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A Developmental Twin Study of Emotion Recognition and Its Negative Affective Clinical Correlates

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Abstract

Objective.—Youth with psychiatric disorders distinguished by irritability, including depression and associated trait neuroticism, show deficits in the ability to recognize facial expressions of emotion, particularly happiness. However, the contribution of genetic and environmental factors to this ability remains unknown. The current study examined this trait in twins to assess the genetic and environmental influences on face-emotion recognition abilities and their association with irritability, neuroticism, and depression.

Method.—Child and adolescent twins (N = 957 from 496 families), aged 9 to 17 rated their irritability (on the Affective Reactivity Index), neuroticism (on the Junior Eysenck Personality Questionnaire), and depression (on the Short Mood and Feelings Questionnaire) and completed a face-emotion labeling task. Faces depicting anger, disgust, fear, happiness, sadness, and surprise were morphed with a neutral face yielding 10 levels of increasing emotional expressivity. Biometrical twin analyses evaluated contributions of genetic and environmental factors to the etiology of face-emotion recognition and its association with irritability, neuroticism, and depression.

Results.—Recognition of each emotion was heritable; common and specific sets of genetic factors influenced all emotions and individual emotions, respectively. Irritability, neuroticism, and depression are modestly, negatively correlated with emotion recognition, particularly the

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The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. The material contained in this article has not been published elsewhere nor is it under consideration elsewhere. Two prior papers, which described the child and adolescent samples that comprise the present study, respectively, are cited in the text. Additionally, one prior paper (Cecilione et al., 2017) reported on the test-retest reliability data, which are described here using an alternative data analytic approach. This is described in the text. Preliminary research findings were presented at the 2017 annual meeting of the Anxiety and Depression Association of America (ADAA) in San Francisco, CA.

recognition of happiness. For irritability and neuroticism, this correlation appears largely due to genetic factors.

Conclusions.—This study maps genetic and environmental contributions to face-emotion recognition and its association with irritability, neuroticism, and depression. Findings implicate common genetic factors in deficits regarding the recognition of happiness associated with irritability and neuroticism in childhood and adolescence.

Keywords

irritability; face-emotion recognition; child; adolescent; twin; genetics

Introduction

Recognizing facial emotions is a fundamental human capability¹. Emotions can be easily recognized by people from many different cultures. This may reflect the central role of face-emotion expression in social communication^{2,3} and the fact that face-emotion recognition is mediated by a core, evolutionarily conserved neural circuit⁴. Psychiatric problems often involve social communication deficits. For example, major depressive disorder⁵, bipolar disorder^{6,7}, autism spectrum disorders⁸, alcohol dependence⁹, attention-deficit/hyperactivity disorder¹⁰, and disruptive mood dysregulation disorder (DMDD), in which irritability¹¹ and elevated neuroticism are common.

Irritable mood, a common complaint amongst youth, is transdiagnostic^{11,12} and a core feature of several emotional disorders, including disruptive mood dysregulation disorder (DMDD)^{13,14}. Youth with chronic, severe irritability (similar to the DMDD phenotype) exhibit deficits in face-emotion recognition^{12,15} and neural perturbations when processing emotional faces^{16,17}, which suggests one aspect of diminished socioemotional functioning. Additionally, a recent meta-analysis implicates deficits in face-emotion recognition, specifically impaired recognition of happiness, in adult major depressive disorder⁵. This is consistent with existing cognitive theory¹⁸ and evidence of impaired recognition of happiness among adults who report high trait neuroticism¹⁹. As an initial investigation, the present study examined the role of common genetic contributions to explain theory and phenotypic evidence of face-emotion recognition deficits in individuals with elevated irritability, depression, and neuroticism severity.

However, potential pathways linking the etiologic underpinnings of the ability to recognize emotional expression in faces and childhood liability to irritability, neuroticism, and depression have not been investigated. Identifying the degree of overlap in shared genetic and environmental contributions to irritability, neuroticism, and depression and face-emotion recognition is necessary to locate diminished socioemotional functioning as a possible process in the development of irritability and internalizing disorders (e.g., depressive and anxiety disorders). To our knowledge, only one prior study has parsed genetic and environmental contributions to face-emotion recognition abilities²⁰, and none consider the degree to which these contributions make shared or independent contributions to psychopathology. The current study addresses the need for such data.

Specifically, the primary aims of the current study are to estimate, in childhood and adolescence: i) the phenotypic correlation (i.e., within-subject association) of irritability, neuroticism, and depression with face-emotion recognition; ii) the contribution of genetic and environmental influences to face-emotion recognition; iii) the structure of these genetic and environmental influences across emotions; and iv) the degree to which the genetic and environmental contributions to face-emotion recognition are correlated with those for irritability, neuroticism, and depression. To achieve these aims, a large, genetically-informed epidemiological sample of child and adolescent twins, with known genetic concordance based on zygosity, self-reported on irritability, neuroticism, and depression and completed a face-emotion labeling task (FELT). Three standard multivariate twin models²¹, similar to those examined previously²⁰, were compared to optimally model genetic and environmental factors in the etiology of face-emotion recognition. Irritability, neuroticism, and depression were then separately added to the best fitting model to examine the genetic and environmental contributions to their covariance with face-emotion recognition.

Methods and Materials

Participants

Child and adolescent participants (N = 1,279, 54.59 % female) were recruited as pairs of twins aged 9 to 20 from the mid-Atlantic area of the United States of America by the Mid Atlantic Twin Registry²². Detailed demographic information is provided in two published overviews of the child²³ and adolescent samples²⁴. The child sample exclusively recruited Caucasian twins; the adolescent sample primarily recruited Caucasian twins. This was done to enhance power for planned subsequent genomic research by reducing genetic heterogeneity due to population stratification²⁵. All twins reported on irritability, neuroticism, and depression and completed the face-emotion labeling task (see below). To minimize the impact of potential confounding factors, participants were excluded from recruitment if they were intellectually disabled, had a prior diagnosis of an autism spectrum disorder, experienced a past or current psychotic episode, were currently using anxiolytic or antidepressant medication, or had been diagnosed with any medical condition that might have adversely impacted participants' safety or ability to complete the study (e.g., unexplained seizures) including aspects of the study not described here^{23,24}, such as hypersensitivity to a carbon dioxide challenge task^{26,27}. To focus on face-emotion recognition during childhood and adolescence, the present study analyzed data on participants under 18 years of age (N = 957).

Rates of psychiatric diagnoses were assessed for all participants based on DSM-IV. Children were assessed by structured K-SADS assessment via parent interview²⁸ by a study psychiatrist or clinical psychologist. Adolescents were assessed by participant-report of psychiatric symptoms on a modified version of the Composite International Diagnostic Interview – Short Form^{26,29}. Diagnostic rates, based on DSM-IV, indicate that the sample is generally representative of children and adolescents in this age range. Lifetime prevalences for any depressive (2.2%) or anxiety disorder (27.4%) among children are consistent with rates reported elsewhere^{30,31}; lifetime prevalences for major depressive disorder (14.8%), panic disorder (1.2%), and generalized anxiety disorder (2.6%) among adolescents are

consistent with rates reported elsewhere³². Participants were recruited at two sites: Virginia Commonwealth University (VCU; Richmond, VA) and the National Institute of Mental Health (NIMH; Bethesda, MD), which included only child participants.

Procedure

Participants completed the FELT in the context of two larger studies on the heritability of potential endophenotypes of internalizing disorders in children or adolescents and young adults. A parent or legal guardian provided informed consent; youth provided assent before completing self-report questionnaires and laboratory-based tasks^{23,24}.

To estimate test-retest reliability of the face-emotion recognition task, child participants attended a second session two weeks after the first. A planned missingness design minimized participant burden; 157 randomly-chosen participants completed the FELT task at follow-up. The protocol was approved by the Institutional Review Boards of VCU and the NIMH.

Materials

Twin Zygosity.—A parent or guardian answered questions regarding physical similarities of the twins (e.g., “Are your twin children often confused for one another?”), such as those described by Jackson and colleagues³³. Prior research demonstrated that these questions assess twin zygosity with high agreement to blood³⁴ and DNA assessments³³ of zygosity including high agreement ($\kappa = 0.95$) with zygosity estimated from single nucleotide polymorphisms (SNPs) in the present sample²⁴.

Clinical Correlates.—Participants reported irritability over the past six months on the Affective Reactivity Index (ARI), which assesses behavioral and emotional components of irritability and anger³⁵. Participants completed neuroticism items from the Junior Eysenck Personality Questionnaire³⁶ and the Short Mood and Feelings Questionnaire³⁷ to assess neuroticism and recent depression symptoms, respectively. Prior research demonstrated reliability and validity of these measures in child and adolescent samples^{35–37} including the present study^{23,24}.

Face-Emotion Labeling Task.—Participants completed a FELT in which they were presented with six fundamental emotions (i.e., happiness, anger, sadness, fear, disgust, and surprise) expressed by Caucasian adults (50% female) from the well-validated Pictures of Facial Affect Series³⁸. The task was based on prior research on the clinical and neurobiological correlates of face-emotion processing^{39–41}.

A photo portraying each emotion was morphed with a neutral face of the same person to produce 10 target images along a gradient from 10% to 100% emotional expression to yield 60 images (6 emotions $\times$ 10 morph steps). Each image was presented at 6 trials for a total of 360 trials. Trial order was randomized but consistent across participants. At each presentation, participants viewed a fixation cross for 250 ms before viewing the target image for 500 ms. Participants then pressed a button to endorse which emotion they saw from the

six possible emotions. Task performance demonstrates high test-retest reliability among adult⁴² and child samples⁴³. The task took approximately fifteen minutes to complete.

Data Analysis

Raw accuracy may reflect a participant's tendency to endorse a specific emotion. To adjust for this association, participant scores were transformed as in Marsh et al.⁴⁰ and Wagner⁴⁴. A participant's score was computed as the product of raw accuracy and differential accuracy (i.e., the proportion of trials in which an emotion is correctly selected). The result was adjusted for guessing by subtracting 1/6 and arcsine transformed to improve the normality of the distribution of scores. Similar to prior research using this task, each participant's performance for each emotion was computed as the average score over all trials. Data from 94 individuals (9.82% of the sample) were removed due to suspected poor compliance with the task, (e.g. pressing the same button throughout the task without looking at the screen), or suspected difficulty attending to the task (e.g., self-reported headache). Research assistants who noted poor compliance were blind to other study data. In total, 189 monozygotic twin pairs, 271 dizygotic twin pairs, and 36 twin pairs of indeterminate zygosity completed the face-emotion labeling task.

Our main analyses proceeded in several steps. First, we verified the test-retest reliability of average performance on the FELT. Second, we examined the phenotypic correlations of face-emotion recognition with irritability, neuroticism, and depression. Third, we investigated the structure of genetic and environmental contributions to the recognition of all six emotions. Finally, the role of face-emotion recognition as an affective endophenotype was examined by estimating the degree of genetic and environmental correlations of face-emotion recognition with irritability, depression, and neuroticism. As participants are nested within families, test-retest reliability, phenotypic correlations, heritability, and genotypic correlations were estimated based on the biometrical model, which incorporates known nonindependence due to shared genetic and environmental influences that contribute to the correlation between mono- and dizygotic twins²¹.

Three multivariate models were fitted to face-emotion recognition data: a common pathway model (CPM); an independent pathway model (IPM); and a "saturated" model, based on a Cholesky decomposition of the variance-covariance matrix. The latter allows for six sets of three factors that account for different structures of additive genetic (A), shared (between twins) environmental (C), and nonshared environmental (E) factors to influence the recognition of each emotion. The CPM and IPM are nested within the Cholesky decomposition to evaluate whether separate A, C, and E factors directly influence the measures (i.e., the independent pathways model), or covariance in the etiology of performance between emotions is best summarized by a single, latent common factor (i.e., CPM). Performance on the face emotion recognition task was standardized prior to analyses. Analyses of face-emotion recognition were repeated adjusting for the impact of age and sex on mean performance; results regarding the optimal model, structure of the model (see Figure 1), and estimated heritability were unchanged. An additional analysis examined potential differences in the contribution of genetic and environmental factors by participant sex. While female participants demonstrated higher face-emotion recognition across all six

emotions (see Supplemental Table 1), multivariate sex limitation models⁴⁵ demonstrated equivalent genetic and environmental contributions to face-emotion recognition across sex (see Supplemental Table 2).

Once a suitable, best-fitting model for the etiology of face-emotion recognition was identified, irritability, neuroticism, and depression were separately added to examine shared etiology with face-emotion recognition. Preliminary linear mixed effects models, which adjust for nesting of twins within family, estimated the influence of age and sex on irritability, neuroticism, depression, and face-emotion recognition (see Supplemental Table 1). Older age was associated with reduced irritability, neuroticism, and depression but improved face-emotion recognition across all emotions. The present research may clarify mechanisms underlying age-related changes in irritability, neuroticism, depression, and face-emotion recognition. For example, age-related changes in irritability and face-emotion recognition may co-occur due to common developmental pathways and due to shared genetic risk. Age and sex also accounted for a substantial proportion of variance in face-emotion recognition (conditional R^2 range: 0.33 – 0.43; see Supplemental Table 1). Therefore, adjusting for age and sex may statistically underestimate the correlation of face-emotion recognition with irritability, neuroticism, and depression.

Parameters from each structural equation model were iteratively fixed to zero to evaluate the statistical significance of additive genetic (A) and shared environmental (C) factors to face-emotion recognition, irritability, neuroticism, and depression and the covariance between them. Profile-based confidence intervals were computed using maximum likelihood estimation within OpenMx. Analyses were conducted in R version 3.2.3⁴⁶ using the following packages: psych⁴⁷, car⁴⁸, plyr⁴⁹, ggplot2⁵⁰, nlme⁵¹, umx⁵², and OpenMx⁵³.

Results

Descriptive Statistics and Face-Emotion Recognition Deficits

Table 1 lists descriptive data on the FELT outcomes. Test-retest reliability for performance on the FELT is high for each emotion and similar across emotions as evidenced by overlapping confidence intervals for reliability estimates. Performance in recognizing each emotion is correlated across emotions. However, as also shown in Table 1, mean performance for each emotion differs significantly, reflecting greater accuracy to identify certain emotions (e.g. happiness) over others (e.g. disgust).

The correlation of irritability, neuroticism, and depression with each emotion was estimated within the biometrical model to account for nonindependence between twins (see Data Analysis, above). Elevated depression, neuroticism, and irritability are associated with reduced recognition of facial expression of happiness (see Table 1), which appears to be linear upon investigation of bivariate scatterplots (see Supplemental Figure 1). Additionally, participants who reported elevated irritability and neuroticism demonstrate reduced recognition of sadness and fear. Elevated neuroticism is, more broadly, also associated with reduced recognition of surprise and disgust.

As noted in previous literature²⁰, a common factor may usefully describe overall face-emotion recognition ability. The phenotypic correlation of irritability, neuroticism, and depression with overall face-emotion recognition ability was estimated within a common factor reflecting all six emotions, which is identical to the CPM (see Supplemental Table 3, Common Factor Model, for factor loadings). Irritability, $r = -0.10$, $p = 0.007$, and neuroticism, $r = -0.13$, $p = 0.0007$, though not depression, $r = -0.05$, $p = 0.23$, were modestly, negatively correlated with overall face-emotion recognition.

Etiology of Face-emotion Recognition

Evaluation of the three multivariate twin models for each of the six emotions differentiated potential etiological contributors to emotion-recognition ability. The comparison of model fits for the Cholesky vs. the CPM and IPM indicated that the Cholesky model fit the data better than either of the other two models (Table 2). This suggests that several additive genetic and environmental factors explain etiology more comprehensively than does a single common factor. Specifically, the parameter estimates for the three models demonstrated a high level of consistency between the factor loadings for the CPM, the additive genetic factor loadings for the IPM, and the first latent factor for the Cholesky decomposition (see Figure 1, Supplemental Table 3). The CPM and IPM, however, did not account for additional genetic components for emotion recognition, particularly for happiness and disgust, nor the residual genetic correlation between fear and surprise. Moreover, the factor loading results for both the CPM and the IPM diverged substantially from the results for the best fitting model regarding shared and unique environmental influences.

Genetic and environmental contributors to face-emotion recognition were ascertained from the best-fitting model based on the Cholesky decomposition. Strong evidence arose for familial aggregation of emotion recognition ability (i.e., cannot drop both A and C sources of familial aggregation, Table 3), with additional evidence suggesting that similarity among twins' performance predominantly reflected genetic factors (Table 4).

For specific emotions, consistent evidence implicated genetic factors with similarly strong influences among emotions as indicated by overlapping confidence intervals (Table 4). Significance testing in nested-model comparisons generated little evidence of shared environmental factors influences (Tables 3 and 4), although these comparisons may be underpowered for tests with larger degrees of freedom⁵⁴.

Shared Etiology of Face-emotion Recognition with Irritability, Neuroticism, and Depression

The phenotypic correlation of irritability, neuroticism, and depression with recognition of happiness appears largely due to common genetic factors. Genetic contributions to irritability, λ (factor loading) = -0.54 , 95% CI [-0.66 , -0.02], $p = 0.04$, and neuroticism, λ = -0.58 , 95% CI [-0.72 , -0.11], $p = 0.01$, loaded significantly onto the residual genetic factor for the recognition of happiness.^A Notably, the loading of the genetic contribution to depression onto the factor for the recognition of happiness was similar but did not quite

^AResidual genetic variance for irritability, neuroticism, and depression is sufficiently small that genetic correlations of each with the genetic factor specific to the recognition of happiness approach -1 . For this reason, the standardized loading is presented to index each association distinct from the amount of residual variance, which obscures the genetic correlation.

reach statistical significance, $\lambda = -0.58$, $p = 0.09$. There is no evidence of additional associations of genetic contributions to irritability, neuroticism, or depression with other genetic factors underlying face-emotion recognition, such as A1, A3, and A4 (see Figure 1). These correlations could be constrained to zero without appreciable loss of model fit for irritability, $\chi^2(5) = 2.50$, $p = 0.78$, neuroticism, $\chi^2(5) = 2.07$, $p = 0.84$, and depression, $\chi^2(5) = 3.15$, $p = 0.68$.

Discussion

The present study used a genetically-informed epidemiological sample of child and adolescent twins to examine the contribution of genetic and environmental factors to face-emotion recognition and its association with irritability, neuroticism, and depression. After demonstrating strong test-retest reliability for the FELT task, the study demonstrated the heritability of both a common emotion recognition ability and specific abilities to recognize happiness, disgust, and fear/surprise. The present study replicated and extended prior findings linking childhood irritability^{12,55} and adult neuroticism¹⁹ and depression⁵ with impaired face-emotion recognition, specifically impaired recognition of happiness. Finally, the present study provides the first exploration of the contribution of common genetic factors to these face-emotion recognition deficits.

The study replicates the only prior twin study of emotion recognition, which found a large additive genetic contribution (i.e., 75%)²⁰. This prior study attributed the ability to recognize each emotion to a single latent common factor, whereas the present, considerably larger study found that additional additive genetic factors for specific emotions augment this common factor¹⁸. The current study then extends prior research by leveraging the structure of genetic factors to clarify the contribution of shared genetic factors to the phenotypic association of negative affect with impaired face-emotion recognition.

Impaired face-emotion recognition has been implicated in many psychiatric conditions in children, adolescents, and adults including major depressive disorder⁵, bipolar disorder^{6,7}, autism spectrum disorders⁸, alcohol dependence⁹, and attention-deficit/hyperactivity disorder¹⁰, several of which are characterized by irritability¹¹ and neuroticism¹⁹. As hypothesized¹⁸, elevated depression symptoms were associated with a specific deficit in recognizing happiness. Additionally, irritability and neuroticism showed deficits in recognizing a broader range of emotions. Along with similar genetic associations of irritability, neuroticism, and depression with the recognition of happiness, this suggests that face-emotion recognition deficits may clarify points of convergence and divergence in the socioemotional functioning of multiple psychiatric disorders. Given evidence of common genetic influences to emotion recognition, irritability, and neuroticism, further research is needed to clarify the role of common genetic influences to the association of face-emotion recognition with the range of psychiatric conditions in children, adolescents, and adults. With further research, evidence of common genetic influences to face-emotion recognition deficits within multiple disorders might clarify a socioemotional pathway in the development of multiple comorbid psychiatric disorders⁵⁶.

To clarify the role of face-emotion recognition as a socioemotional developmental pathway, the present data begin to satisfy the proposed criteria⁵⁷ to suggest that face-emotion recognition, specifically the recognition of happiness, may present a useful psychiatric endophenotype⁵⁸. The present data demonstrate that face-emotion recognition deficits, specifically regarding happiness, co-segregate with irritability, neuroticism, and depression within families partially due to mutual genetic influences. Additionally, the present community sample satisfies the requirement that the endophenotype be ‘state independent,’ i.e., manifest outside of active psychiatric episodes. While the sample approximates developmentally expected psychopathology rates^{23,26}, results demonstrate the contribution of genetic and environmental factors to the association of face-emotion recognition with irritability, neuroticism, and depression in a community sample. Finally, the present study replicates existing evidence of impaired emotion recognition in children with elevated irritability¹², neuroticism¹⁹, and related conditions¹⁴.

Several limitations should be addressed in future research. First, data were collected at a single time point. Developmental mechanisms are presumed to originate prior to the associated disorder. Longitudinal research is needed to characterize the development of face-emotion recognition and associated clinical phenotypes. For example, recent statistical developments estimate causal relationships within longitudinal research of mono- and dizygotic twins⁵⁹. Second, although the genetic and environmental contributions to face-emotion recognition were similar between male and female participants (see Supplemental Table 3), sex effects in the genetic and environmental contributions to emotion recognition warrant further investigation. Lau and colleagues examine sex effects²⁰ though both their sample and ours are underpowered to robustly estimate whether sex moderates genetic contributions⁶⁰. Third, since participants are predominantly from one ancestral group, the present study optimizes statistical power for genomic analysis. However, further research is needed to generalize results to youth from other backgrounds. Finally, the present analyses are consistent with a small contribution of shared, familial environment to face-emotion recognition (Table 4). Though this is not statistically significant, careful interpretation is warranted as the chi-square approach is biased downwards in multivariate models⁵⁴.

Despite the above limitations, these data demonstrate the importance of face-emotion recognition to socioemotional functioning in childhood and adolescence. Given deficits associated with irritability, neuroticism, and depression, the present study implicates face-emotion recognition in multiple psychiatric conditions. This supports incorporating emotion recognition and the amelioration of related deficits into existing treatment for childhood irritability, depression, and related psychopathologies. The present study encourages further study into the potentially broad clinical impact of existing interventions that address emotion recognition and regulation in childhood and adolescence⁶¹. Finally, considering improved emotion recognition during adult pharmacotherapy⁶², further research might examine face-emotion recognition as mechanism-of-action for pharmacological and cognitive-behavioral treatment of child and adolescent conditions.

Conclusion.

Deficits in recognizing others' facial expression of emotion have been identified in multiple psychiatric conditions, particularly those characterized by negative affect. The present study provides a critical first step to evaluate the contribution of genetic and environmental influences to face-emotion recognition deficits. Moreover, the present study demonstrates that common genetic influences might explain specific deficits recognizing happiness among children and adolescents who report elevated irritability and neuroticism. Finally, to examine whether face-emotion recognition may present a candidate psychiatric endophenotype⁵⁷, the present study demonstrates that face-emotion recognition deficits are, within a representative community sample, associated phenotypically with irritability, neuroticism, and depression; heritable; and co-segregate with irritability and neuroticism based on correlated genetic factors.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Best Fitting Model for Genetic and Environmental Contributions to Face-emotion Recognition

Note. A = additive genetic factor; C = shared environmental factor; E = nonshared-environmental factor; Factor numbers correspond to Supplemental Table 2 under “Cholesky Factor Loading Estimates,” specifically factors 1–6; factors with estimated loadings below 0.3 are omitted for clarity. Loadings are standardized. Variances for latent variables, not depicted, are constrained to 1.

Table 1.
Test-Retest Reliability, Descriptive Statistics, and Phenotypic Correlations Among Recognition of Each Emotion

Emotion	Mean (SD)	Test-Retest Reliability: <i>r</i> (95% CI)	Correlation Matrix					
			Anger	Happiness	Sadness	Fear	Surprise	Disgust
Anger	0.03 (0.07) ^{a,b}	0.69 (0.52, 0.80)	1					
Happiness	0.38 (0.17) ^{a,b}	0.69 (0.51, 0.80)	0.55	1				
Sadness	0.08 (0.11) ^{a,b}	0.76 (0.63, 0.83)	0.51	0.62	1			
Fear	0.06 (0.13) ^{a,b}	0.64 (0.43, 0.77)	0.52	0.59	0.61	1		
Surprise	0.19 (0.14) ^{a,b}	0.67 (0.50, 0.79)	0.50	0.58	0.53	0.64	1	
Disgust	-0.04 (0.09) ^{a,b}	0.75 (0.61, 0.85)	0.50	0.37	0.38	0.33	0.31	1
Irritability	2.96 (2.66) ^b	--	0.01 (-0.06, 0.08)	-0.11 ^{**} (-0.18, -0.04)	-0.11 ^{**} (-0.17, -0.04)	-0.12 ^{**} (-0.18, -0.05)	-0.05 (-0.12, 0.02)	-0.03 (-0.1, 0.04)
Neuroticism	7.04 (4.57) ^b	--	-0.05 (-0.13, 0.02)	-0.16 ^{****} (-0.23, -0.09)	-0.14 ^{****} (-0.21, -0.07)	-0.14 ^{****} (-0.21, -0.06)	-0.08 [*] (-0.15, -0.01)	-0.09 ^{***} (-0.17, -0.02)
Depression	5.28 (4.24) ^b	--	0.05 (-0.02, 0.12)	-0.08 [*] (-0.15, -0.01)	-0.07 (-0.14, 0)	-0.02 (-0.1, 0.05)	-0.02 (-0.09, 0.05)	0 (-0.07, 0.07)

Note.

* *p* 0.05,

** *p* 0.01,

*** *p* 0.001,

**** *p* 0.0001;

^a All means are significantly different from one another; an overall ANOVA and equivalence of each pair of means were tested within the biometrical model. Significance was evaluated using the chi-square difference test, which evaluates worsened model fit due to constraining the mean of all emotions (the overall ANOVA; $p = 3.34e^{-255}$) or each pair of two emotions to be equal ($p = 2.16e^{-8}$).

^b Mean face-emotion recognition differs as a function of participant age and sex; female participants generally demonstrated better recognition as well as higher neuroticism and depression, but similar irritability severity (see Supplemental Table 1).

Table 2.

Nested Model Fit for Multivariate Models

model	np	-2LL	df	AIC	LL	df	p
“Saturated”, Cholesky	69	12411.09	5156	2099.09	--	--	--
CPM	33	12525.79	5193	2139.79	114.70	37	7.01 e ⁻¹⁰
IPM	42	12476.10	5183	2110.10	65.01	27	5.60 e ⁻⁹⁵

Notes. IPM = independent pathway model; CPM = common pathway model; np = number of parameters; -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike Information Criteria; LL = log likelihood difference, df = degree of freedom difference.

Table 3.

Nested Models Fit for Variance Components of Best-Fitting Multivariate Model

comparison	np	-2LL	df	AIC	LL	df	p
ACE	69	14103.12	5870	2363.12	--	--	--
CE	48	12455.76	5177	2101.76	44.67	21	0.002
AE	48	12424.80	5177	2070.80	13.71	21	0.88
E	27	12627.50	5198	2231.50	216.41	42	2.47 e ⁻²⁵

Notes. np = number of parameters; -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike Information Criteria; LL = log likelihood difference, df = degree of freedom difference.

Table 4.

Variance Components for Recognition of Each Emotion

Emotion	Additive Genetic Factors (A)	Shared Environmental Factors (C)	Nonshared Environmental Factors (E)
Anger	0.40 (0.23, 0.53)	0.08 (0, 0.20)	0.53 (0.43, 0.63)
Happiness	0.57 (0.35, 0.67)	0.03 (0, 0.21)	0.40 (0.32, 0.49)
Sadness	0.34 (0.18, 0.49)	0.10 (0, 0.26)	0.56 (0.47, 0.65)
Fear	0.44 (0.24, 0.60)	0.12 (0, 0.29)	0.43 (0.35, 0.53)
Surprise	0.49 (0.30, 0.59)	0.01 (0, 0.16)	0.50 (0.41, 0.60)
Disgust	0.41 (0.16, 0.54)	0.06 (0, 0.25)	0.53 (0.44, 0.65)