). A well-established experimental measure of fear activation during aversive cuing is enhancement (potentiation) of the startle reflex response to an abrupt noise probe. Davis and colleagues (Davis, 1989; Davis *et al*. 1993) demonstrated that the mechanism for this effect in animals is a pathway from the central nucleus of the amygdala to the nucleus reticularis pontis caudalis, the brainstem junction of the primary

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startle circuit. Human startle studies have, in turn, demonstrated that the startle blink response to sudden noise is reliably potentiated during viewing of aversive pictures in comparison with neutral pictures (Lang *et al*. 1990; Lang, 1995). This potentiation effect tends to be stronger for directly threatening scenes (aimed weapons, attackers) than for vicarious aversive scenes (Levenston *et al*. 2000; Bradley *et al*. 2001; Bernat *et al*. 2006), and is blocked by diazepam (Patrick *et al*. 1996), a drug that inhibits activity in the amygdala and that blocks fear-potentiated startle in animals (Davis, 1979).

Startle potentiation and individual difference variables

Following from animal and human work establishing startle potentiation as a valid index of fear activation, considerable human research has been conducted to evaluate affect-modulated startle as an indicator of emotional pathology and fear-related individual differences. With regard to psychological disorders, augmented startle potentiation during viewing of fear-relevant scenes has been demonstrated in individuals with phobic disorders (e.g. Vrana *et al*. 1992; Hamm *et al*. 1997)[1†] and reduced fear-potentiated startle has been demonstrated in offenders diagnosed with psychopathy (cf. Patrick & Bernat, 2009), a condition theorized to entail a deficiency in fear.

Individual differences in startle potentiation during aversive cuing have also been reported in relation to scores on varying questionnaire measures of fear, fearlessness and psychopathy. Cook *et al*. (1992) reported that individuals selected as high in fearfulness [according to scores on version 3 of the Fear Survey Schedule (FSS-III); Arrindell *et al*. 1984] showed robust potentiation of startle during viewing of aversive as compared with neutral pictures, whereas individuals low in FSS fearfulness showed no evidence of potentiation. Parallel results were obtained in two studies (Corr *et al*. 1995, 1997) of individuals selected to be high *versus* low on the Harm Avoidance (HA) scale of Cloninger's (1987) Tridimensional Personality Questionnaire (TPQ). In contrast, individuals high on the Thrill and Adventure Seeking subscale of Zuckerman's (1979) Sensation Seeking Scale (SSS), a measure reflecting fearlessness, displayed no evidence of startle potentiation for aversive *versus* pleasant pictures, whereas individuals low in thrill-adventure seeking showed robust potentiation (Lissek & Powers, 2003). Similarly, individuals high on the fearless dominance factor of the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), a variable reflecting fearlessness, emotional resiliency and social surgency (Benning *et al*. 2003, 2005*a*), also exhibit a deficit in startle potentiation during aversive picture viewing (Benning *et al*. 2005*b*).

From reported associations for these varying scale indicators, Vaidyanathan *et al*. (2009*a*) hypothesized that ASP operates as a physiological indicator of an underlying trait continuum, ranging from extreme fearlessness to extreme fearfulness. These investigators tested this hypothesis in a college sample and administered the FSS, the subscales of the TPQ–HA scale and the PPI fearless dominance factor, the SSS Thrill-Adventure Seeking Scale and the fearfulness subscale of the Emotionality-Activity-Sociability (EAS) Temperament Survey (Buss & Plomin, 1984). A composite index of trait fear was computed, consisting of scores aggregated across these differing self-report inventories. Startle was assessed during exposure to pictures and a robust positive relation was found between scores on the trait fear composite and degree of startle potentiation during aversive (in particular, direct-threat) scenes relative to neutral scenes. Based on this result, it was postulated that these differing measures of fear and fearlessness may be indicators of a common underlying dimension that reflects proneness to defensive reactivity.

Structure and etiology of self-report measures of dispositional fear and fearlessness: the current study

The current study was undertaken to clarify and refine measurement of defensive reactivity in the domain of self-report by formally evaluating the structure of fear and fearlessness scales that have been linked to variations in ASP. Measures including the FSS-III, SSS Thrill-Adventure Seeking, TPQ–HA and PPI Fearless Dominance subscales and an index of temperamental fear, the EAS Fearfulness Scale, were administered to a large mixed-gender sample of adult twins. We hypothesized that a general bipolar factor would emerge, reflecting variance in common among these various scale measures of fear and fearlessness, interpretable as proneness to defensive reactivity (i.e. trait fear) in the domain of self-report. In addition, the twin feature of the current design provided for evaluation of congruency between the genetic and environmental structures of these fear/fearlessness measures and their observed phenotypic structure.

We hypothesized, in view of evidence for shared genetic contributions to the structure of fear-related disorders (e.g. Kendler *et al*. 1995), that a coherent bipolar factor, paralleling the phenotypic fear/fearlessness factor, would emerge from a structural analysis of the genotypic variance in the scales. Further, based on documented associations of these varying scale

† The notes appear after the main text.

measures with a common physiological indicator (i.e. ASP), we hypothesized that ASP (operationalized as difference in average magnitude of startle during threat pictures as compared with neutral pictures) would exhibit a moderate, selective relationship with the bipolar fear/fearlessness factor of the domain.

Method

Participants

Participants were twins born in Minnesota between the years 1971 and 1985, identified through birth records provided by the Minnesota State Health Department, who were not already enrolled in the Minnesota Twin Family Study (MTFS; Iacono *et al*. 1999, 2003). The base sample comprised 8016 individual twins. Individuals were deemed ineligible for participation ($n=816$) if at the time of birth they met one or more of the following conditions: triplet or higher-order multiple birth; deceased; adopted; born out of wedlock or birth parent deceased; birth certificate missing; physical or mental disability posing limits to testing. From the 7200 twins classified as eligible for participation prior to contact, 6243 (87%) were located. Of these, 495 were deemed ineligible after being located due to one of the aforementioned conditions or because they declined to participate. Thus, the number of eligible located twins was 5748. All individuals in this target sample were mailed materials consisting of a biographical questionnaire, questionnaires assessing fear and fearlessness (see below), a general inventory of personality [the brief version (Patrick *et al*. 2002) of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982)] and a consent form covering the questionnaire protocol. Recipients were 18–33 years of age at the time of this mailing.

Of the eligible located twins, 2592 (45.09%) returned the biographical questionnaire. A total of 51 who returned this questionnaire were deemed ineligible due to one of the aforementioned conditions and 24 did not return the fear-related questionnaires, leaving $n=2517$. Four individual twins returned the fear questionnaires without the biographical questionnaire and 10 were excluded from the phenotypic analyses owing to missing scale score data – resulting in $n=2511$ for these analyses. The biometric analyses included these 2511 twins, less the four for whom data from the biographical questionnaire used to determine zygosity were unavailable, resulting in $n=2507$. The zygosity composition of the final study sample was 651 monozygotic (MZ) pairs (188 male pairs, 463 female pairs) and 334 dizygotic (DZ) pairs (84 male pairs, 250 female pairs). The sample also included 537 individual twins from incomplete pairs (244 men, 293 women).

We also collected additional data for the purpose of evaluating the representativeness of participants who completed and returned questionnaires from this initial mailing. Specifically, we re-sent the biographical questionnaire alone to eligible twins born between 1971 and 1980 who did not return the initial mailing, advising them it would still be valuable for us to receive their data for this questionnaire. Younger twins from later birth years were not included in this mailing, given their greater mobility and enhanced difficulty in re-locating. In response to this second mailing, an additional 909 twin participants returned the biographical questionnaire, raising the overall percentage of respondents for this age cohort (born 1971–1980) to 76%. As one approach to assessing representativeness, we compared biographical data for the portion of the 1971–80 age cohort who returned the original mailing ($n=1677$) with data for those who returned the biographical questionnaire from the second mailing ($n=909$). Group differences were evaluated using Cohen's d for continuous variables (e.g. age, years of education) and χ^2 effect-size (Cramer's φ) for discrete variables (e.g. gender, race). The only group differences that exceeded the level of a small effect size were those for gender (i.e. the female:male ratio was somewhat higher in the cohort that returned the original mailing; $\varphi=0.24$), age (i.e. older by 1.13 years at the time of the second mailing; $d=0.37$) and years of education (i.e. higher among those returning the original mailing; $d=0.33$). Effect sizes for variables reflecting race, family of origin (number of siblings, rearing by both biological parents), marital and parental status, medical experiences of varying types (e.g. any medical condition, admitted to a hospital or emergency room, hospitalized for head injury, currently prescribed medication, etc.) and life-style choices (e.g. smoking cigarettes, serving in the military) were all very small to negligible (d's/φ's ranged from 0 to 0.12).

To further evaluate representativeness, we compared scores on the 11 trait scales of the MPQ for current study participants born between 1971 and 1980 who returned the initial mailing (435 men, 1242 women) with those of age-matched participants from the MTFS (964 men; 822 women). The representativeness of the MTFS sample has been established in previous research (Holdcraft *et al*. 1998). Given known gender differences in some personality traits (e.g. Costa *et al*. 2001), cross-sample comparisons were undertaken separately for men and women. An effect size difference >0.20 was evident for only one of the 11 MPQ trait scales (i.e. achievement; d's $=0.26$ and 0.29 for men and women); median d's for the other

Table 1. *Descriptive statistics reflecting mean endorsement and internal consistency of fear and fearlessness item scales*

		Descriptive statistics				Cronbach's α	
Scale	Items	Mean	S.D.	Range	n	α	n
EAS–Fearfulness	4	1.19	0.60	0–3.00	2511	0.66	2483
Fear Survey Schedule-III	52	1.00	0.47	0–2.79	2501	0.93	2381
PPI–Fearlessness	19	1.35	0.58	0–3.00	2511	0.89	2442
PPI–Social Potency	24	1.58	0.48	0.17–2.88	2511	0.90	2377
PPI–Stress Immunity	11	1.60	0.52	0–3.00	2510	0.83	2440
SSS–Thrill and Adventure Seeking	10	1.69	0.71	0–3.00	2462	0.85	2333
TPQ–HA1 Anticipatory Worry & Pessimism	10	1.13	0.49	0–2.90	2509	0.82	2457
TPQ–HA2 Fear of Uncertainty	7	1.55	0.63	0–3.00	2507	0.79	2464
TPQ–HA3 Shyness with Strangers	7	1.33	0.64	0–3.00	2509	0.85	2479
TPQ–HA4 Fatigability and Asthenia	10	0.93	0.51	0–2.90	2511	0.83	2469

EAS, Emotionality-Activity-Sociability; PPI, Psychopathic Personality Inventory; SSS, Sensation Seeking Scale; TPQ, Tridimensional Personality Questionnaire; HA, Harm Avoidance.

10 MPQ scales were 0.08 and 0.09, respectively, for men and women. The lack of salient differences in personality traits between current sample participants and those in the MTFS provides further evidence of the representativeness of the current sample with regard to self-reported individual differences.

Measures

Scales from self-report measures reflecting cued defensive (fear) reactivity, as evidenced by their relations with ASP in prior work, were administered to this sample of twins. Three measures assessing constructs reflecting fearfulness were: (1) the FSS-III (Arrindell *et al*. 1984), which entails rating one's level of experienced fear in relation to various objects and situations (i.e. item domains include agoraphobia, blood injury, harmless animals, sex and aggression and social fears), with total scores indexing general fearfulness; (2) the four items comprising the fearfulness subscale of the EAS Temperament Survey (Buss & Plomin, 1984); (3) the HA scale of the TPQ (Cloninger, 1987). The four lower-order scales comprising the latter have demonstrated relations with ASP as an omnibus measure in prior research, reflecting situational fear in relation to novelty, danger or risk [fear of uncertainty (HA2)] and social situations [shyness with strangers (HA3)] and susceptibility to fear/distress in the face of future events [anticipatory worry and pessimism (HA1)] and minor setbacks [fatigability and asthenia (HA4)]. Regarding indicators of fearlessness, one set consisted of the three subscales of the PPI (Lilienfeld, 1990; Lilienfeld & Andrews, 1996) that demarcate the fearless dominance factor of the PPI (Benning *et al*. 2003; Lilienfeld & Widows, 2005): (1) social potency, reflecting interpersonal assertiveness and absence of social fear; (2) stress immunity, reflecting the capacity to remain calm rather than panic in situations entailing urgency or threat; (3) fearlessness, reflecting tolerance of danger and preference for activities entailing risk. The other fearlessness measure was the Thrill-Adventure Seeking subscale of the SSS, version 5 (Zuckerman, 1994).

Items from all scales other than the FSS were ordered randomly with a 4-point response format (true, somewhat true, somewhat false, false, definitely A, somewhat A, somewhat B, definitely B). The FSS was administered separately with a 5-point response format ('not at all' to 'very much'). Given the variability in number of items per scale (e.g. four EAS–Fearfulness items, 52 FSS items), scores for each scale were expressed as mean item endorsement values to aid in the interpretability of scores across scales. Scale scores were coded as missing if $>25\%$ of items for the scale were missing; scores were otherwise prorated. Missing values were imputed through full-information maximum likelihood missing data analytic methods for structural modeling analyses.

Descriptive statistics and internal consistency reliabilities of scale scores are presented in Table 1. In general, scales showed strong reliabilities as indexed by Cronbach's α (mean $=0.83$, S.D. $=0.07$), with α somewhat lower for EAS–Fearfulness (0.66) due to its small item set ($n=4$).

Structural modeling analyses

Structural modeling of the questionnaire scale measures of fear and fearlessness was undertaken in two parts. First, phenotypic models were run, examining the structure of the observed scores on these various scale measures. Exploratory factor analysis (EFA) was

conducted to elucidate the number of factors underlying the scales and to identify viable candidate models of scale structure. Confirmatory factor analyses (CFAs) were then run to compare the fit of alternative phenotypic structural models. The second set of structural models consisted of biometric analyses partitioning the observed variances and covariances among the 10 fear and fearlessness scales into etiologic components of variance (i.e. additive genetic, shared environmental and non-shared environmental). This was done using the triangular/Cholesky decomposition method (Neale & Cardon, 1992), which provided for identification of relevant sources of etiologic variation and estimation of genetic and environmental correlations among measures. Next, factor analysis was applied to these etiologic correlation matrices in order to evaluate the consistency between the phenotypic and etiologic structures of the domain.

Phenotypic modeling

EFA was used initially to investigate the number and nature of the factors underlying covariation among the scales. Mplus (version 5; Muthén & Muthén, 1998–2007) was used to estimate maximum likelihood model parameters from the fear and fearlessness scales. Factor loadings were subjected to geomin rotation, an oblique rotation method allowing factors to correlate. Based on evidence for a hierarchical structure to the data (i.e. presence of a general factor, together with additional subordinate factors; see below), an exploratory bifactor model was also estimated.

Next, factors derived from the EFA were utilized in CFAs to evaluate the fit of alternative competing models, including higher order and hierarchical (bifactor) models. A higher order model represents the observed covariance among interrelated measures in terms of an overarching factor that breaks into correlated subfactors, delineated by separate subsets of the individual measures. By contrast, a hierarchical (bifactor) model represents the data in terms of a broad common factor that accounts for variance in each individual measure, together with uncorrelated subfactors reflecting covariation among subsets of the individual measures not accounted for by the general factor (for figural depictions of higher order *versus* bifactor models, see Krueger *et al*. 2007).

To take into account the dependence of twin observations in these phenotypic CFA models, maximum likelihood estimates with standard errors and χ^2 test statistic robust to non-normality and non-independence of observations were utilized (Muthén & Muthén, 1998–2007). In addition to the χ^2 statistic and root mean square error of approximation (RMSEA), which index overall fit of the model, the Akaike's Information Criterion (AIC) and Bayesian Information Criterion (BIC) fit statistics were used to index the efficiency of confirmatory model parameters in accounting for observed data while taking into account the complexity of the model. Values of AIC and BIC are lower for models superior in parsimony as well as fit.

Biometric modeling

The classical twin design assumes that the variance of an observed phenotype can be decomposed into three distinguishable sources of influence: additive genetic (A, i.e. the summation of genetic effects across loci); shared environmental (C); non-shared environmental (E)[2]. Biometric modeling was performed on the current data to determine whether all or only some of these etiologic sources contributed to observed (phenotypic) variance in scale measures and to delineate the structure of covariance among individual scales attributable to these relevant etiologic sources. The Cholesky decomposition method as implemented in Mx (Neale *et al*. 2002) was employed to identify relevant sources of etiologic variance in the individual scale measures (i.e. additive genetic, shared environmental, and/or non-shared environmental) and to estimate genetic and environmental correlation matrices (i.e. patterns of relations among the genetic or environmental components of the differing scales). The Cholesky method (Neale & Cardon, 1992) does not impose a particular structure on the etiologic effects and thus is less restrictive than other multivariate biometric models. Specifically, in cases of the present type where members of twin pairs are measured on multiple phenotypes (scale variables), the Cholesky method fits a model to all calculable variances and covariances (i.e. cross-twin, within-trait variances and covariances; cross-twin, cross-trait variances and covariances), providing for decomposition of phenotypic covariances into genetic and environmental covariances and estimation of genetic and environmental correlations among all available measures. This enabled us to evaluate, following the biometric decomposition, the structure of covariance attributable to each etiologic source in a manner paralleling our analysis of phenotypic structure.

Alternative Cholesky models specifying all or only some etiologic sources as contributory were fit to the raw data using full information maximum likelihood estimation, which adjusts parameter estimates to account for the reduced precision due to incomplete data. The fit of these alternative models was compared using AIC and BIC, with smaller values indicating better fit. Then, to evaluate coherence between the phenotypic and etiologic structures of the domain,

Table 2. *Fear and fearlessness scale scores correlation matrix*

Scale	1	2	3	4	5	6	7	8	9	10
EAS–Fearfulness	–									
Fear Survey Schedule-III	0.55	–								
PPI–Fearlessness	−0.42	−0.37	–							
PPI–Social Potency	−0.42	−0.34	0.38	–						
PPI–Stress Immunity	−0.74	−0.56	0.37	0.44	–					
SSS–Thrill and Adventure Seeking	−0.37	−0.35	0.78	0.30	0.34	–				
TPQ–HA1 Anticipatory Worry & Pessimism	0.66	0.46	−0.33	−0.47	−0.72	−0.30	–			
TPQ–HA2 Fear of Uncertainty	0.61	0.47	−0.73	−0.50	−0.61	−0.64	0.56	–		
TPQ–HA3 Shyness with Strangers	0.45	0.39	−0.27	−0.81	−0.47	−0.24	0.49	0.47	–	
TPQ–HA4 Fatigability and Asthenia	0.52	0.34	−0.22	−0.31	−0.55	−0.24	0.52	0.39	0.35	–

EAS, Emotionality-Activity-Sociability; PPI, Psychopathic Personality Inventory; SSS, Sensation Seeking Scale; TPQ, Tridimensional Personality Questionnaire; HA, Harm Avoidance.

genetic and environmental correlation matrices derived from the best-fitting Cholesky model were utilized in conventional EFA and exploratory bifactor models that paralleled those of phenotypic analyses. As in the phenotypic exploratory analyses, model parameters were estimated using maximum likelihood and loadings were subjected to geomin rotation. The degree of similarity between factors extracted from each correlation matrix across separate EFAs of the phenotypic and etiologic matrices was quantified by correlating phenotypic, genetic and environmental loading vectors. The magnitude of these associations indexed congruency of the phenotypic and etiologic components underlying the model's structure.

ASP as an indicator of trait fear

In addition to demonstrating parallels in the phenotypic and etiologic structures of fear and fearlessness, we sought to validate our structural model by directly evaluating ASP to specific threat cues as an indicator of distinguishable subdomains (factors) of the fear/fearlessness domain. Within an independent participant sample ($n=88$), Vaidyanathan *et al.* (2009*a*) reported an association of 0.31 between ASP to threat cues and a composite of scores on the fear and fearlessness scales utilized in the current study. The availability of a common set of scale measures across the two studies enabled us to quantify covariance between these scale measures and ASP, as a basis for incorporating self-report and physiological (startle) data for this independent sample into the structural model of the current study (combined $n=2599$).

Results

This section is organized into five subsections. The first describes EFAs of the observed (phenotypic) covariance among scales that were performed to identify viable candidate models of scale structure. The second section describes results of CFAs conducted to evaluate the fit of alternative candidate models emerging from the exploratory analyses. The third section describes biometric modeling analyses that were performed to identify etiologic sources contributing to the phenotypic covariance among scales and to evaluate the structure of covariation corresponding to each etiologic source. The fourth section describes analyses that were performed to evaluate similarity between the structure for each etiologic source and the observed phenotypic structure of scales. The fifth and final section reports findings from analyses evaluating the fit of a physiological index of fear, ASP, within the structural model of the fear/fearlessness domain.

Phenotypic structure of the fear/fearlessness scales: EFA

An EFA incorporating all 10 fear/fearlessness measures was first performed to identify the number of factors underlying covariation in the domain and to identify viable candidate models of scale structure. The scree plot from the initial EFA revealed three factors with eigenvalues >1 (5.22, 1.39 and 1.05). The three-factor EFA model provided a good absolute fit to the data (e.g. RMSEA = 0.047): with PPI–Stress Immunity, EAS–Fearfulness, FSS, TPQ–HA1 Anticipatory Worry and Pessimism and TPQ–HA4 Fatigability and Asthenia scales loading on one factor; PPI–Fearlessness, SSS Thrill-Adventure Seeking and TPQ–HA2 Fear of Uncertainty scales loading most strongly on a second factor; PPI–Social Potency and TPQ–HA3 Shyness with strangers scales loading on a third factor. The exploratory analyses were also strongly indicative of the presence of a general factor underlying the data. The manifold of correlations among the scales ranged from strongly positive to strongly negative (Table 2), with the ratio of the first to

Table 3. *Schmid–Leiman solutions for phenotypic scales, and for additive-genetic and non-shared environmental correlations derived from the AE Cholesky biometric analysis*

	Phenotypic				Additive Genetic				Non-shared environmental			
Scale	Gen	1	2	3	Gen	1	2	3	Gen	1	2	3
EAS–Fearfulness	**0.74**	**0.40**	−0.07	0.03	**0.77**	**0.54**	−0.09	0.03	**0.58**	**0.44**	−0.04	−0.03
TPQ–HA1 Anticipatory Worry & Pessimism	**0.71**	**0.37**	0.02	−0.06	**0.72**	**0.54**	0.00	0.00	**0.59**	**0.39**	0.03	−0.12
TPQ–HA4 Fatigability and Asthenia	**0.54**	**0.31**	0.04	−0.01	**0.57**	**0.42**	0.02	−0.03	**0.41**	**0.34**	0.01	−0.01
Fear Survey Schedule-III	**0.56**	**0.26**	−0.11	−0.04	**0.63**	**0.39**	−0.16	0.02	**0.38**	0.24	−0.03	−0.07
PPI–Stress Immunity	**−0.77**	**−0.45**	−0.01	−0.03	**−0.76**	**−0.58**	−0.04	0.03	**−0.63**	**−0.58**	0.01	−0.07
PPI–Fearlessness	**−0.49**	0.02	**0.83**	−0.01	**−0.56**	0.04	**0.79**	0.00	**−0.44**	0.04	**0.81**	0.02
SSS–Thrill and Adventure Seeking	**−0.44**	−0.02	**0.67**	−0.03	**−0.54**	−0.04	**0.68**	−0.05	**−0.31**	−0.04	**0.63**	−0.10
TPQ–HA2 Fear of Uncertainty	**0.69**	0.18	**−0.48**	−0.07	**0.76**	0.23	**−0.53**	−0.06	**0.58**	0.22	**−0.38**	−0.09
PPI–Social Potency	**−0.59**	0.01	0.11	**0.63**	**−0.64**	0.01	0.02	**1.01**	**−0.64**	0.00	0.10	**0.55**
TPQ–HA3 Shyness with Strangers	**0.63**	0.02	0.04	**−0.71**	**0.59**	0.20	0.01	**−0.52**	**0.65**	0.04	0.02	**−0.57**

EAS, Emotionality-Activity-Sociability; TPQ, Tridimensional Personality Questionnaire; HA, Harm Avoidance; PPI, Psychopathic Personality Inventory; SSS, Sensation Seeking Scale.

Loadings of $\geqslant|0.25|$ are shown in bold.

second eigenvalue (3.76:1.00) indicating a substantial amount of common variance among the scales consistent with the presence of a dominant, overarching factor.

Extraction of a single factor using EFA resulted in scale loadings from 0.54 to 0.83. The presence of subsidiary factors to be accounted for in the model was indicated by the varying degrees to which the scales were correlated at the zero-order level, by the evidence for additional systematic variance in the data indicated by the emergence of salient second and third factors and by observation of systematic correlations among the residuals of scales after extraction of the general factor. For these reasons, an exploratory bifactor model, a variant of the Schmid & Leiman (1957) solution[3] specifying a single second-order factor (Yung *et al*. 1999), was conducted to explore the relations between three first order factors and a single, higher order general factor.

The exploratory bifactor solution was computed using syntax provided in Wolff & Preising (2005). First, the factor correlation matrix from the three-factor EFA model was factor-analyzed using maximum likelihood to estimate the loadings of first-order factors on a single, second-order factor. Together with scale loadings on the first-order factors of this model, the contributions of the general and the orthogonal first-order factors to variance in each of the scales were calculated. The loadings for the exploratory bifactor model are presented in Table 3 (columns 1–4). The general, second-order factor accounted for 57% of the variance among the scales, with the first, second and third first-order factors accounting for 10%, 20% and 13% of the unique variance, respectively.

To summarize, conventional EFA approaches yielded evidence both for a three-factor structure to the fear/fearlessness domain and the presence of a broad general factor accounting for variance in each individual measure of this domain. Subsequent exploratory bifactor modeling provided support for a hierarchical structure to the domain. In order to formally compare alternative higher order and bifactor models of the data, the fit of these models was next evaluated in a confirmatory framework.

Phenotypic structure of the fear/fearlessness scales: CFA

A series of CFAs was run to evaluate the fit of differing models of the interrelations among scores on the various fear/fearlessness measures. The first of these, a one-factor model in which all measures served as indicators of a latent, bipolar fear/fearlessness dimension, was used as a base model against which to evaluate alternative, more complex models. In addition, we evaluated three other models based on findings from the initial EFA. The first of these was a two-factor model in which scales indexing fearlessness and excitement seeking (PPI–Fearlessness, SSS Thrill-Adventure Seeking, TPQ–HA2 Fear of Uncertainty) loaded on one factor and scales measuring fearfulness and social assertiveness (PPI–Stress Immunity,

Table 4. *Fit statistics for confirmatory factor analyses of fear and fearlessness scales* ($n = 2511$)

Model	χ^2	df	RMSEA	AIC	BIC
One-factor	4576	35	0.227	31 068	31 243
Two-factor	2742	34	0.178	29 042	29 223
Higher order	1099	32	0.115	27 181	27 373
Bifactor	335	26	0.069	26 331	26 558

χ^2, Adjusted χ^2 fit statistic with robust standard errors; AIC, Akaike's Information Criterion; BIC, Bayesian Information Criterion; RMSEA, root mean square error of approximation.

PPI–Social Potency, EAS–Fearfulness, TPQ–HA1 Anticipatory Worry and Pessimism, TPQ–HA3 Shyness with Strangers, TPQ–HA4 Fatigability and Asthenia, FSS) loaded on the other. This model served as a test of the hypothesis that fear and fearlessness are correlated unipolar dimensions.

The other two models, a higher order model and a hierarchical (bifactor) model, were suggested by the aforementioned evidence of a general factor underlying the data. The fit of a confirmatory bifactor model, analogous to the exploratory bifactor (Schmid–Leiman) model, was compared with that of a higher order model with three first-order factors. Both of these models specified a broad common factor, together with distinct subsidiary factors. The higher order model specified three correlated subfactors emanating from an overarching common factor. In this model, scales tapping experiential fearfulness and distress (PPI–Stress Immunity, EAS–Fearfulness, TPQ–HA1 Anticipatory Worry and Pessimism, TPQ–HA4 Fatigability and Asthenia, FSS) served as indicators of one first-order factor, scales reflecting tolerance of danger and excitement seeking (PPI–Fearlessness, TPQ–HA2 Fear of Uncertainty, SSS Thrill-Adventure Seeking) served as indicators of a second first-order factor, and scales indexing social assertiveness (PPI–Social Potency, TPQ–HA3 Shyness with Strangers) served as indicators of a third subfactor. In these models, the fearlessness dimension bifurcated into excitement seeking and social assertiveness factors.

The bifactor model specified a broad, bipolar fear/fearlessness factor on which all scales loaded, with some scales also loading on one of three distinct subfactors reflecting domains that varied independently of the general factor and of one another. The subfactors specified in this model were deduced from the three-factor EFA utilized in the Schmid–Leiman solution. The first subfactor was marked by EAS–Fearfulness, FSS, PPI–Stress Immunity, TPQ–HA1 Anticipatory Worry and TPQ–HA4 Fatigability; the second was indicated by PPI–Fearlessness, TPQ–HA2 Fear of Uncertainty and SSS Thrill-Adventure Seeking; the third was specified by PPI–Social Potency and TPQ–HA3 Shyness scales.

Table 4 shows the fit statistics for these competing models. The bifactor model with three uncorrelated subfactors fit best. This model had lower χ^2, RMSEA, AIC and BIC values than the competing one- or two-factor models or the three-factor higher order model. The finding of best-fit for the bifactor model indicates that a general factor saturates each individual scale, that each scale also contains variance tied to one of the three residual factors and that the general and specific factors are mutually uncorrelated. Loadings of individual scales on the general factor of this model ranged from strongly positive to strongly negative, with all scales exhibiting absolute loadings >0.45 (see Fig. 1). The bipolar fear/fearlessness dimension saturating each of the measures was marked at the high end by scales assessing susceptibility to fear (e.g. EAS–Fearfulness, FSS, TPQ–HA2 Fear of Uncertainty, TPQ–HA1 Anticipatory Worry) and at the low end by thrill seeking, dominance and resiliency (e.g. SSS Thrill-Adventure Seeking, PPI–Social Potency, PPI–Stress Immunity). The three subfactors, reflecting variance in common among individual scale indicators after accounting for relations with the general factor, were marked by distinctive subsets of scales.

To help characterize the nature of the general factor and the subfactors, illustrative items were identified by computing maximum likelihood estimates of scores for the factors and examining correlations of individual items with each. Table 5 lists, for each factor, items exhibiting moderate to high correlations with that factor and lesser (in many instances negligible) correlations with other factors. Based on the content of representative items as depicted, the first subfactor was interpreted as reflecting generalized distress (as distinct from cue-specific fear) and the second and third subfactors as reflecting stimulation seeking and sociability, respectively.

To summarize, comparisons of alternative higher order and hierarchical (bifactor) models of scale structure suggested by EFA findings revealed the best fit for the bifactor model that specified a broad, bipolar fear/fearlessness factor accounting for variance in each scale measure, together with subfactors labeled 'distress', 'stimulation seeking' and 'sociability' accounting for residual variance in subsets of individual measures. Next, we evaluated the structure of components of variance in individual scale measures attributable to differing etiologic sources using biometric (Cholesky) decomposition followed by EFA modeling[4].

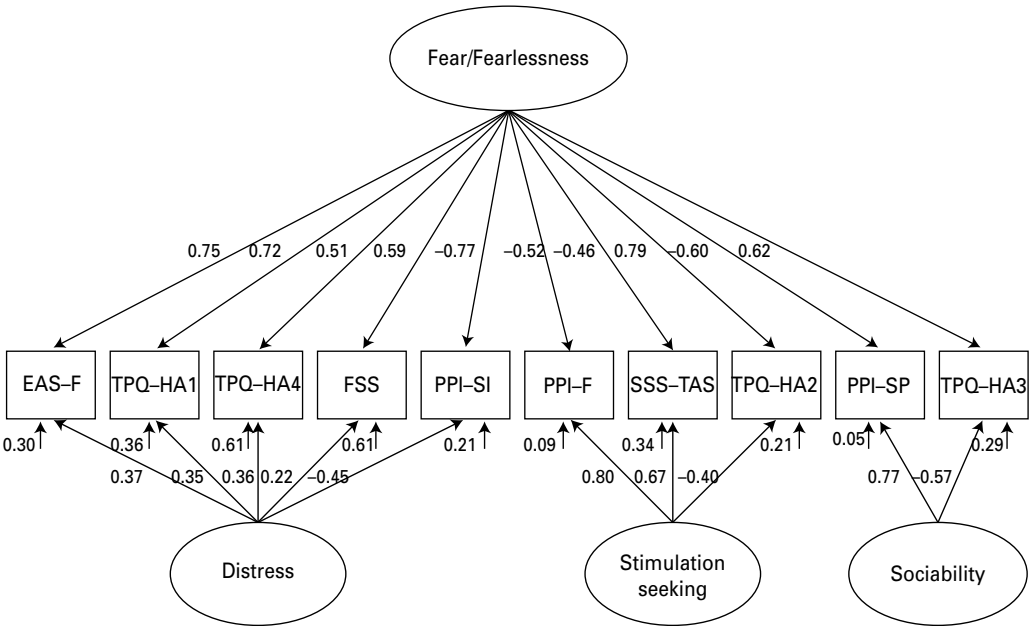

Fig. 1. Standardized parameter estimates for best-fitting confirmatory bifactor model of fear and fearlessness scales. EAS–F, Emotionality-Activity-Sociability–Fearfulness; TPQ, Tridimensional Personality Questionnaire; HA, Harm Avoidance; TPQ–HA1, Anticipatory worry & pessimism; TPQ–HA2, Fear of Uncertainty; TPQ–HA3, Shyness with strangers; TPQ–HA4, Fatigability and asthenia; FSS, Fear Survey Schedule; PPI, Psychopathic Personality Inventory; PPI–SI, Stress Immunity; PPI–F, Fearlessness; PPI–SP, Social Potency; SSS, Sensation Seeking Scale; SSS–TAS, Thrill-adventure seeking.

Genetic and environmental structure of the fear/fearlessness scales

Biometric structural modeling analyses were conducted in two steps. First, Cholesky models specifying alternative etiologic sources of variance in the domain were compared. Next, genetic and environmental correlation matrices (Table 6) estimated in the best-fitting Cholesky model were utilized in EFAs to delineate the structure of etiologic contributions to variation in the domain. Models were fit allowing for all three sources of variation, additive genetic (A), shared environmental (C) and non-shared environmental effects (ACE; $-2LL = 23\,787$, df = 24 758, AIC = −25 729, BIC = −78 817), two sources of variation (CE; $-2LL = 23\,989$, df = 24 813, AIC = −25 637, BIC = −78 917; AE; $-2LL = 23\,807$, df = 24 813, AIC = −25 819, BIC = −79 009) and non-shared environmental variance and measurement error (E; $-2LL = 24\,898$, df = 24 868, AIC = −24 838, BIC = −78 665). The model allowing for genetic (A) and non-shared environmental (E) sources of variance and covariance among the 10 fear/fearlessness scales yielded the best overall fit. In particular, the AE model displayed the lowest AIC and BIC values relative to the others. The log-likelihood (–2LL) value was slightly lower for the full ACE model, but unlike AIC and BIC this fit statistic does not incorporate the criterion of parsimony, which was superior for the AE model.

Following up on the Cholesky modeling results, EFA with maximum likelihood estimation was used to evaluate the structures of additive genetic and non-shared environmental correlations among fear measures. EFA revealed three factors with eigenvalues >1 for both the additive genetic (6.08, 1.39, 1.09) and non-shared environmental (4.27, 1.42, 1.02) correlation matrices, which were extracted and geomin rotated. Findings here suggested that a bifactor structure might best account for correlations among the scales attributable to each etiologic source (i.e. additive genetic and non-shared environmental factors). Therefore, Schmid–Leiman transformations were undertaken that paralleled the phenotypic analyses. Factor loadings for each scale in the genetic and environmental solutions are presented in Table 3 (columns 5–8 and 9–12, respectively), alongside loadings from the phenotypic solution (columns 1–4). The general fear factor and the distress, stimulation seeking and sociability subfactors accounted for 52%, 16%, 17% and 15% of the variance in the genetic correlations among scale indicators and 51%, 16%, 22% and 12% of the shared variance in the non-shared environmental correlations.

In sum, biometric analyses revealed that observed variance in scale measures of fear/fearlessness was attributable primarily to additive genetic and non-shared environmental influences, with negligible contribution of shared environment. EFA structural

Table 5. *Sample items delineating the general factor and subfactors of the best-fitting bifactor model*

General factor	Gen	F1	F2	F3
Experiences tension in unfamiliar situations when others perceive no danger[8]	0.71	0.17	0.03	−0.08
Feels afraid in novel situations[8]	0.64	0.12	0.02	−0.10
Recovers slowly from embarrassing situations[4]	0.56	0.28	0.02	−0.25
Is readily frightened[1]	0.54	0.37	−0.09	0.13
Experiences fear in unfamiliar places[2]	0.50	0.18	−0.08	−0.05
Fears unfamiliar people[2]	0.47	0.14	0.02	−0.19
Is not intimidated by other people[4]	−0.46	−0.14	0.09	0.16
Can suppress fear in scary situations[5]	−0.50	−0.25	0.21	0.00
Almost always remains calm when most others are frightened[7]	−0.53	−0.38	0.10	−0.06
Has fewer fears than most people[1]	−0.55	−0.33	0.14	−0.04
Recovers quickly from embarrassing situations[7]	−0.55	−0.25	0.03	0.24
Remains relaxed in situations where most others would panic[5]	−0.56	−0.34	0.12	0.01
Subfactor 1: Distress				
At times is distressed by ruminating about the day's events[5]	0.51	0.52	0.14	0.04
Requires more rest and reassurance than usual to recover from minor setbacks[10]	0.35	0.48	0.09	0.12
Has a short fuse when under stress[5]	0.36	0.45	0.16	0.09
Is irritable when faced with too many tasks[5]	0.42	0.43	0.07	0.05
Is more easily fatigued than most people[10]	0.32	0.40	0.05	0.06
Recovers slowly from minor stress or illness[10]	0.22	0.37	0.06	0.10
Subfactor 2: Stimulation seeking				
At times does dangerous things after being dared to do them[3]	−0.24	0.13	0.57	0.06
Thinks being part of a roving motorcycle gang and causing some chaos may be fun[3]	−0.21	0.12	0.56	0.07
Shares the attitude that taking risks eschews boredom[3]	−0.25	0.15	0.51	0.08
Takes chances for excitement when life becomes boring[3]	−0.26	0.11	0.44	0.20
(A) prefers remaining on the water's surface (B) would enjoy exploring beneath it[6]	0.28	0.02	−0.45	0.02
(A) would not enjoy pilot training (B) would enjoy pilot training[6]	0.33	0.05	−0.46	0.04
Subfactor 3: Sociability				
Enjoys being particularly noticeable relative to others in a group of people[4]	−0.33	0.20	0.18	0.64
Easily facilitates conversation[4]	0.38	0.07	−0.08	0.63
Can interest others in getting to know them better through one smile[4]	−0.21	0.08	0.03	0.50
Is more shy than the average person when first meeting a group of people[9]	0.49	−0.05	0.06	−0.56
Is rarely the centre of attention socially[4]	0.27	−0.16	−0.06	−0.60
Rarely keeps a party fun and exciting for others[4]	0.37	−0.12	−0.16	−0.62

Superscripts indicate the items' source measures: [1]Emotionality-Activity-Sociability–Fearfulness; [2]Fear Survey Schedule; [3]Psychopathic Personality Inventory (PPI)–Fearlessness; [4]PPI–Social Potency; [5]PPI–Stress Immunity; [6]Sensation Seeking Scale–Thrill-adventure seeking; [7]Tridimensional Personality Questionnaire (TPQ)–Harm Avoidance (HA)1 Anticipatory Worry and Pessimism; [8]TPQ–HA2 Fear of uncertainty; [9]TPQ–HA3 Shyness with strangers; [10]TPQ–HA4 Fatigability and asthenia.

analyses of variance attributable to each of these etiologic sources revealed a bifactor structure similar to that emerging from structural analysis of the overall phenotypic variance in scales. As a follow-up to this, we undertook analyses to directly evaluate the degree of correspondence between the structures for these etiologic sources and the observed phenotypic structure.

Congruence between phenotypic and etiologic structures of the fear/fearlessness scales

The above-described exploratory bifactor analyses of the phenotypic, additive genetic and non-shared environmental correlations among fear and fearlessness scales revealed structural similarities in patterns of relationships across the differing factor solutions.

Table 6. *Fear and fearlessness scale additive genetic (lower diagonal) and non-shared environmental (upper diagonal) correlation matrices*

Scale	1	2	3	4	5	6	7	8	9	10
PPI-Stress immunity	–	−0.61	−0.59	−0.38	−0.45	−0.49	0.26	0.23	−0.39	0.37
EAS-Fearfulness	−0.89	–	0.53	0.34	0.40	0.44	−0.27	−0.22	0.40	−0.39
TPQ-HA1 Anticipatory Worry & Pessimism	−0.86	0.83	–	0.28	0.34	0.44	−0.23	−0.14	0.45	−0.46
Fear Survey Schedule-III	−0.71	0.75	0.61	–	0.26	0.23	−0.18	−0.20	0.32	−0.26
TPQ-HA4 Fatigability and Asthenia	−0.65	0.67	0.75	0.39	–	0.28	−0.15	−0.16	0.29	−0.26
TPQ-HA2 Fear of Uncertainty	−0.69	0.76	0.68	0.65	0.50	–	−0.56	−0.43	0.45	−0.43
PPI-Fearlessness	0.37	−0.48	−0.38	−0.47	−0.29	−0.83	–	0.64	−0.27	0.38
SSS-Thrill and Adventure Seeking	0.39	−0.49	−0.44	−0.44	−0.33	−0.78	0.83	–	−0.14	0.20
TPQ-HA3 Shyness with Strangers	−0.57	0.54	0.55	0.46	0.43	0.54	−0.30	−0.34	–	−0.72
PPI-Social Potency	0.51	−0.45	−0.47	−0.40	−0.38	−0.56	0.36	0.34	−0.90	–

PPI, Psychopathic Personality Inventory; EAS, Emotionality-Activity-Sociability; TPQ, Tridimensional Personality Questionnaire; HA, Harm Avoidance; SSS, Sensation Seeking Scale.

In order to empirically evaluate these apparent coherences, we computed congruency coefficients among loading vectors for factors emerging from the phenotypic, additive genetic and non-shared environmental solutions. Congruency coefficients were uniformly very high (0.95–1.00), indicating strong correspondence between loading vectors for the differing solutions.

Evaluating ASP as an indicator of trait fear

The availability of an independent sample of participants who completed the fear and fearlessness questionnaires and participated in an affect-modulated startle assessment (Vaidyanathan *et al*. 2009*a*; $n=88$) provided for evaluation of the effectiveness and specificity of ASP as a physiological indicator of the general fear factor. The bivariate correlation between scores on the general factor (estimated from the best-fitting model using maximum likelihood) and ASP difference scores (threat-cuing condition – neutral-cuing condition) for this subsample was $r=0.30$, $p<0.01$. By contrast, correlations between scores on the three subfactors of the model were uniformly small ($\leqslant 0.11$) and non-significant. When incorporated into the best-fitting structural model as an indicator of the general fear/fearlessness factor only, the loading for ASP on the general factor was 0.35. The fit of this model (AIC = 27 595, BIC = 27 841, RMSEA = 0.062) was as good or better than that of models in which ASP was specified as loading on the general factor and concurrently on either the distress (AIC = 27 597, BIC = 27 849, RMSEA = 0.062), stimulation seeking (AIC = 27 597, BIC = 27 849, RMSEA = 0.062) or sociability (AIC = 27 598, BIC = 27 856, RMSEA = 0.063) subfactor. Notably, the loadings of ASP on the subfactors of the latter models were small (0.03, −0.09 and −0.11, respectively) and nonsignificant.[5] Taken together, these findings demonstrate a selective association between cued defensive reactivity as indexed by startle reflex potentiation during viewing of threat scenes and the general factor of the fear/fearlessness model.

Discussion

The current study evaluated the phenotypic, genetic and environmental structure of various self-report measures of fear and fearlessness that have evidenced relations with ASP in prior work. Our findings provide evidence for the coherency of this individual-difference domain, both psychometrically and etiologically, and for the model's utility in indexing neurobiological defensive reactivity in the modality of self-report. Before reviewing the major findings and their conceptual and practical implications, we first consider some specific limitations.

One limitation had to do with the modest participation rate (45.09%) for twins targeted in the original questionnaire mailing. This raises potential concerns about the representativeness of questionnaire data utilized in the analyses and generalizability of findings. We addressed this issue in two ways. We collected follow-up biographical data from additional participants within a targeted age cohort, raising the overall return rate for this cohort to 76%. Comparisons of initial respondents and follow-up participants revealed generally good representativeness on various demographic and biographical variables. In addition, we compared MPQ personality scores for twins in the current study who returned the original questionnaires with MPQ scores for a population-representative sample of similar-aged twins surveyed in a different project. As with biographical variables, differences in personality scores across the two samples were

uniformly small in magnitude. Although some caution is still warranted in interpreting study results, these comparisons help to alleviate concerns about sample representativeness.

A second limitation is that ASP data were not available for the twin sample and thus were estimated in the model through full-information maximum-likelihood imputation based upon its associations with fear and fearlessness measures in an independent sample. Although the loading magnitudes of ASP in the structural model are best interpreted with caution, they were nonetheless comparable to magnitudes of bivariate correlations with factor scores in the independent sample alone. Despite these limitations, the current research yielded findings with potentially important implications for conceptualization of individual differences in fear reactivity and for efforts to link psychopathological conditions to neurobiological systems. These points are addressed in the remaining sections below.

Phenotypic structure of fear/fearlessness domain

A key finding of this study was that the phenotypic structure of fear/fearlessness as indexed by scale measures associated with ASP is multi-dimensional and hierarchical. The bifactor structure of the domain functioned to demarcate the general dimension reflecting common variance among the scale indicators while partitioning proportions of residual variance in each scale into three subfactors that varied independently of the broad, bipolar factor of fear/fearlessness. That is, a common dimensional variable accounted for appreciable variance in all scales, with some scales (those reflecting fearfulness) serving as high-pole indicators of this factor and others (those reflecting fearlessness) serving as low-pole indicators. After accounting for relations with the general fear/fearlessness factor, portions of remaining variance in each scale indicator were associated with one of three distinctive subfactors. Specifically, residual variance in scales indexing perceived experience of negative emotion in relation to threatening or stressful objects or situations comprised the first of these subfactors (labeled 'distress'). Scales indexing preference for activities entailing, danger, risk or novelty served as indicators of both the general fear factor and the second subfactor (labeled 'stimulation seeking'). The third subfactor (labeled 'sociability') reflected portions of remaining variance in scales that indexed a bold/outgoing *versus* timid/avoidant interpersonal style.

Notably, the strongest indicators of the general fear factor (i.e. loadings of $\geqslant 0.60$) included scales from all three of these content domains (perceived experience, activity preference, interpersonal style) and each content domain included one or more scales that loaded positively on the general fear factor and one or more that loaded negatively on the general factor. The implication is that variance in measures from all three domains was represented strongly in the general factor, with each domain contributing to the bipolarity of the general factor. This bipolarity and breadth of content domains indicates that trait fear, when defined as the variable in common among scales that predict physiological defensive reactivity, encompasses both phobic fear (e.g. FSS) and (reverse) HA (e.g. PPI–Fearlessness). This perspective may help to reconcile persisting debates about whether dispositional fear should be operationalized in terms of perceived experience of fear or in terms of reported preference for uncertainty/risk *versus* familiarity (cf. Sylvers *et al*. 2011).

In this regard, the distinct content domains of the current model can be viewed as differing points of reference for reports of fear experience. Items that functioned as strong and relatively pure indicators of the general fear factor (cf. Table 5) included items dealing with readiness to react with fear in various contexts (i.e. susceptibility *versus* immunity to fear states, few as opposed to many fears), fear associated with social situations (i.e. presence *versus* absence of social fear, slow *versus* rapid recovery from embarrassing situations), and fear in relation to unfamiliar or novel situations. Further, the scale measure that loaded most selectively on the general factor in the bifactor structural model (Fig. 1) was the FSS, an index of experiential fear in relation to various specific objects and situations; the FSS exhibited the weakest cross-loading of any of the scales on its affiliated (distress) subfactor. The general factor therefore appears to index fear activation in relation to a broad range of threat cues.

At the same time, however, distinct subfactors emerged in the best-fitting model that reflected variance in common among particular measures after accounting for variance associated with the general factor. Scales contributing to the first subfactor can be viewed as capturing some aspect of proneness to negative affective experience that is distinct from fearfulness. Per Table 5, items that correlated preferentially with this subfactor dealt with rumination-related distress, irritability when overburdened or stressed, fatigability and slowness to recover from setbacks or illness. Our interpretation of this 'distress' factor is that it reflects general anxiousness and emotional instability as opposed to defensive reactivity in relation to specific objects and situations that characterize dispositional fear. Scales identified with the second subfactor can be viewed as capturing, in addition to fearfulness, some other component of

affinity for situations involving novelty or risk. Items indicative of this subfactor dealt with proneness to boredom, pursuit of excitement and rebelliousness. We interpreted this 'stimulation seeking' subfactor as reflecting impulsive, sensation-seeking tendencies (Zuckerman, 2007) associated with a general disinhibitory ('externalizing') style (Sher & Trull, 1994; Krueger *et al*. 2007). Scales that defined the third subfactor can be viewed as tapping some component of a reserved *versus* outgoing interpersonal style that is distinct from dispositional fear. Items associated preferentially with this subfactor reflected ease of engagement with others, enjoyment of groups and interest *versus* disinterest in social visibility.

Etiologic determinants

Because our research participants were twins, we were able to go beyond the evaluation of phenotypic structure to also consider underlying etiologic contributions to this structure. In line with previous findings regarding relative genetic and environmental contributions to personality (cf. Krueger & Johnson, 2008), additive genetic and non-shared environmental influences contributed about equally to the observed phenotypic factors, with no evidence of shared environmental effects. Additionally, examination of the structure of genetic and non-shared environmental correlations among scale measures revealed very close correspondence with the phenotypic structure of the scales (Table 3), with genetic and environmental structures of the scales closely mirroring the phenotypic structure (loading vector *r*'s 0.95–1.00). This finding is notable because it suggests that the phenotypic factors we observed are etiologically coherent. The implication is that the general bipolar fear–fearlessness factor appears to reflect genetic and environmental effects working in concert to delineate this domain, making this dispositional factor an appealing target for investigation of biological contributions to disorders involving excessive or deficient fear (cf. Waldman, 2005).

Implications for research on affective individual differences and psychopathology

The current study took a novel approach by focusing on self-report trait measures that have exhibited relations (either positive or negative) with a well-established physiological index of defensive reactivity – namely, aversive potentiation of the protective startle reflex. Although measures selected using ASP as a referent appeared to reflect differing content domains (affective states, social interaction, behavioral preference), they nonetheless were found to cohere around a common, bipolar factor. Our interpretation of this common factor is that it represents the counterpart, in the domain of self-report, to a biological dimension of weak *versus* strong defensive (fear) reactivity – defined as readiness of the brain's defensive system to become activated in the face of explicit threat cues (Lang *et al*. 1990; Davis *et al*. 1997).

We evaluated the validity of this interpretation by extending the basic model to include data from an independent sample tested in a picture-startle procedure (Vaidyanathan *et al*. 2009*a*). Incorporating ASP into the model revealed a robust loading of 0.35 on the general factor and negligible loadings on the subfactors of the model, indicating that ASP functioned as a selective indicator of trait fear. Defined in this manner, the dimension of dispositional fear provides an example of a neurobehavioral construct (i.e. a construct with direct referents in brain physiology and behavior; Depue & Iacono, 1989; Patrick & Bernat, 2010) that can serve as a referent for research on the neurobiology of individual differences. As a follow-up to the current work, it will be valuable to identify additional physiological indicators of trait fear and to evaluate their structure. Examples of other physiological variables besides ASP that might be expected to correlate with trait fear include speed of acquisition and/or extinction of a conditioned fear response, magnitude of autonomic reactivity during anticipation of a cued aversive event (e.g. shock or loud noise) and magnitude of amygdala reactivity to prepotent aversive cues (e.g. phobic images, fear faces). Research along these lines would provide the basis for a psychometrically coherent, neurobiologically based method for assessing individual differences in defensive (fear) reactivity (cf. Patrick & Bernat, 2010; Nelson *et al*. 2011).

The individual difference domain identified in the current study also has potential relevance to research on the neurobiology of mental disorders. At one end, the general trait fear dimension intersects with the domain of internalizing psychopathology through its coverage of phobic fears (i.e. shyness and social anxiety; fear of specific objects/situations). At the other end, the dimension of trait fear intersects with the syndrome of psychopathy through coverage of tendencies involving social potency, affective imperturbability and thrill-seeking. Consistent with affect–startle studies reporting heightened and reduced ASP in individuals with phobic disorders (cf. Vaidyanathan *et al*. 2009*b*) and psychopathy (cf. Patrick & Bernat, 2010), respectively, the general fear/fearlessness dimension delineated here may link these syndromes in their neurobiological underpinnings. A further implication of the model is that dispositional fear can be distinguished from generalized distress-proneness, in the domain of

self-report and perhaps also in the domain of physiology. For example, animal research has implicated structures extending beyond the amygdala (e.g. bed nucleus of the stria terminalis) in non-specific distress states (cf. Davis *et al*. 1997; Rosen & Schulkin, 1998) and human research points to distinct physiological correlates for distress-proneness (Lader & Wing, 1964; Vaidyanathan *et al*. 2009*b*).

In conclusion, the findings of the current study highlight the construct of dispositional defensive reactivity – operationalized in the domain of self-report as trait fear/fearlessness – as an important referent for neurobiological studies of psychopathology. Systematic research focusing on constructs of this sort is likely to be crucial in efforts to reconceptualize mental disorders in neurobiological terms (Hyman, 2007; Insel *et al*. 2010) and identify gene-based liabilities for such disorders (Sherman & Waldman, 1999; Waldman, 2005).

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Declaration of Interest

None.

Notes

[1] By contrast, phobic individuals with co-morbid depression or patients with disorders entailing pervasive anxiousness (e.g. post-traumatic stress disorder, generalized anxiety disorder) do not show enhanced startle potentiation during aversive cuing (e.g. Grillon & Morgan, 1999; Cuthbert *et al*. 2003; Lang *et al*. 2007; Taylor-Clift *et al*. 2011; for a recent review, see Vaidyanathan *et al*. 2009*b*). The implication is that ASP covaries with individual differences in cue-specific defensive (fear) reactivity but not with the more pervasive, dysregulated negative affect that characterizes pathological distress (cf. Rosen & Schulkin, 1998).

[2] Shared environmental variance is composed of environmental effects that contribute to twin similarity, while non-shared environmental variance is composed of environmental effects that contribute to differences between members of a twin pair and measurement error. The genetic and environmental variance components can be estimated by comparing the similarity of monozygotic (MZ) and dizygotic (DZ) twins because MZ twins share 100% of additive genetic effects whereas DZ twins share, on average, 50%, and shared environmental influences are assumed to affect MZ and DZ twins equally. The models we tested further assumed no gene–environment correlations, gene–environment interactions or assortative mating.

[3] The Schmid–Leiman solution, implemented as a bifactor model in this case, facilitates interpretation of factors relative to higher order factor analysis by computing direct relations between primary variables and second-order factors. Contributions of first- and second-order factors to variance in the observed variables (i.e. loadings) are estimated to maximize the contribution of second-order factors and first-order factor loadings are transformed to residual loadings not explained by the general factor. First-order factor loadings are essentially transformed into partial correlations, with first-order factors orthogonal to the second-order factor, but not necessarily independent of one another (Wolff & Preising, 2005).

[4] As a supplement to the biometric decomposition and etiologic-structural analysis of scales, we also evaluated the relative contributions of genetic and environmental influences to scores on the phenotypic factors from the best-fitting bifactor model, computed using maximum likelihood estimation. Contributions of additive genetic, shared environmental and non-shared environmental influences to scores on each of the factors were estimated by fitting univariate biometric models to cross-twin, within-trait covariances (i.e. the covariance between Twin A and Twin B factor scores) using maximum likelihood estimation as implemented in Mx. Using this approach, contributions of additive genetic, shared environmental and non-shared environmental loadings on the general trait fear factor were estimated to be 0.71, 0.00 and 0.70, respectively – indicating that additive genetic influences accounted for 51% (0.712) of the variance in the general trait dimension, with non-shared environmental effects accounting for the remaining 49%. Additive genetic and non-shared environmental effects accounted for 35% and 65% of variance in the distress subfactor, respectively, and 55% and 45% of variance in the sociability subfactor. Additive genetic and non-shared environmental effects each accounted for 50% of the variance in stimulation seeking. Shared environmental effects accounted for none of the variance in the general factor or any of the three subfactors.

[5] Given that studies examining aversive startle potentiation (ASP) in relation to Tridimensional Personality Questionnaire (TPQ) Harm Avoidance (HA) have focused on scores for the scale as a whole (Corr *et al*. 1995, 1997), we included all four HA subscales in our primary structural model. As a supplemental analysis, we evaluated the performance of ASP as an indicator of general fear/fearlessness in an alternative model in which two TPQ HA scales [anticipatory worry/pessimism (HA1) and fatigability/asthenia (HA4)] were omitted as indicators in order to: (1) balance the representation of content domains (experiential fear, risk aversion, social fear) in the model; (2) limit scale indicators in the model to those most clearly indicative of fear. ASP was specified as loading only on

the general fear/fearlessness factor in this alternative nine-indicator model (eight scale measures + ASP). The absolute fit of this model (root mean square error of approximation = 0.068) was similar to that of the full model (10 scale measures + ASP), with ASP loading to a comparable robust degree on the general factor (0.36) and negligibly on the subfactors of the model.

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