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Research paper

Demarcation of anxiety and fear: Evidence from behavioral genetics

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ABSTRACT

Anxiety and fear are emotions often intertwined in response to aversive stimuli, complicating efforts to differentiate them and understand their distinct consequences. This study explores the common genetic and environmental factors contributing to the co-occurrence of anxiety disorders and dimensions of the revised Reinforcement Sensitivity Theory (rRST). A sample of 356 monozygotic (22.5 % males; $M = 25.73$, $SD = 8.3$) and 386 dizygotic (33.9 % males; $M = 24.21$, $SD = 8.33$) twins from the Serbian Twin Advanced Registry was analyzed. The Psychiatric Diagnostic Screening Questionnaire (PDSQ) provided scales for panic disorder, agoraphobia, social phobia, and generalized anxiety disorder (GAD), while the Reinforcement Sensitivity Questionnaire (RSQ) measured the Behavioral Inhibition System (BIS), Behavioral Activation System (BAS), and Fight/Flight/Freeze System (FFFS). Common additive genetic effects accounted for most of the variance in BIS, Fight, and panic, agoraphobia, and social phobia, while specific additive genetic effects were highest for Flight. Shared environmental effects were most pronounced for Fight across all models, with additional shared influences on BAS and BIS for panic, and BAS and Freeze for agoraphobia and social phobia. Nonshared environmental effects were the highest specific contributors across variables. Genetic overlap between anxiety disorders and rRST dimensions suggests pleiotropy, with unique environmental factors playing an important role in disorder development. While anxiety and fear may stem from distinct etiologies, their shared symptomatology complicates differentiation, highlighting the importance of considering both genetic and environmental influences in anxiety disorders.

1. Introduction

Anxiety and fear are emotions associated with responses of the organism to aversive stimuli, whether external or internally generated. Forming the basis for stable personality traits, they are often intertwined, blurring the fine distinctions needed for a better understanding of them and their behavioral consequences. Previous editions of the DSM (e.g., American Psychiatric Association, 2003) did not delineate fear from anxiety (McNaughton, 2011). Although DSM-5 (APA, 2013) distinguishes fear as an emotional response to a real or perceived immediate threat, from anxiety which is related to the anticipation of a future threat, generalized anxiety disorder (GAD), phobias and panic have been classified in the same group of disorders, named anxiety disorders. However, there is a growing body of evidence for different biological bases for these emotions, confirmed in animal models (McNaughton, 2011; McNaughton and Corr, 2022; Tovote et al., 2015), personality models (Lippold et al., 2020; McNaughton and Corr, 2016; Smederevac et al., 2022), and neuroimaging studies (e.g., Duval et al., 2015; Liu et al., 2022). In this study, we consider the nature of anxiety and fear from the perspective of the Revised Reinforcement Sensitivity Theory (rRST), a prominent model of personality neuroscience with significant implications for psychopathology (Gray and McNaughton, 2000).

1.1. Anxiety and fear in the rRST

The original Reinforcement Sensitivity Theory (RST) included three independent systems, related to impulsivity (Behavioral Approach System - BAS), anxiety (Behavioral Inhibition System - BIS) and aggressive or avoiding reactions (Fight-Flight System - FFS) (Gray, 1982). The

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revised RST (rRST) emerged from evidence distinguishing the biological bases of anxiety and fear (Gray and McNaughton, 2000). Evidence from pharmacological studies indicates that anxiolytics do not specifically target phobias, suggesting differing neurobiological foundations for these responses. Additionally, research on defensive distance (Blanchard et al., 2001) highlights threat proximity as crucial in defensive behavior. The rRST postulates that anxiety, which is regulated by the BIS, represents a reaction to a potential threat, while fear, which is regulated by the Fight/Flight/Freeze System (FFFS), is more of a reaction to an immediate threat. Therefore, the specific defensive behaviors generated by the FFFS depend on perceptive defensive distance, triggering Fight at small distance, Flight at long distance, and Freeze at intermediate distance.

Anxiety involves vigilance and arousal triggered by goal conflict, often arising from perceived insufficient resources to address ambiguous stimuli (Gray and McNaughton, 2000). This internal scanning process assesses coping capacity (Gray and McNaughton, 2000). Namely, stimuli alone are insufficient to cause conflict, since their meaning determines the response (McNaughton and Corr, 2004). Prolonged anxiety states can amplify responses to benign stimuli, while real threats evoke fear responses such as fight, flight, or freeze. Avoidant behavior underlies simple phobias, while Freeze, which reflects immobilization due to perceived futility in other responses, is linked to panic and inhibitory anxiety (Mihić et al., 2015).

BIS, Flight, and Freeze are interdependent, despite neurobiological distinctions (Smederevac et al., 2014). This interdependence is reflected in the comorbidity of anxiety spectrum disorders, psychometric challenges in distinguishing anxiety from fear, and linguistic vagueness in describing negative emotional states (Barlow et al., 2014; McNaughton, 2011; Ranđelović et al., 2018).

Previous studies have shown that defensive behavior is organized depending on the proximity of the relevant threat and the available behavioral options. Moreover, the dynamic organization of the process of encoding threat-related information and coordinating behavioral and physiological adaptation (Hamm, 2019), imply the combined involvement of the BIS, Flight, and Freeze in evaluating and responding to threats. Specifically, given the BIS' role in scanning the environment and encoding danger-relevant information, it likely has a fundamental role in all avoidance or freezing behaviors, especially in approach-avoidance conflict situations (Corr and McNaughton, 2012).

From the perspective of rRST, BIS is expected to be associated with anxiety as a response to potential threats, while FFFS forms fear reactions to immediate danger, modulated by the proximity of the threat.

1.2. Anxiety and fear in the theories of psychopathology

The distinction between anxiety and fear, and relatedly panic, is an important consideration within the realm of psychopathology. Panic attacks involve extreme fear, marked by fight-or-flight tendencies and intense autonomic responses, while anxiety reflects somatic tension and anticipation of future danger (Barlow, 2002). Regarding panic and fear, these states, while phenomenologically similar, may differ neurobiologically, though evidence remains limited (Barlow, 2002).

There is structural (statistical), experimental, and neuroimaging evidence supporting discrimination of anxiety and fear and their pathological forms (Barlow, 2002; Watson, 2005; Watson et al., 2022). When structural aspects are concerned, Krueger (1999) demonstrated that the internalizing disorders defined two different, but related factors: Anxious-Misery (GAD, depression, and dysthymia) and Fear (panic disorder, agoraphobia, social phobia, and specific phobia). Additional studies suggested that post-traumatic stress disorder (PTSD) belongs to Anxious-Misery whereas obsessive-compulsive disorder defines Fear (Slade and Watson, 2006; Vollebergh et al., 2001). Watson (2005) renamed the Anxious-Misery factor into the Distress factor in order to emphasize the dominant presence of non-specific negative affectivity in the disorders defining this factor as opposed to the dominant presence of fear/avoidance. Mood and anxiety disorders may be understood through the interplay of a common factor (e.g., negative affectivity), specific factors (e.g., anxious arousal in panic disorder), and unique factors (e.g., low positive affect in depression) (Kotov et al., 2007; Mineka et al., 1998; Watson, 2009). The nature of these factors remains unclear, but frameworks like rRST, emphasizing subsystems like Fight, Flight, and Freeze, provide a broader perspective (Clark and Watson, 2006).

Experimental studies further distinguish anxiety and fear. High baseline anxiety predicts panic attacks in individuals with panic disorder compared to control (Barlow, 2002), while conditioning to cues during initial panic attacks is central to panic disorder development (Bouton et al., 2001). Finally, a different degree of hypo- and hyper-activation in the neural circuits involved in emotional generation and modulation has been observed in different classes of anxiety disorders, in addition to the existence of common functional deficits (Duval et al., 2015). Also, a recent neuroimaging meta-analysis revealed that GAD seems to be characterized by decreased volumes in left insula and lateral/medial prefrontal cortex and increased right putamen volume compared to fear-based anxiety disorders (Liu et al., 2022).

From the perspective of psychopathological theories, it can be assumed that disorders classified as anxiety disorders in the DSM-5 share a common foundation reflecting general vulnerability, while exhibiting distinct features that align them more closely with either anxiety or fear.

1.3. Behavioral genetic foundation of the anxiety and fear

The twin-based design employed in this study is particularly relevant for testing the rRST framework because it allows for disentangling the genetic and environmental contributions to personality traits and psychopathological symptoms. Monozygotic twins, who share nearly all their genetic material, and dizygotic twins, who share approximately half, provide a unique opportunity to estimate heritability and the influence of shared versus non-shared environmental factors. This approach helps identify the common genetic and environmental influences that shape both personality traits and psychopathological symptoms, as well as the extent to which the etiology of specific characteristics is unique to each one, thereby enhancing understanding of their foundations.

Previous behavioral genetic studies have demonstrated moderate heritability for rRST dimensions: 33–34 % for BIS and 28–35 % for BAS (Takahashi et al., 2007), and 37 % for Flight to 44 % for Freeze (Smederevac et al., 2022). Avoidance dimensions like BIS, Flight, and Freeze share common genetic variance, with unique genetic variance suggesting generalized avoidance as a learned reaction to perceived threats (Smederevac et al., 2022). However, molecular genetic evidence further distinguishes genetic patterns underlying anxiety, fear, and panic within avoidance strategies (Smederevac et al., 2022).

Independent of personality traits, heritability estimates across the disorders ranged from 30 to 40 %, with individual environmental factors accounting for most variance (Hettema et al., 2001). A high comorbidity of anxiety disorders can be explained by common genetic risk factors (Hettema et al., 2005). Kendler et al.'s findings support the existence of common genetic risk for internalizing disorders, further suggesting that this risk can be divided into an ‘anxious-misery’ factor (i.e., depression, generalized disorder, and panic) and a ‘fear’ factor (i.e., animal and situational phobia) (Kendler et al., 2003). A recent study identified a common genetic factor underlying the symptoms of fear, anxiety, and depression, on the one hand, and the personality vulnerability (neuroticism, inhibition, anxiety sensitivity), on the other (Hettema et al., 2020). Moreover, two individual-specific environmental factors accounted for differentiation between anxiety/fear and depression (Hettema et al., 2020).

While distress and fear disorders share a genetic basis, subtypes of phobias are influenced by distinct genetic factors (Hettema et al., 2005; Tambs et al., 2009). Research has shown that there are no coherent genetic and environmental factors that explain all fears/phobias (Loken

et al., 2013; Mosing et al., 2009).

From a behavioral genetics' perspective, it is possible to expect both common and specific genetic and environmental influences on personality traits and internalizing disorders, with a special focus on identifying unique genetic and environmental factors that illuminate the etiology of anxiety and fear.

1.4. Current study

This study aims to investigate the shared genetic and environmental factors underlying traits from the Revised Reinforcement Sensitivity Theory (rRST) and symptoms of anxiety and fear as defined by DSM-5. From the rRST perspective, BIS is associated with anxiety as a response to potential threats, while the FFFS generates fear reactions to immediate danger, modulated by the proximity of the threat. In contrast, psychopathological theories suggest that anxiety and fear disorders in the DSM-5 share a common vulnerability but are distinct in their manifestation, with some disorders being more closely linked to anxiety and others to fear, depending on a constellation of additional specific and unique factors. This discrepancy creates a challenge in reconciling the rRST's biological framework for psychopathological syndromes anxiety and fear within clinical psychology.

From a behavioral genetics perspective, it is expected that both genetic and environmental factors influence personality traits and internalizing disorders, yet it remains unclear how these factors contribute differently to anxiety-related (BIS) versus fear-related (FFFS) traits. These varying perspectives suggest that while BIS and FFFS traits are expected to show genetic overlap with anxiety and fear symptoms, the complexity of these associations may differ depending on whether the focus is on broad personality dimensions or specific psychopathological outcomes. This presents a significant challenge in differentiating these constructs at both phenotypic and genetic levels.

The current study addresses this challenge by investigating the shared genetic and environmental factors between rRST dimensions and symptoms of anxiety and fear. While previous research has predominantly examined traits within the Five Factor model (Bienvenu et al., 2007; Welander-Vatn et al., 2019), or the original RST dimensions (Hettema et al., 2020), the rRST dimensions themselves remain underexplored in this context. This study aims to differentiate anxiety-related (BIS) and fear-related (FFFS) traits, specifically examining their interdependence and overlap with psychopathological symptoms. A core research question is whether it is feasible to distinguish symptoms of anxiety and fear-related disorders based on shared genetic and environmental factors with rRST personality traits, given that the genetic and phenotypic complexity of these symptoms may not correspond straightforwardly to the proposed associations of BIS with anxiety or Flight with phobias.

This study also incorporates the often-overlooked Fight system, which is less explored in anxiety and fear contexts, yet may play a significant role in understanding aggressive responses associated with these emotional states. The use of a twin-study design allows us to test hypotheses regarding the genetic and environmental contributions to BIS, FFFS, and Fight traits, and to explore whether the covariance between rRST dimensions and anxiety or fear-related symptoms arises from shared genetic versus environmental influences.

By examining the shared genetic and environmental underpinnings of rRST dimensions and symptoms of anxiety and fear, this study will contribute to understanding how these perspectives can inform a more integrated model of the etiology of anxiety and fear disorders.

2. Method

2.1. Participants and procedure

Participants were selected from the Serbian Twin Advanced Registry (see Smederevac et al., 2019). For this study, we selected a cohort consisting of twins who had available data for both the Reinforcement Sensitivity Questionnaire (RSQ) and the Psychiatric Diagnostic Screening Questionnaire (PDSQ). The final sample for analysis included 742 participants, corresponding to 371 twin pairs. The sample includes 178 monozygotic (MZ) (22.5 % males; age 17–60 years; $M = 25.73$; $SD = 8.3$) and 193 dizygotic (DZ) (33.9 % males; age 16–68; $M = 24.21$; $SD = 8.33$) adult twin pairs from the general population in Serbia (77,7 % pairs of same gender). All participants were White. Most of the participants had a master's degree (49.6 %), followed by those with high school degree (25.1 %) or college/bachelor's degree (19.9 %). A small percentage of twins had completed elementary school (1.6 %) and 3.5 % were students. Two participants did not provide information regarding their education background.

The research was approved by the Ethical Committee of the Faculty of Philosophy (#02-374/15) and the Committee for Ethics of Clinical Trials at the Faculty of Medicine (#01-39/229/1), University of Novi Sad. The call for participation was announced publicly through traditional and social media, as well as through the researchers' personal networks. Participants were recruited from the entire territory of Serbia (regions of Vojvodina, Central Serbia, West Serbia, and Southeast Serbia). Participation was voluntary and informed consent was obtained prior to the examination. The dataset is available online on the OSF platform: https://osf.io/gh3r8/.

2.2. Measures

Psychiatric Diagnostic Screening Questionnaire (PDSQ; Zimmerman and Mattia, 2001, for Serbian adaptation see Mihić et al., 2019) is a self-report measure consisting of 125 true/false items and designed to screen for 13 common DSM-IV Axis I disorders. Given our focus on anxiety disorders, only scales referred to the anxiety disorders in the DSM-5 (APA, 2013) were included in the analysis: panic disorder (6 items, $\alpha = 0.84$), agoraphobia (6 items, $\alpha = 0.80$), social phobia (6 items, $\alpha = 0.89$), and generalized anxiety disorder (6 items, $\alpha = 0.87$). Summed scores on each scale, reflecting the level of symptomatology, were used rather than diagnostic categories.

The Reinforcement Sensitivity Questionnaire (RSQ; Smederevac et al., 2014) is based on the Revised Reinforcement Sensitivity Theory (Gray and McNaughton, 2000). It contains 29 self-report items distributed in five scales: Behavioral Inhibition System - BIS (7 items, $\alpha = 0.79$), Behavioral Activation System - BAS (6 items, $\alpha = 0.73$), Fight/Flight/Freeze system (with 5 items each, $\alpha = 0.79, 0.75, 0.77$, respectively). Items are rated on a four-point Likert scale (from 1 = *completely disagree* to 4 = *completely agree*).

Twin zygosity was determined in two ways. First, by DNA analysis of the buccal swabs, tested using short tandem repeat (STR) megaplex kits, Investigator 24plex GO! (Qiagen®, Valencia, CA, USA) or GlobalFiler (Applied Biosystems®, ThermoFisher Scientific, Waltham, MA, USA) providing the two categories for each twin pair: monozygotic (MZ) or dizygotic (DZ) twin pair. Second, for twins who did not undergo DNA analysis of a buccal swab, zygosity was determined by the Questionnaire of Twins' Physical Resemblance (QTPR; Oniszczenko et al., 1993, for Serbian adaptation see Čolović et al., 2018). This self-report measure about mostly physical similarity between twins has proven to be a reliable indicator of zygosity with estimated accuracy around 95 % (Čolović et al., 2018). The measure, scoring procedures, and the discriminant functions used for classification, are described in detail in Lenau and Hahn (2017).

2.3. Data analysis

The estimation of the required sample size was calculated in the package *semTools* for R (Jorgensen et al., 2020) and was based on the RMSEA index (Kim, 2005). For additive genetic and nonshared environmental (AE) twin models with H0 RMSEA = 0.04, H1 RMSEA = 0.08, $\alpha = 0.05$, and power = 0.80, the optimal sample size (Sham et al., 2020)

for biometric analysis is about 500 participants. Moreover, for genetic and environmental correlations, even smaller samples are required (Zhang and Schumacher, 2021).

There was 1.38 % of missing data on the measures used. Little's MCAR test ($\chi^2(30) = 27.26$, $p > .05$) indicated that data are missing completely at random. Missing data was subjected to multiple imputation analysis with 5 imputations in IBM SPSS Statistics v.23 (IBM Corp., 2015). Imputed values from the 5th imputation were used for further analysis. To control for data distortion, regression was implemented to partial out sex and age of the used measures. The subsequent analyses were based on the standardized residuals.

Descriptive analysis and phenotypic correlations were carried out in IBM SPSS Statistics v.23 (IBM Corp., 2015). Phenotypic correlations were computed between twins within pairs on each scale (cross-twin correlations), as well as between two scales in one twin and the same two scales in their co-twin (cross-twin cross-trait correlations). A higher cross-twin correlation for MZ twins compared to DZ twins suggests a stronger influence of genetic factors relative to environmental factors on the measured phenomena. The cross-twin cross-trait correlation assesses the degree of covariation between one scale in a twin and another scale in both that twin and their co-twin. Substantial cross-twin cross-trait correlations indicate shared genetic or environmental influences contributing to the co-occurrence of phenomena measured by the two scales. Therefore, there is justification for testing multivariate biometric models.

Twin modelling was carried out in the *lavaan* package for R (Rosseel, 2012) as structural equation modelling (SEM), including both univariate and multivariate biometric approaches. Biometrical twin models compare the similarities between MZ twins, who share nearly 100 % of their genes, and DZ twins, who share about 50 % of their segregating genes, to estimate the relative contributions of genetic and environmental factors to variation in traits. Parameter A (Additive genetic factor) in biometrical twin modelling represents the additive effects of individual genes. If a trait is significantly influenced by additive genetic factors, MZ twins will be more similar than DZ twins. Parameter C (Common or shared environmental Factor) represents environmental factors that contribute to similarity between twins, regardless of zygosity. These are influences that both twins experience equally, such as the family environment. Parameter E (unique or non-shared Environmental factor) represents environmental influences that contribute to differences between twins. These are unique experiences that each twin encounters individually and include the measurement error (Figs. 1 and 2). Mathematically, the unique environmental variance (E) is calculated as the residual variance left unexplained by the additive genetic (A) and common environmental (C) factors, as the portion of variance that does not correlate within twin pairs, regardless of zygosity.

Univariate and multivariate twin modelling, based on the RSQ scales and one of the PDSQ anxiety scales, was conducted using customized R scripts (Čolović, 2019) to explore the phenotypic associations between these scales and identify the best-fitting phenotypic models. Genetic and environmental influences on phenotypic similarities between MZ and DZ twins were examined for each RSQ scale and the PDSQ anxiety scale using structural equation modelling (SEM), incorporating both univariate and multivariate biometric approach. Independent pathways (Fig. 1) and common pathways (Fig. 2) multivariate models (Rijsdijk and Sham, 2002) were employed to estimate additive genetic (A), shared environmental (C), and non-shared environmental factors (E), as well as specific (s) and common (c) genetic and environmental sources of variance. Independent models assume that the genetic and environmental influences on a trait operate independently of each other. The A, C, and E components are estimated separately without accounting for potential interactions or correlations between them. In contrast, common pathway models hypothesize the existence of a common underlying latent factor influenced by genetic and environmental factors, which subsequently affects the observed traits. This approach offers a more nuanced understanding of how different influences interact. For each independent and common pathway, full (ACE) as well as two reduced models (AE, CE) were tested. Consequently, six models were assessed for the multivariate model, comprising a combination of all RSQ scales and one of the PDSQ anxiety scales. The full model included Additive genetic factor (A), Common environmental factor (C), and non-shared Environmental factor (E). The reduced AE model comprised only the Additive genetic factor (A) and non-shared Environmental factor (E), assuming negligible influence from the Common environmental factor (C). Similarly, the reduced CE model included solely the Common environmental factor (C) and non-shared Environmental factor (E),

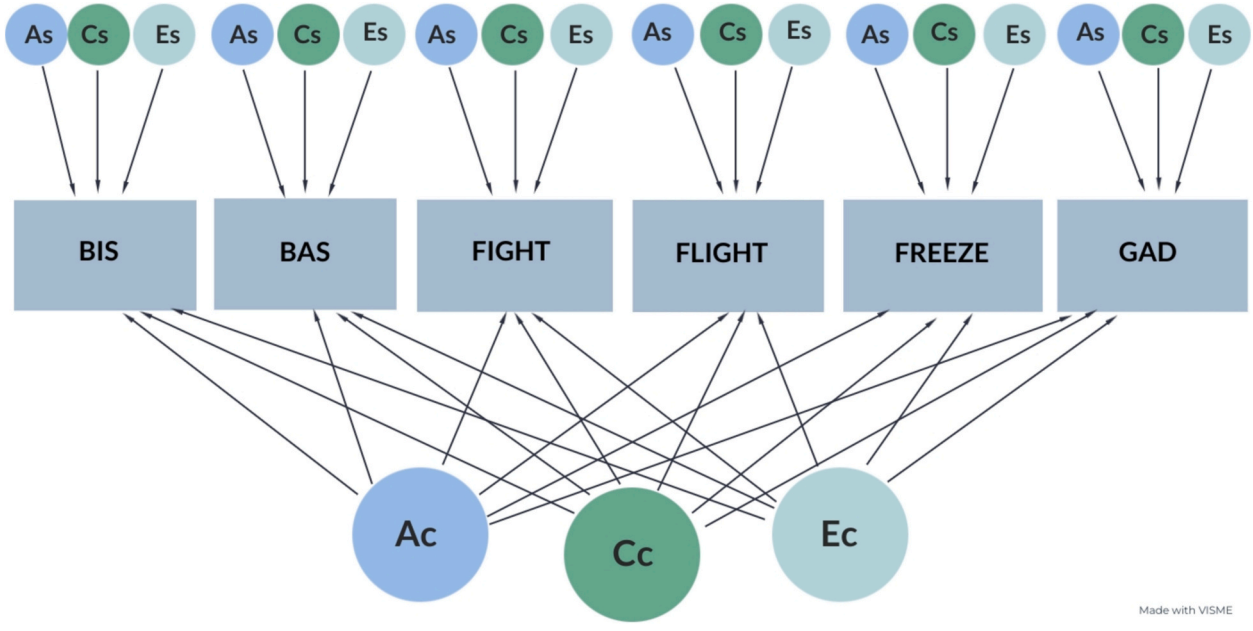

Fig. 1. Example of the independent pathway model – multivariate AE model for the RSQ dimensions and the PDSQ scale of generalized anxiety disorder (GAD). Notes. A_c – common additive genetic factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; E_s – specific nonshared environmental factor.

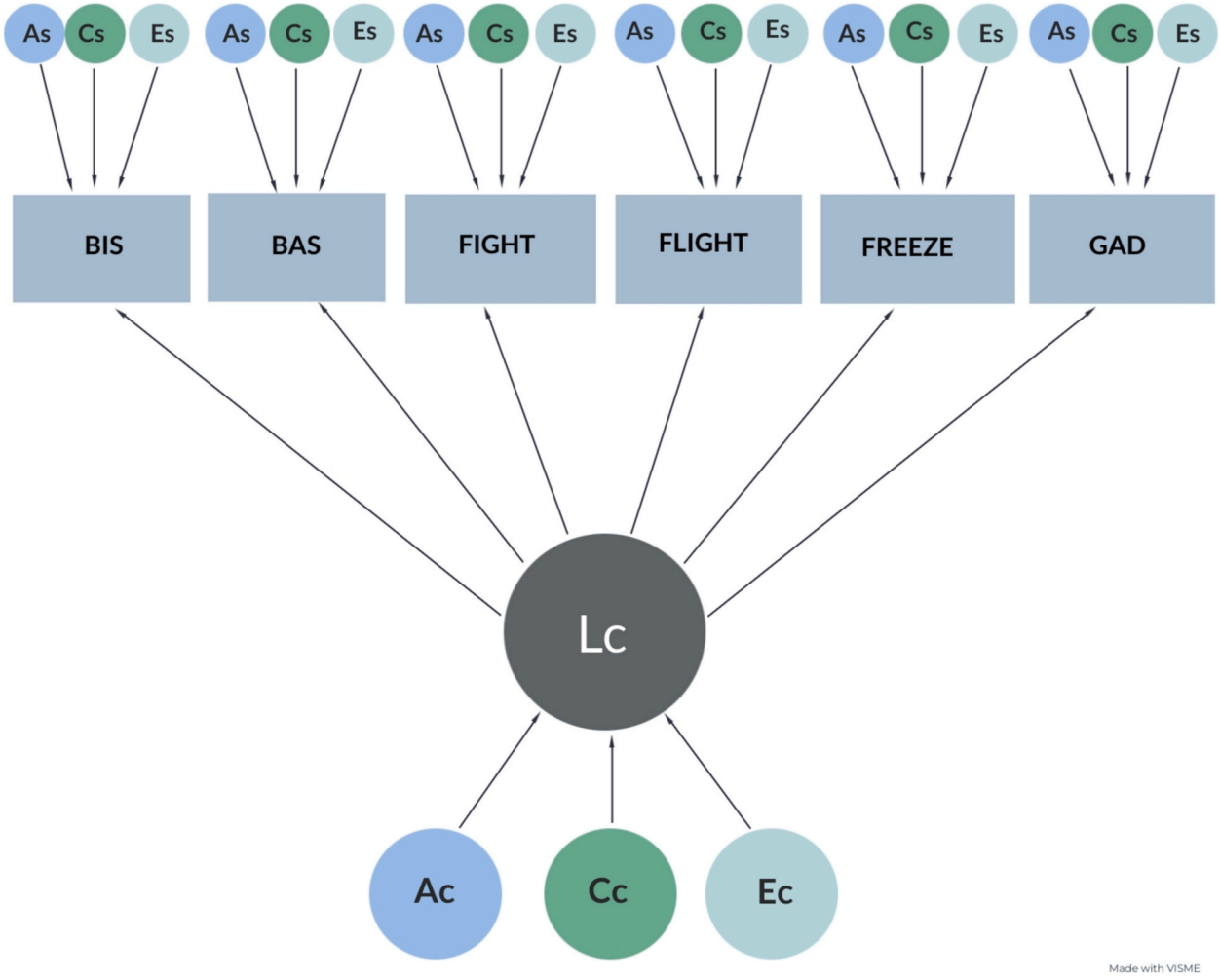

Fig. 2. Example of the common pathway model – multivariate AE model for the RSQ dimensions and the PDSQ scale of generalized anxiety disorder (GAD). Note. A_c – common additive genetic factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; E_s – specific nonshared environmental factor.

assuming negligible contribution from the Additive genetic factor (A).

Nested models were compared by using the $\Delta\chi^2$ test; the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) with a lower value indicating better fit; Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI) with optimal values higher than 0.95 and acceptable higher than 0.90; the Root Mean Square Error of Approximation (RMSEA) with optimal values lower than 0.05 and acceptable lower than 0.08; the Standardized Root Mean Square Residual (SRMR) with an acceptable value below 0.08. Furthermore, the patterns of genetic and environmental correlations among the RSQ and PDSQ dimensions were explored using Cholesky decomposition (see Gardiner et al., 2019) in the same R script.

3. Results

Table 1 shows the means and standard deviations of the RSQ traits as well as PDSQ anxiety scales for the MZ and DZ twins. The variables were normally distributed (acceptable values of skewness and kurtosis fall between −3 and +3, see Brown, 2006), except for panic and agoraphobia.

Table 1
Descriptive statistics of the RSQ traits and PDSQ anxiety scales.

	M		*SD*		Skewness		Kurtosis	
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ
BIS	15.27	16.17	4.23	4.37	0.23	0.20	−0.41	−0.42
BAS	16.63	16.63	3.32	3.21	−0.19	−0.25	−0.03	0.15
Fight	13.87	14.18	3.69	3.67	0.12	0.45	−0.17	0.03
Flight	13.22	13.42	2.74	3.04	−0.27	−0.14	0.42	−0.40
Freeze	10.17	10.17	3.00	3.25	0.41	0.36	0.11	−0.27
Panic	0.61	0.65	1.44	1.49	2.95	2.78	8.82	7.77
Agoraphobia	0.98	1.14	1.83	1.88	2.19	1.75	4.70	2.43
Social Phobia	2.33	2.50	3.37	3.41	1.45	1.51	1.19	1.55
GAD	2.33	2.33	2.77	2.90	1.12	1.09	0.20	−0.05

Note. MZ = monozygotic twins, DZ = dizygotic twins, GAD = general anxiety disorder.

MZ twins show stronger phenotypic cross-twin correlations than DZ twins in all variables except for panic, agoraphobia and GAD (Table 2). These results suggest that additive genetic effects are likely more pronounced across all variables, with the exception of panic, agoraphobia, and GAD, which appear to be more influenced by shared environmental effects. Moreover, significant cross-twin cross-trait correlations between the RSQ and the PDSQ anxiety scales indicate that there is a basis for testing multivariate twin models.

3.1. Genetic twin modelling

Univariate genetic twin modelling for all the RSQ personality traits and the PDSQ anxiety scales is given in Supplementary (Table A). Six different types of multivariate genetically informative models were tested for the RSQ scales and the each of the PDSQ anxiety scales. Genetically informative models, including the comparison of the independent and common pathways models, as well as of the full (ACE) and reduced (AE, CE) twin models, were performed (Table B in Supplementary). Overall, independent pathways models showed a better fit than common pathways models for all the multivariate models.

For the panic model, the reduced independent model, whereas all the genetic effects were assumed to be additive (AE), fit the data significantly poorer than the independent full twin model (ACE) ($\Delta\chi^2(12) = 37.036, p < .05$). Moreover, the reduced independent model with shared environmental effects (CE) also fitted the data significantly poorer than the full twin model (ACE) ($\Delta\chi^2(12) = 44.595, p < .05$). For the agoraphobia model, the best fitting model was the ACE independent pathways model, based on the same criteria ($\Delta\chi^2(12) = 51.185, p < .05$; $\Delta\chi^2(12) = 60.559, p < .05$, respectively). For the social phobia model, the most appropriate fit indices were also for the ACE independent pathways model ($\Delta\chi2(12) = 52.504, p < .05$; $\Delta\chi^2(12) = 66.750, p < .05$, respectively). When it comes to GAD, the independent reduced (AE) model also fits the data significantly poorer than the independent full (ACE) twin model ($\Delta\chi^2(12) = 42.893, p < .05$). Furthermore, the reduced independent model with shared environmental effects (CE) also fitted the data significantly poorer than the full twin model (ACE) ($\Delta\chi^2(12) = 52.685, p < .05$).

Table 3/6 presents parameter estimates for the retained ACE models. For all four models, the common additive genetic effects accounted for most of the variance of BIS (18 %), followed by Fight (16 %) and panic (14 %) for the panic model (Table 3), then agoraphobia (23 %) and Fight (12 %) for the agoraphobia model (Table 4), social phobia (31 %) and Freeze (10 %) for the social phobia model (Table 5) and BAS (35 %) and Freeze (17 %) for the GAD model (Table 6). The common shared environmental effects accounted for most of the variance of Fight (from 10 % to 30 %) in all models, along with BAS (21 %) and BIS (13 %) for the panic model, BAS (19 % to 22 %) and Freeze (7 % to 9 %) for the agoraphobia and social phobia model, and GAD (15 %) and BIS (9 %) for the GAD model. Moreover, specific additive genetic effects were the highest for Flight (20 % to 22 %) in all models. Specific shared environmental effects were generally low, while specific nonshared environmental effects were the highest specific effects for all the variables (Tables 3, 4, 5 and 6).

Cholesky behavior genetic ACE analyses were performed to explore total genetic and total environmental correlations of the RSQ personality traits and the PDSQ anxiety scales (Table 7). There were significant genetic and environmental (except agoraphobia) correlations between BIS and the anxiety scales. However, BAS was negatively genetically related to panic and social phobia, and positively environmentally related to panic and negatively to social phobia. Fight showed significant positive genetic correlations with all the anxiety scales but was significantly negative environmentally related to agoraphobia and social phobia. Moreover, Flight and Freeze also showed significant positive genetic and environmental correlations with all the anxiety scales, except for environmental correlations with agoraphobia for Flight and Freeze and except for environmental correlations with panic for Freeze (Table 7).

4. Discussion

This study examined to explore common genetic and environmental influences contributing to the co-occurrence of anxiety disorders and dimensions of the rRST. Building on existing evidence that distinguishes anxiety from fear (Gray and McNaughton, 2000), and their respective psychopathological expressions (Barlow, 2002; Krueger, 1999; Watson, 2005), our focus was specifically on identifying potential differences between fear-related and anxiety-related syndromes in terms of their underlying origins.

4.1. Personality and symptoms of mental disorders

Consistent with the previous studies (Smederevac et al., 2022; Takahashi et al., 2007), our findings revealed that genetic factors (37–44 %) and nonshared environmental influences account for individual differences in rRST systems. However, the psychopathological syndromes are primarily shaped by the environment, as heritability estimates for these disorders mostly do not exceed 0.30, except for social phobia. This result aligns with previous findings that the environment largely shapes distinct manifestations of emotional disorders (Kendler et al., 1987; Hettema et al., 2001).

The results also align with DSM-5 (APA, 2013), grouping GAD, phobias, and panic as anxiety disorders. Namely, genetic correlations showed that dimensions such as heightened BIS sensitivity and elements of the Fight, Flight, and Freeze systems collectively contribute to vulnerability to anxiety syndromes. An essential personality-related risk factor for developing anxiety disorders, regardless of whether the psychopathology leans toward fear or anxiety symptomatology, is the heightened sensitivity of the BIS, which implies increased anxiety. BIS activation often leads to activation of the FFFS (see McNaughton and Corr, 2018). Given that the BIS is responsible for scanning the

Table 2
Cross-twin cross-trait phenotypic correlations of the RSQ traits and PDSQ anxiety scales.

	BIS	BAS	Fight	Flight	Freeze	Panic	Agoraphobia	Social Phobia	GAD
BIS	0.38**/0.17*	−0.32**	0.00	0.50**	0.63**	0.27**	0.26**	0.53**	0.35**
BAS	−0.32**	0.46**/0.23**	0.31**	−0.24**	−0.25**	0.02	0.00	−0.14**	−0.01
Fight	0.00	0.31**	0.33**/0.31**	−0.06	−0.11**	0.11**	0.03	0.02	0.12**
Flight	0.5**	−0.24**	−0.06	0.40**/28**	0.51**	0.18**	0.16**	0.31**	0.18**
Freeze	0.63**	−0.25**	−0.11**	0.52**	0.39**/0.16*	0.22**	0.19**	0.39**	0.29**
Panic	0.27**	0.02	0.12**	0.18**	0.22**	0.19*/0.31**	0.43**	0.44**	0.49**
Agoraphobia	0.26**	−0.00	0.03	0.16**	0.19**	0.43**	0.20**/0.25**	0.49**	0.45**
Social Phobia	0.53**	−0.14**	0.02	0.31**	0.39**	0.44**	0.49**	0.46**/0.24**	0.54**
GAD	0.35**	−0.01	0.12**	0.18**	0.29**	0.49**	0.45**	0.54**	0.26**/0.29**

Note. GAD = general anxiety disorder; Cross-twin correlations of monozygotic and dizygotic twin pairs are given in the table diagonal and are separated by /.
* $p < .05$.
** $p < .01$.

Table 3
Common and specific genetic and environmental effects on the traits in multivariate twin models for panic.

Trait	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	0.18 (0.16, 0.67)	0.13 (0.04, 0.33)	0.41 (0.21, 0.66)	0.00 (0.00, 0.17)	0.04 (0.00, 0.13)	0.25 (0.08, 0.37)	0.18	0.16	0.66
BAS	0.00 (−0.04, 0.09)	0.21 (−0.25, 0.28)	0.06 (−0.08, 0.02)	0.18 (0.00, 0.34)	0.00 (0.00, 0.00)	0.55 (0.42, 0.65)	0.18	0.21	0.61
Fight	0.16 (0.06, 0.43)	0.22 (0.18, 0.38)	0.00 (0.00, 0.08)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)	0.62 (0.56, 75)	0.16	0.22	0.62
Flight	0.06 (−0.42, 0.03)	0.10 (0.08, 0.23)	0.21 (0.06, 0.32)	0.21 (0.13, 0.37)	0.00 (−0.03, 0.21)	0.42 (0.28, 0.50)	0.27	0.10	0.63
Freeze	0.07 (−0.44, 0.18)	0.12 (−0.13, 0.27)	0.38 (0.15, 0.56)	0.03 (0.00, 0.18)	0.10 (0.00, 0.24)	0.29 (0.18, 0.43)	0.11	0.22	0.67
Panic	0.14 (−0.04, 0.21)	0.01 (0.00, 0.03)	0.01 (0.00, 0.11)	0.00 (0.00, 0.00)	0.14 (0.00, 0.21)	0.70 (0.59, 0.85)	0.14	0.15	0.71

Note. Parameter estimates derived from the best fitting models. A_c – common additive genetic factor; C_c – common shared environmental factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; C_s – specific shared environmental factor; E_s – specific nonshared environmental factor; ΣA – total additive genetic effects; ΣC – total shared environmental effects; ΣE – total nonshared environmental effects; 95 % confidence intervals are given in parentheses.

Table 4
Common and specific genetic and environmental effects on the traits in multivariate twin models for agoraphobia.

Trait	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	0.17 (0.15, 0.43)	0.11 (0.09, 0.30)	0.43 (0.26, 0.71)	0.03 (0.00, 0.14)	0.04 (0.00, 0.13)	0.22 (0.03, 0.34)	0.20	0.15	0.65
BAS	0.01 (−0.47, 0.47)	0.19 (0.00, 0.40)	0.08 (−0.01, 0.21)	0.18 (0.00, 0.32)	0.00 (0.00, 0.00)	0.54 (0.44, 0.69)	0.19	0.19	0.62
Fight	0.12 (−0.17, 0.37)	0.24 (0.02, 0.46)	0.00 (−0.05, 0.02)	0.00 (0.00, 0.00)	0.00 (0.00, 0.24)	0.64 (0.56, 0.75)	0.12	0.24	0.64
Flight	0.08 (−0.05, 0.51)	0.10 (−0.01, 0.26)	0.20 (0.11, 0.31)	0.20 (0.00, 0.30)	0.00 (0.00, 0.19)	0.42 (0.31, 0.52)	0.28	0.10	0.62
Freeze	0.09 (−0.16, 0.35)	0.13 (0.01, 0.30)	0.35 (0.18, 0.56)	0.02 (0.00, 0.18)	0.10 (0.00, 0.18)	0.31 (0.20, 0.44)	0.12	0.22	0.66
Agoraphobia	0.23 (0.08, 0.34)	0.03 (−0.22, 0.30)	0.00 (−0.03, 0.01)	0.00 (0.00, 0.00)	0.02 (0.00, 0.22)	0.72 (0.58, 0.84)	0.23	0.05	0.72

Note. Parameter estimates derived from the best fitting models. A_c – common additive genetic factor; C_c – common shared environmental factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; C_s – specific shared environmental factor; E_s – specific nonshared environmental factor; ΣA – total additive genetic effects; ΣC – total shared environmental effects; ΣE – total nonshared environmental effects; 95 % confidence intervals are given in parentheses.

Table 5
Common and specific genetic and environmental effects on the traits in multivariate twin models for social phobia.

Trait	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	0.32 (0.01, 0.52)	0.07 (−0.11, 0.31)	0.36 (0.19, 0.53)	0.00 (0.00, 0.00)	0.00 (0.00, 0.10)	0.25 (0.17, 0.36)	0.32	0.07	0.61
BAS	0.01 (−0.43, 0.39)	0.22 (0.00, 0.32)	0.04 (0.00, 0.14)	0.17 (0.00, 0.37)	0.00 (0.00, 0.00)	0.56 (0.46, 0.72)	0.18	0.22	0.60
Fight	0.06 (−0.18, 0.36)	0.30 (0.07, 0.47)	0.00 (−0.06, 0.02)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)	0.64 (0.56, 0.75)	0.06	0.30	0.64
Flight	0.09 (−0.13, 0.29)	0.06 (0.01, 0.22)	0.22 (0.09, 0.34)	0.22 (0.10, 0.32)	0.00 (0.00, 0.21)	0.41 (0.31, 0.51)	0.31	0.06	0.63
Freeze	0.10 (−0.17, 0.31)	0.07 (−0.02, 0.24)	0.39 (0.21, 0.58)	0.03 (0.00, 0.59)	0.12 (0.03, 0.17)	0.29 (0.17., 0.40)	0.13	0.19	0.68
Social Phobia	0.31 (0.09, 0.60)	0.02 (−0.25, 0.32)	0.07 (0.01, 0.16)	0.12 (0.00, 0.28)	0.00 (0.00, 0.00)	0.48 (0.37, 0.58)	0.43	0.02	0.55

Note. Parameter estimates derived from the best fitting models. A_c – common additive genetic factor; C_c – common shared environmental factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; C_s – specific shared environmental factor; E_s – specific nonshared environmental factor; ΣA – total additive genetic effects; ΣC – total shared environmental effects; ΣE – total nonshared environmental effects; 95 % confidence intervals are given in parentheses.

Table 6
Common and specific genetic and environmental effects on the traits in multivariate twin models for GAD.

Trait	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	0.25 (0.00, 0.52)	0.09 (0.06, 0.31)	0.38 (0.19, 0.59)	0.00 (0.00, 0.16)	0.01 (0.00, 0.12)	0.28 (0.19, 0.37)	0.25	0.10	0.65
BAS	0.35 (0.08, 0.58)	0.01 (−0.20, 0.27)	0.00 (−0.05, 0.03)	0.09 (0.00, 0.29)	0.02 (0.00, 0.24)	0.53 (0.43, 0.64)	0.44	0.03	0.53
Fight	0.15 (−0.16, 35)	0.15 (0.05, 0.39)	0.01 (−0.02, 0.06)	0.00 (0.00, 0.00)	0.05 (0.00, 0.21)	0.64 (0.57, 0.78)	0.15	0.20	0.65
Flight	0.15 (0.00, 0.43)	0.00 (−0.13, 0.17)	0.22 (0.07, 0.34)	0.22 (0.14, 0.38)	0.00 (0.00, 0.21)	0.42 (0.28, 0.49)	0.37	0.00	0.63
Freeze	0.17 (0.00, 0.48)	0.01 (−0.19, 0.23)	0.41 (0.21, 0.63)	0.01 (0.00, 0.17)	0.12 (0.06, 0.20)	0.28 (0.14, 0.40)	0.18	0.13	0.69
GAD	0.01 (−0.26, 0.17)	0.15 (0.00, 0.35)	0.08 (0.02, 0.21)	0.00 (0.00, 0.00)	0.10 (0.00, 0.25)	0.66 (0.56, 0.79)	0.01	0.25	0.74

Note. Parameter estimates derived from the best fitting models. A_c – common additive genetic factor; C_c – common shared environmental factor; E_c – common nonshared environmental factor; A_s – specific additive genetic factor; C_s – specific shared environmental factor; E_s – specific nonshared environmental factor; ΣA – total additive genetic effects; ΣC – total shared environmental effects; ΣE – total nonshared environmental effects; 95 % confidence intervals are given in parentheses.

environment and detecting conflicting goals (Gray and McNaughton, 2000), pronounced sensitivity of this system may increase alertness to threatening stimuli, which can also facilitate the conditioning of fearful reactions. Thus, it seems that partly inherited increase in trait anxiety is the main vulnerability factor.

Apart from the so-called "avoidant" dimensions, the Fight system also shows mild to moderate genetic correlations with all psychopathological symptoms, indicating a shared genetic basis between defensive aggression and anxiety disorders. This indirectly implies the involvement of fearful-aggressive arousal in these conditions, particularly notable in agoraphobia and GAD.

These findings align with the triple vulnerability hypothesis (Barlow, 2000), positing heritable traits as prerequisites for mental disorders. The study also supports the view that negative affectivity, encompassing BIS and related traits, constitutes a general biological vulnerability for anxiety disorders (Brown and Naragon-Gainey, 2013; Barlow et al., 2014). Shared genetic factors across rRST systems and anxiety-related symptoms highlight pleiotropy and suggest potential avenues for future research into patterns of comorbidity.

4.2. Anxiety-related symptoms

Anxiety-related symptoms, such as GAD, demonstrate, as expected, strong genetic associations with BIS, followed by those with Freeze and

Table 7
Genetic and environmental correlations between the RSQ personality traits and the anxiety disorder – panic, agoraphobia, social phobia and GAD.

	Panic		Agoraphobia		Social Phobia		GAD	
	r_G	r_E	r_G	r_E	r_G	r_E	r_G	r_E
BIS	0.60** (0.56, 0.64)	0.09* (0.02, 0.16)	0.80** (0.77, 0.82)	−0.02 (−0.09, 0.05)	0.84** (0.82, 0.86)	0.28** (0.21, 0.34)	0.60** (0.56, 0.64)	0.21** (0.14, 0.28)
BAS	−0.08* (−0.15, −0.01)	0.08* (0.01, 0.15)	0.01 (−0.06, 0.08)	−0.02 (−0.09, 0.05)	−0.25** (0.18, 0.32)	−0.10* (−0.17, −0.03)	−0.05 (−0.12, 0.02)	0.03 (−0.04, 0.10)
Fight	0.22** (0.15, 0.29)	0.06 (−0.01, 0.13)	0.41** (0.35, 0.47)	−0.17** (−0.24, −0.10)	0.19** (0.12, 0.26)	−0.11* (0.04, 0.18)	0.42** (0.36, 0.48)	−0.01 (−0.08, 0.06)
Flight	0.29** (0.23, 0.35)	0.11* (0.04, 0.18)	0.52** (0.46, 0.57)	−0.01 (−0.08, 0.06)	0.49** (0.43, 0.54)	0.18* (0.11, 0.25)	0.22** (0.15, 0.29)	0.12* (0.05, 0.19)
Freeze	0.50** (0.44, 0.55)	0.05 (−0.02, 0.12)	0.64** (0.59, 0.68)	0.00 (−0.07, 0.07)	0.62** (0.57, 0.66)	0.20** (0.13, 0.27)	0.44** (0.38, 0.5)	0.18** (0.11, 0.25)

Note. Confidence intervals are given in parentheses.
* $p < .05$.
** $p < .01$.

Flight system sensitivities. The BIS's heightened alertness to threatening stimuli and its conflict-detection role (Gray and McNaughton, 2000) likely facilitate conditioning of worry, thereby contributing to trait anxiety and vulnerability to the development of GAD symptoms. Freeze responses, which inhibit cognitive processing in novel situations, are particularly prominent in GAD. Namely, Freeze may have an important role in the failure of controlled processing that leads to an inability to adjust cybernetic weights of the behavioral control system, important for all anxiety-related symptoms (Corr, 2011).

However, unique environmental factors, that do not influence other personality traits and psychopathological syndroms, play a crucial role in shaping GAD. Shared environmental influences, such as family dynamics fostering alertness to potential threats, may contribute to its development. These findings align with prior evidence suggesting the importance of both general and disorder-specific psychological vulnerabilities, shaped by environmental factors, in the manifestation of anxiety disorders (Barlow, 2000; Mitrović et al., 2023).

4.3. Fear-related symptoms

Fear-related symptoms, including panic, social phobia and agoraphobia, exhibited distinct genetic and environmental patterns. Panic disorder demonstrates strong associations with BIS and Freeze system sensitivities, similar to GAD. However, panic disorder's association with a unique shared environment suggests that specific family rules or experiences might inhibit cognitive processing capacities, promoting freezing as a reaction to fear. These findings align with modern learning theories emphasizing the roles of fear and anxiety in the development of panic disorder (Bouton et al., 2001).

Previous research has shown significant phenotypic and genetic associations between various forms of avoidant behavior (Hettema et al., 2020; Smederevac et al., 2022). However, the magnitudes of individual correlations in this study may raise questions about the nature of certain disorders, with possible implications for their classification. For example, social phobia exhibited the most pronounced connection with BIS, surpassing even GAD both in phenotypic and genetic associations. This is consistent with Corr's and McNaughton's view that social phobia should be considered more anxiety- than fear-related, despite the phobia label implying fear as its basis (McNaughton and Corr, 2016). Besides, in accordance with recent findings, in DSM-5 the label of this disorder was altered, resulting in its current designation as Social Anxiety Disorder (Social Phobia) (American Psychiatric Association, 2013). These results provide arguments for the classification of social phobia more with anxiety-related than fear-related disorders. The syndrome's negative genetic correlation with BAS suggests that reduced sensitivity to rewarding stimuli may underlie its etiology, consistent with the phenotypic association between low extraversion and social phobia (Smillie, 2013; Bienvenu et al., 2004).

In contrast, agoraphobia demonstrated generalized excitation across Fight, Flight, Freeze, and BIS dimensions. The Fight system's involvement suggests a predisposition for aggressive defensive responses to perceived threats, potentially linked to traits like low trust and high hostility (Bienvenu et al., 2004). Unlike social phobia, agoraphobia's genetic underpinnings reveal potential mechanisms of cognitive bias in threat assessment, highlighting an interplay between fear and defensive aggression in its manifestation.

Environmental factors also influenced fear-related symptoms of mental disorders. While some shared environmental influences are present, the impact of the nonshared environment is dominant. Unlike genetic factors, which psychopathological symptoms mostly share with the RSQ dimensions, environmental influences, especially nonshared, are primarily unique to individual disorders. Social phobia's unique environmental variance suggests individual-specific experiences, while agoraphobia's shared and nonshared environmental influences emphasize family and personal contexts in shaping disorder-specific vulnerabilities. According to the triple vulnerability model for anxiety disorders

(Barlow, 2000), a general biological vulnerability is important for any anxiety disorder to arise. The other two diatheses, general and disorder-specific psychological vulnerabilities, are both due to environmental influences. However, the most noteworthy finding in comprehending the environmental sources of anxiety disorder symptoms is that these individual experiences are unique to specific disorders and do not contribute to variations in personality traits (Mitrović et al., 2023). These results prompt further exploration into general environmental risk factors, and particularly specific individual experiences, which contribute to the development of anxiety disorders while not markedly affecting personality.

4.4. Limitations and future directions

The limitations of this study that may affect generalizability suggest that the results should be considered with caution. First, twin samples are very demanding to recruit, which contributes to the sample size, which can weaken the statistical power of the results. In this study, the sample allows for biometric models, since it exceeds the necessary lower limit (Sham et al., 2020). This limitation applies especially to the estimation of common shared environmental effects, which tend to be smaller in magnitude compared to genetic and non-shared environmental effects. Therefore, a larger sample size in future replication is certainly necessary to detect these effects.

Second, because the twins participated on a volunteer basis, there is a possibility of bias regarding phenomena relevant to the study. For example, it is possible that potential subjects with extreme scores on anxiety or phobias did not even apply to participate in the study. Therefore, we cannot guarantee the representativeness of the examined phenomena for the general population.

One limitation of the study is the homogeneity of our participant sample, which aligns with the WEIRD (Western, Educated, Industrialized, Rich, and Democratic) criteria outlined by Henrich et al. (2010). While Serbia encompasses a multicultural community with various nationalities such as Hungarians, Slovaks, Romanians, Ruthenians, or Croats, all participants in our study were White. Therefore, future research will require the inclusion of participants from a more diverse range of cultural, educational, and socioeconomic backgrounds.

Next, the PDSQ was developed before the new classifications of mental disorders and may not adequately separate anxiety and fear symptoms. At the time the study was conducted, DSM-5 had not yet been published, and we continued using the PDSQ, which aligns with DSM-IV criteria. Future studies might benefit from including measures that assesses the full range of phobias (e.g., the Phobic Stimuli Response Scale or Fear Survey Schedule-III) supplemented with the Inventory for Depression and Anxiety (IDAS-II) that taps 18 components of internalizing symptoms (Cutshall and Watson, 2004; Watson et al., 2012; Wolpe and Lang, 1964).

Also, a nonshared environment in a behavioral genetic design usually includes measurement error, which leaves the possibility that part of its variance can be attributed to the psychometric properties of the questionnaires. Namely, the PDSQ assesses the symptoms of mental disorders that usually do not have a normal distribution in the population (Mitrović et al., 2023), while the basic premises of the RST are difficult to fully include in the questionnaires, due to many limitations arising from linguistic problems and comorbidities among phenomena (Corr and Cooper, 2016; Krupić et al., 2016; Smederevac et al., 2014).

5. Conclusions

The results show that the symptoms of mental disorders, except for social phobia, do not have a specific genetic variance, but rather share it with personality traits. There is no simple isomorphism between one personality system and psychopathological syndromes, and the differential activation of all rRST systems plays an important role in the dimensions of anxiety syndromes. Despite arguments about the common genetic basis of all internalizing disorders, there is a need to delineate their specificities, in which personality traits play a key role. This result has important implications for all contemporary theories of psychopathology (e.g., Kotov et al., 2007) which favor a dimensional approach to mental disorders.

A key finding is the dominant role of the hierarchically superordinate BIS system, strongly associated with anxiety, in contributing to comorbidity across most syndromes (Kendler et al., 1995). The BIS serves as a general vulnerability factor, aligning with the Integrative Hierarchical Model (Kotov et al., 2007; Mineka et al., 1998). Additionally, the Freeze system, traditionally linked to panic, plays a pivotal role in all anxiety-related symptoms. Its function as a cognitive regulator, impeding the processing of novel and threatening stimuli, suggests its involvement in generating fear through the perception of insufficient internal resources to address challenges.

Second, the results show that symptoms of social phobia correspond more to states of anxiety than fear, which is in line with Corr's and McNaughton's view (McNaughton and Corr, 2016) and its current designation as Social Anxiety Disorder (American Psychiatric Association, 2013). Furthermore, the negative genetic correlation between BAS and social phobia reinforces the role of low positive affectivity, which is characteristic of these symptoms (Watson, 2009). Importantly, the contributions of Fight and BAS systems to anxiety syndromes, often overlooked, emphasize the relevance of approach behavior in understanding these disorders.

Third, while genetic factors shared with personality traits are integral, environmental influences emerge as primary drivers of symptom specificity. Adaptation within shared and non-shared environments, facilitated by learning processes that develop skills to manage threats, significantly shapes the manifestation of anxiety disorders.

Although anxiety-related symptoms can have an independent etiology, rooted in the environment as in the case of GAD, or a more specific genetic basis as in the case of social phobia, fear-related symptoms invariably include an anxiety component. This overlap contributes to the challenges in clearly delineating these disorders, highlighting the complexity of their shared and unique vulnerabilities.

CRediT authorship contribution statement

Snežana Smederevac: Writing – original draft, Methodology, Investigation, Funding acquisition, Conceptualization. **Dušanka Mitrović:** Writing – original draft, Investigation, Conceptualization. **Ljiljana Mihić:** Writing – original draft, Conceptualization. **Selka Sadiković:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Bojana M. Dinić:** Writing – original draft, Investigation, Data curation. **Aleksandra Milutinović:** Investigation, Data curation. **Radomir Belopavlović:** Formal analysis, Data curation. **Philip J. Corr:** Writing – original draft, Supervision.

Ethics approval statement

The data were collected in a manner consistent with ethical standards for the treatment of human subjects. The research was approved by the Institutional Ethics Committee of the Faculty of Philosophy (#02-374/15) and the Committee for Ethics of Clinical Trials at the Faculty of Medicine (#01-39/229/1), University of Novi Sad, Serbia.

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Declaration of competing interest

The author(s) declare that there were no conflicts of interest with

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None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2024.12.101.

Data availability

The dataset of this study is openly available in the Open Science Framework (OSF) repository at https://osf.io/gh3r8/.

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