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Citation: Smederevac, S., Mitrović, D., Mihić, L., Sadiković, S., Dinić, B. M., Milutinović, A., Belopavlović, R. & Corr, P. J. (2025). Demarcation of anxiety and fear: Evidence from behavioral genetics. *Journal of Affective Disorders*, 373, pp. 208-218. doi: 10.1016/j.jad.2024.12.101

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Permanent repository link: <https://openaccess.city.ac.uk/id/eprint/34923/>

Link to published version: <https://doi.org/10.1016/j.jad.2024.12.101>

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Demarcation of anxiety and fear: Evidence from behavioral genetics

Abstract

Anxiety and fear, as emotions associated with responses of the organism to aversive stimuli, are often intertwined, blurring the fine distinctions necessary for a better understanding of them and their consequences. This study examined common genetic and environmental influences that contribute to the co-occurrence of anxiety disorders and dimensions of the revised Reinforcement Sensitivity Theory (rRST). The sample included 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. Scales for panic disorder, agoraphobia, social phobia, and generalized anxiety disorder from the Psychiatric Diagnostic Screening Questionnaire, and Behavioral Inhibition System (BIS), Behavioral Activation System (BAS), and the Fight/Flight/Freeze System (FFFS) scales from the Reinforcement Sensitivity Questionnaire (RSQ) were used. Common additive genetic effects accounted for: most of the variance of BIS; then fight and panic for the panic model; agoraphobia and fight for the agoraphobia model; social phobia and freeze for the social phobia model; and BAS and freeze for the GAD model. The common shared environmental effects accounted for most of the variance of fight in all models, along with: BAS and BIS for the panic model; BAS and freeze for the agoraphobia and social phobia model; and GAD and BIS for the GAD model. Specific additive genetic effects were the highest for flight in all models. Specific shared environmental effects were generally low, while specific nonshared environmental effects were the highest specific effects for all the variables. The genetic factors underlying individual differences in psychopathological

dimensions are, in most cases, fully shared with the RSQ dimensions, implying a common genetic basis of anxiety disorders and these personality systems. This is further supported by the patterns of genetic correlations between anxiety disorders and personality traits, which generally mirror the patterns of phenotypic correlations. The apparent lower environmental correlations suggested that shared genetics primarily underlie the phenotypic associations. The genetic association of the rRST system and anxiety disorders points to pleiotropy and further highlights the importance of unique environmental factors as pivotal to the development of disorders.

Key words: anxiety, fear, PDSQ, rRSQ, twin study

Introduction

Anxiety and fear are emotions associated with responses of the organism to aversive stimuli, whether external or externally generated. As well as forming the basis for stable personality traits, they are often intertwined, blurring the fine distinctions needed for a better understanding of them and their consequences behavior. 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 & Corr, 2022; Tovote et al., 2015), personality models (Lippold et al., 2020; McNaughton & Corr, 2016; Smederevac et al., 2022), and neuroimaging studies (e.g., Duval et al., 2015; Liu et al., 2021). In this study, we consider the role of anxiety and fear from the perspective of the Revised Reinforcement Sensitivity Theory (rRST), which is a prominent model of personality neuroscience with significant implications for psychopathology (Gray & McNaughton, 2000).

Anxiety and fear in the rRST

The revision of Reinforcement Sensitivity Theory (rRST) was necessitated by evidence of the different biological bases of anxiety and fear (Gray & McNaughton, 2000). One group of evidence arises from pharmacological research, which has shown that anxiolytics have no

specific preferential effect on phobias, pointing to a different neurobiological bases of these clinical domains. Another group of evidence derives from the research on defensive distance (Blanchard et al., 2001), which has shown that the assessment (subjective perception) of the distance of the threat is an important factor in defensive behavior. rRST postulates that anxiety, which is regulated by the Behavioral Inhibition system (BIS), represents a reaction to a *potential* threat (as seen in goal conflict); while fear, which is regulated by the Fight/Flight/Freeze System (FFFS), is more of a reaction to a real (i.e., immediate and high intensity) threat, the specific defensive behaviours of the FFFS depend, crucially, on perceptive defensive distance: at small distance, Fight; at long distance, Flight; and at intermediate distance, Freeze.

Anxiety is closely related to vigilance and attention alertness, which is preceded by the existence of a (goal) conflict that increases arousal (Gray & McNaughton, 2000). The conflict may arise from the assessment of lack of resources to cope with potentially threatening, new or ambiguous stimuli. This "internal scanning", as a search for a previous similar experience, is a resource for assessing the capacity to cope with the situation (Gray & McNaughton, 2000). It is important to emphasize that stimuli are not a sufficient reason for causing conflict, since their "meaning" determines the response (McNaughton & Corr, 2004). In other words, in prolonged anxiety states, even benign stimuli can provoke an intense emotional response.

Unlike a potential threat, a real threat evokes fear, which elicits different types of responses, such as fight, flight, or freeze. If the threat is sufficiently distant, the response is flight since there is an assessment that the danger can be avoided. Avoidant behavior can be the basis for the development of simple phobias (McNaughton & Corr, 2016). Freeze is a reaction to a threat that involves the experience of physical or cognitive immobilization, due to the assessment that any other reaction to danger is useless – it is also a way to reduce stimuli associated with

motion/movement. Although freeze is considered fundamental to panic, it can be associated with physical symptoms of anxiety as well as inhibitory anxiety (Mihic et al., 2015) – however, it can be difficult to dissociate behaviourally BIS-related passive avoidance and FFFS-related freezing (drug challenges are important to dissociating these behavioural propensities). Also, unlike simple phobias, social phobias are related to the anxiety spectrum (McNaughton & Corr, 2016), as they are associated by goal conflict and are thus BIS-related. However, BIS, Flight, and Freeze are interdependent and should not be considered separately (Smederevac et al., 2014). There are, at least, three sets of reasons for this interdependence, which are relevant despite conceptual differences and evidence of their different neurobiological bases. Those reasons are comorbidity between anxiety spectrum disorders, psychometric imperfection, and linguistic vagueness. First, it is still not clear the precise relations of the comorbidity at the level of anxiety, phobia and panic symptoms and the interaction between BIS, Flight and Freeze. For example, an increase in anxiety may be likely in the face of sources of fear, while many situations that provoke fear involve also conflict, which increases anxiety. On the other hand, freeze is not necessarily followed by flight reactions, while active BIS is not necessarily followed by flight or freeze (Mihic et al., 2015). Second, the expectation of complete independence of these three systems is not plausible, since they all cover reactions to unpleasant stimuli, which contributes to their conceptual similarities, as well as similarities at the measurement level. The best example is neuroticism, which in most personality models covers all three types of possible reactions to aversive stimuli (e.g., Barlow et al., 2014). In short, personality questionnaires usually do not allow for the assessment of subtle differences between anxiety and other types of reactions from the spectrum of negative emotionality. Finally, an important issue related to this problem is linguistic. The limited number of words in everyday language used to describe negative

emotional states contributes to the lack of precision in their use. Thus, the same descriptions are often used for reactions associated with both real and potential threats. The tendency of people frequently to use the same terms to describe different phenomena (McNaughton, 2011) affects participants' responses to items containing explicitly named emotional states such as panic, anxiety, or fear, which may be more influenced by the negative connotations of those terms than by their specific meanings. These interdependencies are to be expected on the basis of BIS activation leading to activation of the FFFS, although the obverse is not obligatory (see McNaughton & Corr, 2018).

Previous studies have shown that defensive behavior is dynamically organized by the proximity of a relevant threat and available behavioral options, as well as the dynamic organization of the process of encoding information relevant to the threat and organizing behavioral adaptation, including accompanying physiological changes (Hamm, 2019) implying the combined action of the BIS, Flight, and Freeze systems in threatening situations. In other words, since the BIS has the task of scanning the environment and encoding information relevant to the danger, it is probably often associated with avoidance behavior or freeze, especially in situations containing approach-avoidance conflict (Corr & McNaughton, 2012).

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. Barlow, Brown and Craske (1994) have defined panic attacks as emotional states that are subjectively experienced as extreme fear, related behaviorally to action tendencies of fight or flight, and produce intense autonomic reactions. Different from them, anxiety states are characterized by anticipation of future danger,

ruminative and worrisome thinking, and accompanied by somatic sensations of tension (Barlow, 2002). Within the psychopathology literature, panic attacks are considered clinical manifestations of emotion of fear, but it has been noted that these two states, although phenomenologically similar, might be different at a neurobiological level (Barlow, 2002). However, clear neurobiological evidence for their demarcation within the psychopathology literature is currently lacking.

There is more agreement regarding the distinction between anxiety and fear. There is structural (statistical), experimental, and neuroimaging evidence supporting discrimination of these affective states and their pathological forms (Barlow, 2002; Watson, 2005). When structural aspects are concerned, Kruger (1999) demonstrated, using a series of confirmatory factor analyses on the National Comorbidity Survey data, 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). These findings were replicated subsequently on different national samples (Slade & Watson, 2006; Vollebergh et al., 2001), suggesting additionally that post-traumatic stress disorder (PTSD) belongs to Anxious-Misery whereas obsessive-compulsive disorder defines Fear (Slade & Watson, 2006). 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 in the disorders defining the Fear factor. Based on these findings, it has been proposed that a diagnostic classification system should reflect the fact that GAD has more in common with major depressive disorder than other anxiety disorders (Clark & Watson, 2006; Watson, 2005).

Maybe we could make use of the following?

In a quite separate part of the psychopathology literature, the distinction between fear and anxiety has been identified. A behavioural genetic study of ten major psychiatric disorders, in a sample of 5,600 twins (Kendler, Prescott, Myers and Neale 2003) revealed the following findings: (a) two major dimensions emerged, one relating to internalizing disorders (i.e., major depression, generalized anxiety disorder and phobia), the other to externalizing disorders (i.e., alcohol dependence, drug abuse/dependence, adult antisocial behaviour and conduct disorder); (b) no differences in genetic and environmental influences for males and females, despite the large difference in prevalence rates; (c) unique (i.e., non-shared family) environment effects for internalizing disorders; (d) and, of most relevance to RST, the structure of genetic risk for internalizing disorders broken down into an ‘anxious misery’ factor (i.e., depression, generalized disorder and panic) and a specific ‘fear’ factor (i.e., animal and situational phobia). Earlier, Prescott and Kendler (1998) noted that mild depression and generalized anxiety do not appear to have distinct genetic etiologies, but rather a common genetic basis, perhaps a disposition to dysphoric mood which is shaped by individual experiences into symptoms of depression, anxiety, or both. (See also, Kendler et al. (1992.) As Kendler et al. (2003, p. 935) themselves speculated, ‘It is tempting to speculate that these genetic factors on risk might be mediated through personality.’

In order to explain these structural findings and the patterns of comorbidity among mood and anxiety disorders, the Two-Factor Affective model and its subsequent revisions have been proposed (Clark & Watson, 1991; Mineka et al., 1998; Watson & Tellegen, 1985; Zinbarg &

Barlow, 1996). Its most recent version, Integrative Hierarchical model, stipulates that each mood and anxiety disorder and their comorbidity can be understood by relative contributions of three factors: a) a common factor contributing to all disorders, b) specific factor(s) shared by some but not other disorders, and c) a unique factor that differentiates one disorder from all other disorders (Kotov et al., 2007; Mineka et al., 1998). For example, negative affectivity (NA)/neuroticism is considered one such common factor, anxious arousal is seen as a specific factor contributing to panic disorder and PTSD, whereas low positive affect seems specific to major depression and social phobia (Watson, 2009). However, even this model has its drawbacks: it cannot satisfactorily explain all observed patterns of co-morbidity, and more importantly, it is unclear about the number and the nature of the specific and, in particular, unique factors (Watson, 2009). For example, one stumbling point for the model is the fact that specific and social phobias are strongly correlated, more so than it would be expected given a lesser contribution of NA to these two syndromes (Watson, 2009). Also, the nature (phenotypic or genetic) of these specific/unique factors is unknown (Clark & Watson, 2006). One resolution would be not to rely only on the dimensions originating from psychology of affect but to consider the rRST as a useful framework given its emphasis on the existence of more diverse subsystems (Fight, Flight, Freeze) underlying fear reactions, and their interactions with BIS.

Apart from structural data, there is abundant experimental evidence supporting the distinction between anxiety and fear (panic). By reviewing a number of studies that used very different procedures to provoke panic in the laboratory, Barlow (2002) concluded that high baseline anxiety was a very strong predictor of experimentally induced panic attacks in people with panic disorders compared to controls (Barlow, 2002). Additionally, based on experiments with fear learning, a modern learning account of panic disorder has been proposed (Bouton et al.,

2001). According to this view, during initial panic attacks, there is a surge in anxiety due to the unexpected nature of these attacks, leading to conditioning of anxiety to internal and external cues present during the attacks (Bouton et al., 2001). At the same time, panic attacks themselves can be conditioned to certain internal cues (interoceptive conditioning). Conditioned anxiety underlies anticipatory anxiety and avoidance behavior characterizing panic disorder whereas interoceptive conditioning of certain bodily sensations can lead to unexpected panic attacks (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). For example, all anxiety disorders seem to be characterized by hyperactivation of emotion-generating regions (amygdala and insula) and hypoactivation in prefrontal regions serving emotion regulation and modulation purposes (Duval et al., 2015). However, in fear-based disorders (panic disorder and specific phobias), there is a greater activation in the emotion-generating regions, whereas in GAD, PTSD and some forms of social anxiety disorder greater hypoactivation of medial prefrontal cortex was observed (Duval et al., 2015). Also, a recent neuroimaging meta-analysis revealed distinct structural anomalies characterizing GAD compared to fear-based anxiety disorders (Liu et al., 2021). 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.

Behavioral genetic foundation of the anxiety and fear

Like all other basic personality traits, previous behavioral and genetic studies have shown that the dimensions of rRST manifest moderate heritability, which ranks from 33–34% for BIS

and 28–35% for BAS (Takahashi et al., 2007), or from 37% for Flight to 44% for Freeze (Smederevac et al., 2022). By considering the distinction between response to potential and real threat, which is key to the demarcation of anxiety and fear, the results showed that BIS, Flight and Freeze, as avoidance dimensions, share common genetic variance, with a significant portion of the unique genetic variance (Smederevac et al., 2022). Namely, it is possible that there are similar genetic and central mechanisms underlying all types of avoidance behavior and that learned avoidance is a generalized reaction to all perceived threatening situations. However, molecular genetic evidence indicated the association of the serotonin gene *TPH2* only with BIS, but not with Flight and Freeze, which indicates a clear genetic difference between anxiety and fear. In other words, within the general avoidance strategy, there are specific genetic patterns that allow the distinction between anxiety, fear, and panic (Smederevac et al., 2022).

Considering the symptoms of fear and anxiety independent of personality traits, meta-analysis based on twin and family data showed that estimated heritability across the disorders are in the range of 30%–40%, leaving most of the variance in liability to be largely explained by individual environmental factors (Hettema et al., 2001). Anxiety disorders have a high comorbidity, which can be explained by common genetic risk factors (Hettema et al., 2005). Also, in the children's sample, fear and anxiety showed a similar etiological structure, which included moderate genetic and specific environmental influences for all symptoms (Sawyers et al., 2019), suggesting that relevant genetic and environmental effects are not clearly delineated.

Despite the shared genetic basis, some research suggests that although distress and fear disorders share a genetic basis, at least some subtypes of phobias are influenced by other genetic factors (Hettema et al., 2005; Tambs et al., 2009). More specifically, research has shown that there are no unique genetic and environmental factors that explain all fears/phobias (Loken et al.,

2013; Mosing et al., 2009). Namely, there are results which do not support the separation of agoraphobia from social phobia, clearly indicating the absence of a coherent genetic factor for the DSM-IV and DSM-5 situational phobia (Loken et al., 2013).

Current study

The high comorbidity between anxiety disorders contributes to the difficulties in delimiting their symptoms, both on the phenotypic and on the etiological level. Therefore, studies examining the common etiological factors of personality traits and anxiety disorders are highly informative. For example, there are results that indicate avoidant personality disorder and social anxiety disorder differ in the degree to which they share genetic and environmental influences with neuroticism, extraversion, and openness to experience, showing that these Five factor model (FFM) traits are a source of disorder-specific genetic and environmental influences (Welder-Vatn et al., 2019). Furthermore, genetic factors influencing individual variation in FFM (low) extraversion and neuroticism fully explain the genetic predisposition to social phobia and agoraphobia, but not animal phobia (Bienvenu et al, 2007). Also, examination of the common etiological factors of behavioral inhibition and social anxiety in children indicated the existence of their common genetic roots, but also that the variability of social anxiety is influenced by other genetic effects that are independent of behavioral inhibition (Goldsmith et al., 2022).

The main purpose of this study is to examine the common and specific etiological factors of the rRST traits and symptoms of mental disorders related to anxiety and fear according to DSM-5. Since the rRST is based on evidence that points to a different biological basis of anxiety and fear (Gray & McNaughton, 2000), it represents a relevant personality model for examining

these phenomena. Although rRST postulates possible associations between BIS with anxiety symptoms, Flight with phobias, and Freeze with panic, the complex genetic basis and phenotypic manifestations of these symptoms can hardly have simple isomorphisms. Therefore, an examination of the common genetic and environmental bases of personality and anxiety and fear symptoms will involve all rRST systems, both avoidant and approach. Namely, according to rRST, one of the possible reactions to fear is Fight, as an aggressive response whose role in states of anxiety and fear has not been sufficiently examined. Moreover, the examination of common and specific genetic and environmental factors that can potentially explain the covariation of the rRST dimensions and symptoms of anxiety and fear provides additional insight into the shared etiology of these phenomena.

I suppose there is considerable scope for reducing the above, in order to focus on the central focus of the paper – but, I can see, this is tricky. Maybe one way would be to work back from the results to see what should be the focus in the Introduction and what is more secondary material.

Having said that, so much of the Introduction is relevant!

Method

Participants and Procedure

Participants were selected from the Serbian Twin Advanced Registry (see Smederevac et al., 2019) based on completion of measures by both twins (742 participants or 371 twin pairs). The sample includes 178 monozygotic (22.5% males; age 17–60 years; $M = 25.73$; $SD = 8.3$) and 193 dizygotic (33.9% males; $M = 24.21$; age 16–68; $SD = 8.33$) adult twin pairs (83) different gender?? (22.4%) maybe clarify this section from the general population in Serbia. All participants were Caucasians. Most of the participants had a master's degree (49.6%), high school degree in education (25.1%) or college/bachelor's degree (19.9%), elementary school had 1.6% twins and 3.5% twins were students. Two participants had missing education information.

The twin study was approved by the Institutional Review Board (# 20111020000004_e1b8). One part of the sample (57.95%) was examined through a computer-based platform designed for the examination of twins, and for 42.05%, double entry was performed with a check for mismatches and out-of-range values to avoid missing or incorrectly entered data. Participants were recruited from the entire territory of Serbia (regions of Vojvodina, Central Serbia, West Serbia, and Southeast Serbia) through psychology students of the University of Novi Sad, citizen scientists, as well as via public calls in media, social networks, and similar sources. Participation was voluntary and informed consent was obtained prior to the investigation. The data and instructions for this study are available online on the OSF platform: <https://osf.io/gh3r8/>.

Measures

Psychiatric Diagnostic Screening Questionnaire (PDSQ; Zimmerman and Mattia, 2001, for Serbian adaptation see Mihić et al., 2019) is a self-report measure consists of 125 true/false items and designed to screen for 13 common DSM-IV Axis I disorders, including major

depressive disorder, bulimia, posttraumatic stress disorder, panic disorder, agoraphobia, social phobia, generalized anxiety disorder, obsessive-compulsive disorder, alcohol abuse/dependence, drug abuse/dependence, somatization, hypochondriasis, and psychosis. 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, $\alpha = .84$), agoraphobia (6 items, $\alpha = .8$), social phobia (6 items, $\alpha = .89$), and generalized anxiety disorder (6 items, $\alpha = .87$).

The Reinforcement Sensitivity Questionnaire (RSQ; Smederevac et al., 2014) is based on the Revised Reinforcement Sensitivity Theory (Gray & McNaughton, 2000). It contains 29 items distributed in five scales: Behavioral Inhibition System - BIS (7 items, $\alpha = .79$), Behavioral Activation System - BAS (6 items, $\alpha = .73$), Fight/Flight/Freeze system (with 5 items each, $\alpha = .79, .75, .77$, respectively). Items are rated on a four-point Likert scale (from 1 = *completely disagree* to 4 = *completely agree*).

Twin zygosity was determined using buccal swabs as well as questionnaire responses (for those twins examined through a computer-based platform). DNA zygosity was determined 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.

The Questionnaire of Twins' Physical Resemblance (QTPR; Oniszczenko et al. 1993, for Serbian adaptation see Čolović et al., 2018). This questionnaire is a self-report measure containing 31 sets of items referring to the biological and physical indicators of twins' resemblance. The questionnaire asked the twins to describe and compare themselves to their co-

twin on several physical characteristics (e.g., weight, height, hair color and type, and eye color), as well as to rate the degree to which their parents, relatives, peers, and strangers mixed with each other. The use of a questionnaire to determine zygosity has proven to be a reliable indicator of zygosity (estimate 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).

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 H_0 RMSEA = .04, H_1 RMSEA = .08, α = .05, and power = .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 & 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 scales. 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). Twin modelling was carried out in the *lavaan* package for R (Rosseel, 2012). Univariate and multivariate twin modelling was conducted by using customized R scripts

(Čolović, 2019) to explore the phenotypic associations between the RSQ and PDSQ scales for the best-fitting phenotypic models. Genetic and environmental influences on phenotypic similarities between MZ and DZ were examined for each RSQ scale and PDSQ anxiety scale by using structural equation modelling (SEM), including both univariate and multivariate biometric methods. Independent pathways (Figure 1) and common pathways (Figure 2) multivariate models (Rijsdijk & Sham, 2002) were applied to estimate additive genetic (A); shared environmental (C), and non-shared environmental factors (E); and specific (s) and common (c) genetic and environmental sources of variance.

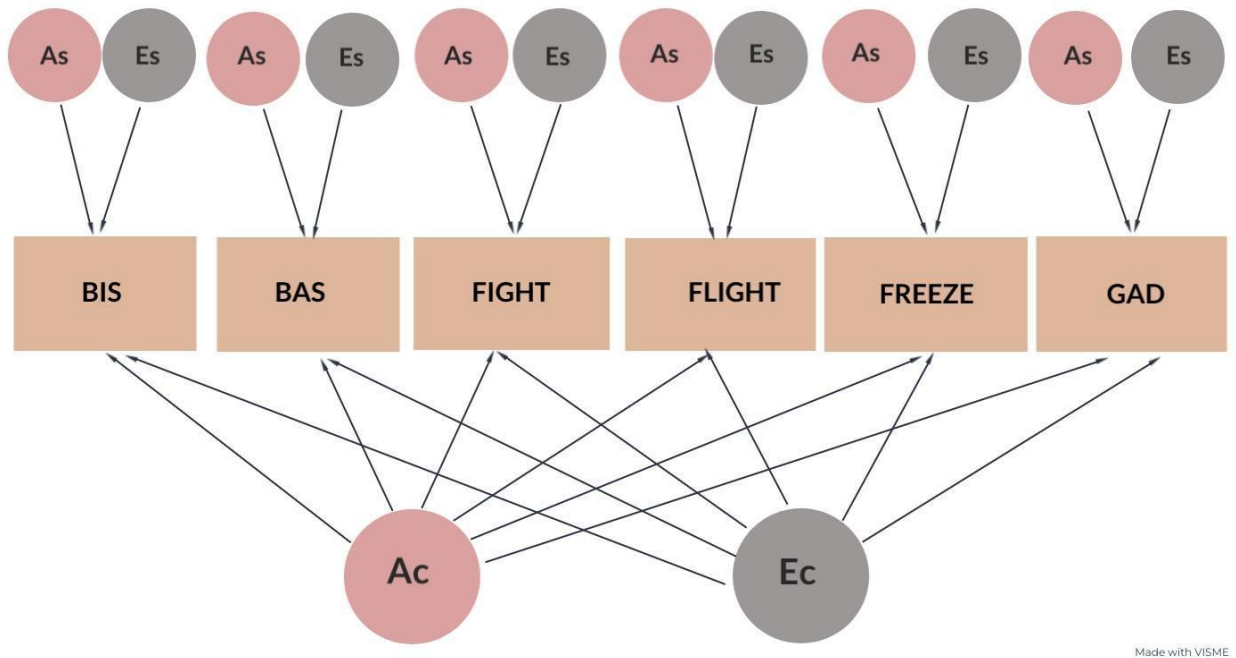


Figure 1. Independent pathway model – multivariate AE model for the RSQ dimensions and the PDSQ scale of generalized anxiety disorder (GAD).

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.

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 .95 and acceptable higher than .90; the Root Mean square Error of Approximation (RMSEA) with optimal values lower than .05 and acceptable lower than .08; the Standardized Root Mean Square Residual (SRMR) with an acceptable value below .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).

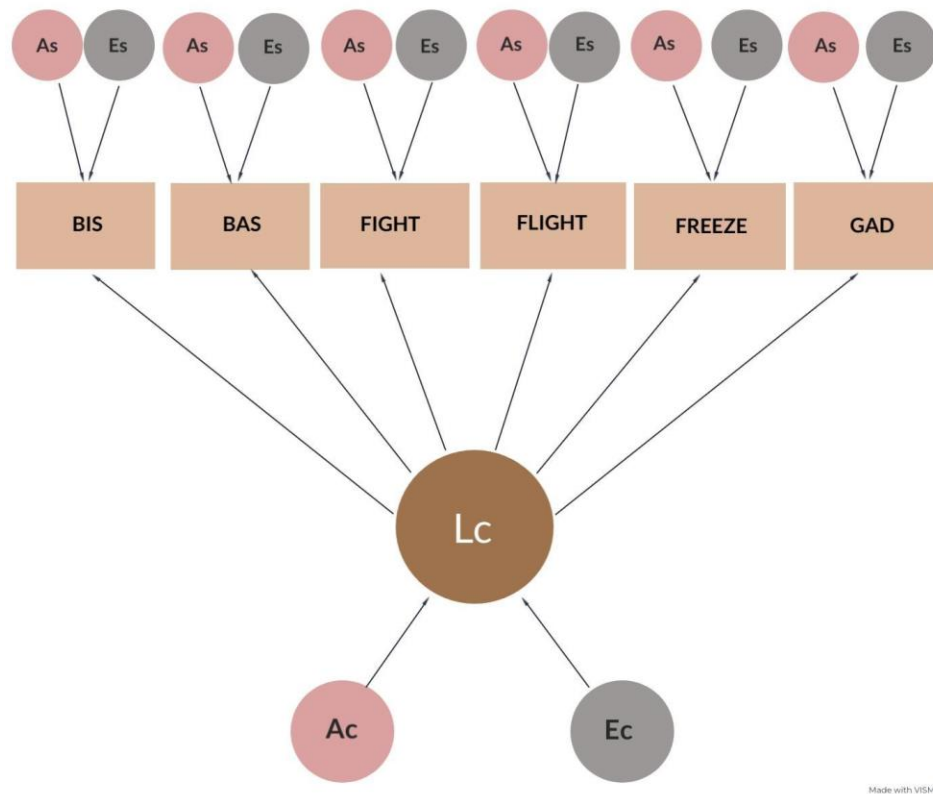


Figure 2. Common pathway model – multivariate AE model for the RSQ dimensions and the PDSQ scale of generalized anxiety disorder (GAD).

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.

Results

Table 1 shows the means and standard deviations of the RSQ traits as well as PDSQ anxiety scales for the monozygotic and dizygotic twins. The variables were normally distributed (acceptable values of skewness and kurtosis fall between -3 and $+3$: Brown, 2006), except for panic (in both twin groups) and agoraphobia (in monozygotic twins).

Table 1

Descriptive statistics of the RSQ traits and PDSQ anxiety scales

	<i>M</i>		<i>SD</i>		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.

Cross-twin correlations were calculated for MZ and DZ twins (Table 2). MZ twins show stronger correlations than DZ twins in all variables except for panic, agoraphobia and GAD, suggesting that additive genetic effects would be present, as well as that shared environmental effects might be significant for panic, agoraphobia and GAD. Moreover, significant cross-trait correlations between the RSQ personality traits and the PDSQ anxiety scales (Table 2) indicate there are basis for testing multivariate twin models.

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	.383**/.172*	-.325**	.000	.503**	.632**	.274**	.258**	.529**	.350**
BAS	-.325**	.459**/.239**	.310**	-.237**	-.248**	.018	.000	-.144**	-.014
Fight	.000	.310**	.333**/.316**	-.061	-.107**	.108**	.029	.016	.124**
Flight	.503**	-.237**	-.061	.404**/.276**	.515**	.180**	.163**	.309**	.185**
Freeze	.632**	-.248**	-.107**	.515**	.396**/.165*	.218**	.197**	.391**	.295**

Panic	.274**	.018	.108**	.180**	.218**	.191*/.309**	.431**	.445**	.491**
Agoraphobia	.258**	-.001	.029	.163**	.197**	.431**	.202**/.248**	.494**	.455**
Social Phobia	.529**	-.144**	.016	.309**	.391**	.445**	.494**	.465**/.245**	.543**
GAD	.350**	-.014	.124**	.185**	.295**	.491**	.455**	.543**	.262**/.294**

Note. Cross-twin correlations of monozygotic and dizygotic twin pairs are given in the table diagonal and are separated by / .

* $p < .05$; ** $p < .01$.

Multivariate genetic twin modelling

Univariate genetic twin modelling for all the RSQ personality traits and the PDSQ anxiety scales - panic, agoraphobia, social phobia, and GAD is given in Appendix A. Six different types of multivariate genetically informative models were tested for the RSQ personality traits and the PDSQ anxiety scales: panic, agoraphobia, social phobia, and GAD, respectively. Genetically informative models included 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 3). Overall, independent pathways models showed a better fit than common pathways models for all the multivariate models regarding the personality traits and the PDSQ anxiety scales: panic, agoraphobia, social phobia, and GAD (see Table 3).

For the panic model, the restricted 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 ($\Delta\chi^2(12) = 52.504, p < .05$; $\Delta\chi^2(12) = 66.750, p < .05$, respectively). When it comes to GAD, the independent restricted (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

Multivariate model fitting statistics of the RSQ traits and PDSQ anxiety scales

Panic	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA (90%CI)	SRMR
independent ACE ^a	183.418 (120)	.941	.935	11637	11871.62	.053 (.037–.068)	.076
common ACE	265.977 (129)	.873	.870	11701	11900.93	.076 (.063–.089)	.098
independent AE	220.454 (132)	.918	.918	11650	11837.66	.060 (.046–.074)	.092
common AE	277.929 (136)	.869	.873	11699	11871.47	.075 (.062–.088)	.099
independent CE	228.013 (132)	.911	.911	11657	11845.22	.063 (.049–.076)	.096
common CE	277.836 (136)	.869	.873	11699	11871.37	.075 (.062–.088)	.098

Agoraphobia	χ^2	CFI	TLI	AIC	BIC	RMSEA (90%CI)	SRMR
independent ACE ^a	185.313 (120)	.940	.934	11635	11870.20	.054 (.038–.069)	.070
common ACE	275.091 (129)	.866	.862	11707	11906.73	.078 (.065–.091)	.096
independent AE	236.498 (132)	.904	.904	11662	11850.39	.065 (.052–.079)	.093
common AE	282.856 (136)	.865	.869	11701	11873.08	.076 (.064–.089)	.097
independent CE	245.872 (132)	.895	.895	11672	11859.76	.068 (.055–.081)	.097
common CE	287.528 (136)	.861	.865	11705	11877.75	.078 (.065–.090)	.096
Social Phobia	χ^2	CFI	TLI	AIC	BIC	RMSEA (90%CI)	SRMR
independent ACE ^a	182.390 (120)	.951	.946	11447	11681.97	.053 (.037–.068)	.073
common ACE	269.879 (129)	.889	.887	11516	11716.21	.077 (.064–.090)	.096
independent AE	234.894 (132)	.919	.919	11476	11663.48	.065 (.051–.078)	.094
common AE	275.976 (136)	.890	.893	11509	11680.9	.074 (.062–.087)	.096
independent CE	249.140 (132)	.908	.908	11490	11677.73	.069 (.056–.082)	.102
common CE	287.962 (136)	.881	.884	11521	11692.88	.078 (.065–.090)	.095
GAD	χ^2	CFI	TLI	AIC	BIC	RMSEA (90%CI)	SRMR
independent ACE ^a	180.289 (120)	.947	.942	11581	11815.55	.052 (.036–.067)	.077

common ACE	284.253 (129)	.864	.861	11666	11866.27	.081 (.068–.093)	.100
independent AE	223.182 (132)	.920	.920	11600	11787.45	.061 (.047–.075)	.095
common AE	294.139 (136)	.861	.865	11662	11834.74	.079 (.067–.092)	.102
independent CE	232.974 (132)	.911	.911	11609	11797.24	.064 (.050–.078)	.099
common CE	295.721 (136)	.860	.864	11664	11836.32	.080 (.067–.092)	.100

Note. ACE – full common/independent factor common pathways model; AE, CE – reduced common/independent factor common pathways model.^a The best model fitting based on fit parameters and parsimony.

Table 4

Common and specific genetic and environmental effects on the traits in multivariate twin models

Trait	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	.18	.13	.41	.00	.04	.25	.18	.16	.66
BAS	.00	.21	.06	.18	.00	.55	.18	.21	.61
Fight	.16	.22	.00	.00	.00	.62	.16	.22	.62
Flight	.06	.10	.21	.21	.00	.42	.27	.10	.63
Freeze	.07	.12	.38	.03	.10	.29	.11	.22	.67
Panic	.14	.01	.01	.00	.14	.70	.14	.15	.71

	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	.17	.11	.43	.03	.04	.22	.20	.15	.65
BAS	.01	.19	.08	.18	.00	.54	.19	.19	.62
Fight	.12	.24	.00	.00	.00	.64	.12	.24	.64
Flight	.08	.10	.20	.20	.00	.42	.28	.10	.62
Freeze	.09	.13	.35	.02	.10	.31	.12	.22	.66
Agoraphobia	.23	.03	.00	.00	.02	.72	.23	.05	.72
	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	.32	.07	.36	.00	.00	.25	.32	.07	.61
BAS	.01	.22	.04	.17	.00	.56	.18	.22	.60
Fight	.06	.30	.00	.00	.00	.64	.06	.30	.64
Flight	.09	.06	.22	.22	.00	.41	.31	.06	.63
Freeze	.10	.07	.39	.03	.12	.29	.13	.19	.68
Social Phobia	.31	.02	.07	.12	.00	.48	.43	.02	.55
	Ac	Cc	Ec	As	Cs	Es	ΣA	ΣC	ΣE
BIS	.25	.09	.38	.00	.01	.28	.25	.10	.65
BAS	.35	.01	.00	.09	.02	.53	.44	.03	.53

Fight	.15	.15	.01	.00	.05	.64	.15	.20	.65
Flight	.15	.00	.22	.22	.00	.42	.37	.00	.63
Freeze	.17	.01	.41	.01	.12	.28	.18	.13	.69
GAD	.01	.15	.08	.00	.10	.66	.01	.25	.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.

Table 4 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, then fight and panic for the panic model, agoraphobia and fight for the agoraphobia model, social phobia and freeze for the social phobia model and BAS and freeze for the GAD model. The common shared environmental effects accounted for most of the variance of fight in all models, along with BAS and BIS for the panic model, BAS and freeze for the agoraphobia and social phobia model, and GAD and BIS for the GAD model. On the other hand, common environmental effects also accounted for most of the variance of BIS, along with freeze and flight. Moreover, specific additive genetic effects were the highest for flight in all models. Specific shared environmental effects were generally low, while specific nonshared environmental effects were the highest specific effects for all the variables.

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 5). There were significant genetic correlations between BIS and the anxiety scales of panic, agoraphobia, social phobia, and GAD. As for the environmental correlations, BIS was also significantly related to the anxiety scales (except agoraphobia). 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 5).

Table 5

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	.60** (.56, .64)	.09* (.02, .16)	.80** (.77, .82)	-.02 (-.09, .05)	.84** (.82, .86)	.28** (.21, .34)	.60** (.56, .64)	.21** (.14, .28)
BAS	-.08* (-.15, -.01)	.08* (.01, .15)	.01 (-.06, .08)	-.02 (-.09, .05)	-.25** (.18, .32)	-.10* (-.17, -.03)	-.05 (-.12, .02)	.03 (-.04, .10)

Fig ht	.22** (.15, .29)	.06 (-.01, .13)	.41** (.35, .47)	-.17** (-.24, -.10)	.19** (.12, .26)	-.11* (.04, .18)	.42** (.36, .48)	-.01 (-.08, .06)
Flig ht	.29** (.23, .35)	.11* (.04, .18)	.52** (.46, .57)	-.01 (-.08, .06)	.49** (.43, .54)	.18* (.11, .25)	.22** (.15, .29)	.12* (.05, .19)
Fre eze	.50** (.44, .55)	.05 (-.02, .12)	.64** (.59, .68)	.00 (-.07, .07)	.62** (.57, .66)	.20** (.13, .27)	.44** (.38, .5)	.18** (.11, .25)

Note. Confidence intervals are given in parentheses.

* $p < .05$; ** $p < .01$.

Discussion

This study examined common genetic and environmental influences that contribute to the co-occurrence of anxiety disorders and dimensions of the rRST. Drawing from the existing evidence that distinguishes anxiety from fear (Gray & McNaughton, 2000), as well as their respective psychopathological expressions (Barlow, 2002; Kruger, 1999; Watson, 2005), our focus was specifically directed towards discerning potential differences between fear-related and anxiety-related syndromes in terms of their underlying origins.

Consistent with the previous findings (Smederevac et al., 2022; Takashi et al., 2007), the results revealed that genetic factors (ranging from 37% for BIS to 44% for BAS) along with nonshared environmental influences account for individual differences in the rRST systems. However, psychopathological syndromes are more significantly shaped by the environment, with heritability estimates mostly not exceeding .30, except for social phobia. This result implies that the nonshared environmental effects seem crucial in shaping anxiety syndromes aligning with

previous findings that the environment largely shapes distinct manifestations of emotional disorders (Kendler et al., 1987; Hetema et al., 2001). Moreover, although small to moderate magnitude, the genetic factors underlying individual differences in psychopathological dimensions are, in most cases, fully shared with the RSQ dimensions, implying a common genetic basis of anxiety disorders and these personality systems. This is further supported by the patterns of genetic correlations between anxiety disorders and personality traits, which generally mirror the patterns of phenotypic correlations. The apparent lower environmental correlations suggested that shared genetics primarily underlie the phenotypic associations.

The genetic correlations suggest that a particular configuration of partially inherited traits, which primarily includes highly sensitive BIS followed by pronounced sensitivity of Freeze, Flight, but also Fight system in the case of most psychopathology dimensions, contributes to a vulnerability to the development of anxiety syndromes. Starting from the standpoint of the triple vulnerability hypothesis (Barlow, 2000) that posits certain heritable traits as the prerequisites for developing mental disorders, these dimensions can be seen as aspects that jointly contribute to the general biological vulnerability. As the responses to aversive stimuli encompassed by these dimensions are often subsumed under neuroticism or negative affectivity in other personality models (Barlow et al., 2014), the findings support the view of negative affectivity as a general biological vulnerability for anxiety disorders (Brown & Naragon-Gainey, 2013). However, considering a broader range of general genetic factors could potentially provide insights into patterns of comorbidity that a single genetic basis, such as high negative affectivity, might not explain.

Given the involvement of all three fear systems in the biological vulnerability for anxiety disorders, a conflict between different available responses in a situation perceived as dangerous

could arise, potentially building on the already existing sense of threat and subjectively leading to overwhelming fear or intense anxiety. However, it is more plausible that the highly sensitive Fight/Flight/Freeze system generates diverse responses to a threat, depending on its perceived proximity.

Apart from the so-called “avoidant” dimensions, the Fight system also shows mild to moderate genetic correlations with all psychopathological dimensions, 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.

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 & Corr, 2018). Given that the BIS is responsible for scanning the environment and detecting conflicting goals (Gray & 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. Not only can it imply a more frequent detection of a threat, but it may also influence the perception of danger proximity or defensive distance (Blanchard et al., 2001), triggering intense fear and panic responses even in situations lacking objective threat. Furthermore, an individual with a highly sensitive BIS, drawing from previous experiences, may perceive their coping resources as inadequate, thereby amplifying sense of defenselessness.

However, the result that there is a significantly greater genetic association of Flight with agoraphobia and social phobia than with GAD and panic implies possible differences in the

activation of avoidance systems that explain the specific behaviors associated with fear and anxiety. The findings pertaining to social phobia symptoms seem to stand out from other results. Not only social phobia displays a more pronounced genetic basis compared to other psychopathological dimensions, but its genetic foundation was somewhat unique and independent of those of personality traits. Previous findings suggested a possible role of specific genotypes within the serotonergic pathway gene 5-HT2A T102C in the development of social phobia, a link not observed with personality traits phenotypically associated with social phobia (Lochner et al., 2007). However, further molecular genetic research is needed to explore genes that contribute specifically to social phobia. Additionally, social phobia is the only psychopathology dimension that is, mildly but substantially, negatively genetically linked to BAS, which points to the role of partially heritable lower sensitivity to rewarding stimuli signals and overall approach behavior in the etiology of this syndrome. Since BAS is often associated with extraversion (Smillie, 2013), this indirectly supports prior findings of a phenotypic connection between low extraversion and social phobia (Bienvenu et al., 2004).

In contrast, in agoraphobia there is a general excitation of all relevant systems, including Fight. Previous research pointed towards a phenotypic connection of agoraphobia with low trust and high anger/hostility (Bienvenu et al., 2004), traits sharing similarities with the Fight system. According to rRST, the Fight system is associated with fear and is activated when a perceived threat is immediate and close (Gray & McNaughton, 2000). Since the perception of a threat, as well as the assessment of its proximity and possibility of avoiding it, plays a crucial role in the propensity to activate an aggressive response, an overly sensitive Fight system could result in biased cognitive processing of stimuli. Thus, the genetically predisposed tendency to activate an aggressive response to a threat may be linked to patterns of perceiving the physical and social

environment as threatening and hostile, potentially contributing to the dynamics of the anxiety disorders examined here. For example, this perception of hostility could trigger avoidance behavior and contribute, along with other mechanisms, to the development of agoraphobia. The existence of genetic linkage between agoraphobia and the Fight, even in the absence of their phenotypic correlation, gives rise to the possibility of hypothesizing a genetic predisposition for aggressiveness that holds significance for this syndrome. Likely due to some environmental influences, this inclination might be confronted with challenges in finding suitable outlets for its expression.

Similar mechanisms regulate GAD, determined dominantly by BIS, Freeze and Fight. However, the biometric model points out that a common additive genetic component is determined by BAS and GAD does not share genetic variance with it. Instead, it is significantly explained by a common shared, unique shared and non-shared environment. This result could not be ignored, as it is unusual for this type of research, in which the variance is typically shared by additive genetic and nonshared environmental variance. The shared environment shapes the atmosphere common to the whole family and it is possible that GAD develops in conditions suitable for the manifestation of alertness and the perception of potential threats.

Besides BIS, panic is dominantly determined by the Freeze system, which contributes to the inhibition of cognitive processes in new situations. Although this experience of inadequate response is common to all anxiety phenomena, it probably forms the backbone of panic. For example, according to modern learning theorists, both emotions, fear (panic) and anxiety, are important in the development of panic disorder and are present during initial panic attacks (Bouton et al., 2001), which are rather common in the general population but do not necessarily

lead to the disorder. High trait anxiety often creates the context from which the initial panic attacks grow (Barlow, 2002; Bouton et al., 2001). A similar account might apply to other anxiety disorders as well. It is interesting that panic has a unique shared environment with other traits, which indicates the importance of the environment for the development of these symptoms. Although the type of environment can only be speculated, it is plausible that restrictive rules may contribute to the underdevelopment of the capacity for active cognitive processing in new or threatening situations.

Given the previously demonstrated phenotypic and genetic connections between various forms of avoidant behavior (Smederevac et al., 2022), the observed similar patterns of association between all four anxiety syndromes and the rRST traits were not unexpected. However, the magnitudes of individual correlations may raise some 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 (Corr & McNaughton, 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). Furthermore, the Freeze system displayed stronger genetic ties to social phobia and agoraphobia compared to panic, underscoring the intricate interplay between different forms of avoidant behavior.

Environmental factors emerged as significant contributors to the development of anxiety syndromes. While some shared environmental influences are present, the impact of the nonshared environment is dominant. Unlike genetic factors, which psychopathological traits

mostly share with the RSQ dimensions, environmental influences, especially nonshared, are primarily unique to individual disorders. Also, environmental correlations between psychopathology dimensions and rRST traits are generally modest and often notably weaker than their genetic counterparts. According to the triple vulnerability model for anxiety disorders (Barlow, 2000), a general biological vulnerability is important for any anxiety disorder to arise. Negative affectivity is most often considered to be a temperamental manifestation of this common genetic basis (Brown & Naragon-Gainey, 2013), a notion roughly supported by the findings of our study. 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, in press). 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.

Overall findings suggest that various personality traits related to responses to aversive stimuli play a role in forming a vulnerability to the development of anxiety symptoms. The findings demonstrated a complex interplay of genetic and environmental factors in shaping individual differences in both the rRST dimensions and psychopathological traits, which blurs the distinction between anxiety-related and fear-related disorders. The genetic association of the rRST system and anxiety disorders points to pleiotropy and further highlights the importance of unique environmental factors as pivotal to the development of disorders. This underscores the necessity of concentrating research efforts on unique and potentially atypical environmental

influences that elicit anxiety symptoms while leaving personality traits unaffected. Such a focus is essential for unraveling the origins of anxiety symptoms.

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). However, there is a possibility that the results would not be completely replicated in larger samples. 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. Third, the PDSQ was developed before the new classifications of mental disorders and may not adequately separate anxiety and fear symptoms.

Nevertheless, the results of the study seem to hold important implications for considering the etiology of symptoms of mental disorders. Namely, the result that the symptoms of mental disorders, except social phobia, do not have a specific genetic variance, but rather share it with personality traits is extremely important, implying that they are common variations of individual differences. Although a clear demarcation of anxiety and fear has not been demonstrated, there is compelling evidence for differential activation of the rRST systems in the examined dimensions of mental disorders. The results support the DSM-5 (APA, 2013) in which GAD, phobias, and panic are classified into the same group of disorders, called anxiety disorders. Namely, BIS, as a hierarchically superior system, which corresponds with anxiety, has a dominant role in all symptoms, explaining the comorbidity between most of them. It can also be seen as a common

factor contributing to all disorders, supporting the Integrative Hierarchical model (Kotov et al., 2007; Mineka et al., 1998). Freeze, which should be associated with panic, also plays an important role in all symptoms, indicating that cognitive blockage prevents the processing of new stimuli, experienced as threatening. However, the results indicate that more specific factors are present in social phobia and agoraphobia. Namely, Flight can be associated to a greater extent with phobias, as expected. Also, the negative genetic correlation of BAS with social phobia corresponds to assumptions about low positive affect, which is specific to this type of symptoms (Watson, 2009). Generally, the roles of Fight and BAS have been shown to be important in spite of approach behavior being often neglected in the research of anxiety disorders. Although GAD, social phobia, agoraphobia, and panic share a similar genetic basis with personality traits, there are specificities that mostly arise from environmental factors. In other words, there is no simple isomorphism between one personality systems and the psychopathological syndromes. The learning process during which skills to overcome threatening stimuli are developed greatly contributes to adaptation in shared and non-shared environments.

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Supplementary material

Table A

Univariate model fitting statistics of the RSQ traits and PDSQ anxiety scales

BIS	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	0.959 (4)	1	1	2066	2089.56	.000 (.000–.041)	.035
ACE	0.959 (3)	1	1	2068	2095.47	.000 (.000–.076)	.035

ADE	0.959 (3)	1	1	2068	2095.47	.000 (.000–.076)	.035
CE	5.002 (4)	.967	.983	2070	2093.60	.037 (.000–.122)	.045
BAS	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	0.737 (4)	1	1	2057	2080.27	.000 (.000–.014)	.028
ACE	0.614 (3)	1	1	2059	2086.06	.000 (.000–.056)	.029
ADE	0.737 (3)	1	1	2059	2086.18	.000 (.000–.065)	.028
CE	5.580 (4)	.968	.984	2062	2085.11	.046 (.000–.128)	.060
Fight	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	4.762 (4)	.981	.990	2070	2093.64	.032 (.000–.119)	.045
ACE	0.148 (3)	1	1	2068	2094.95	.000 (.000–.000)	.012
ADE	4.762 (3)	.956	.971	2072	2099.56	.056 (.000–.147)	.045
CE	0.162 (4)	1	1	2066	2089.04	.000 (.000–.000)	.012
Flight	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	4.422 (4)	.989	.995	2063	2086.76	.024 (.000–.116)	.077
ACE	4.134 (3)	.971	.981	2065	2092.39	.045 (.000–.139)	.073
ADE	4.422 (3)	.964	.976	2065	2092.68	.051 (.000–.143)	.077
CE	7.695 (4)	.906	.953	2067	2090.04	.071 (.000–.145)	.070
Freeze	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	3.283 (4)	1	1	2073	2096.50	.000 (.000–.102)	.063
ACE	3.050 (3)	.998	.999	2075	2102.18	.009 (.000–.125)	.060
ADE	3.283 (3)	.991	.994	2075	2102.41	.023 (.000–.128)	.063
CE	5.843 (4)	.944	.972	2076	2099.06	.050 (.000–.130)	.061
Panic	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	15.472 (4)	.577	.788	2088	2111.65	.124 (.063–.193)	.109
ACE	8.111 (3)	.812	.874	2083	2110.21	.096 (.014–.178)	.090
ADE	15.472 (3)	.540	.693	2090	2117.57	.150 (.082–.227)	.109
CE	8.111 (4)	.848	.924	2081	2104.29	.074 (.000–.149)	.090
Agoraphobia	χ^2	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	7.253 (4)	.808	.904	2092	2115.58	.066 (.000–.142)	.082
ACE	4.636 (3)	.904	.936	2092	2118.88	.054 (.000–.145)	.071
ADE	7.253 (3)	.749	.833	2094	2121.50	.087 (.000–.171)	.082
CE	4.636 (4)	.963	.981	2090	2112.97	.029 (.000–.118)	.071
Social Phobia	χ^2	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	1.310 (4)	1	1	2055	2078.64	.000 (.000–.059)	.035
ACE	1.309 (3)	1	1	2057	2084.55	.000 (.000–.089)	.035
ADE	1.310 (3)	1	1	2057	2084.56	.000 (.000–.089)	.035
CE	8.164 (4)	.918	.959	2062	2085.49	.075 (.000–.149)	.064
GAD	χ^2	CFI	TLI	AIC	BIC	RMSEA	SRMR
AE	6.353 (4)	.914	.957	2084	2107.79	.056 (.000–.135)	.062
ACE	1.033 (3)	1	1	2081	2108.39	.000 (.000–.079)	.034
ADE	6.353 (3)	.878	.918	2086	2113.71	.078 (.000–.163)	.062
CE	1.033 (4)	1	1	2079	2102.47	.000 (.000–.046)	.034

Note. ACE – full additive genetic, shared environmental and nonshared environmental model;
ADE – full additive genetic, nonadditive genetic and nonshared environmental model; AE, CE –
reduced additive genetic/shared environmental and nonshared environmental models.