Research Article

Heritability of Justice Sensitivity

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Abstract: Justice is one of the fundamental principles in human evolution, and justice sensitivity from the pro-self (e.g., as a victim) and the prosocial perspective (e.g., as an observer, beneficiary, and perpetrator) matters in mental wellness and social interaction. However, the extent to which individual difference in justice sensitivity is influenced by genetic versus environmental factors remains unclear. Using a sample with 244 twin pairs, the present research attempts to determine the extent to which genetic factors play a role in the inter-individual difference of justice sensitivity as well as whether different facets of justice sensitivity, namely, pro-self and prosocial perspectives, share a common genetic basis. Results showed that (1) all facets of justice sensitivity were moderately heritable (21–33%) and that the non-shared environmental factors plus measurement error accounted for the rest of the variations (67–79%); (2) associations between the prosocial facets of justice sensitivity were driven by common genetic (r_g = .50–.65) and non-shared environmental (plus measurement error; r_e = .24–.65) influences, whereas no significant genetic link was found between the pro-self and prosocial facets. The current findings provide novel evidence that sensitivity to injustice, especially to others' suffering, is fundamentally grounded upon genetic origin, thereby shedding light on the nature and nurture aspects of justice behavior.

Keywords: justice sensitivity, heritability, twin study, behavioral genetics, individual difference

Justice is a crucial force for social stability and has deep evolutional roots (Buckholtz & Marois, 2012; Wallace et al., 2007). Justice matters to all people (Schmitt et al., 2010), and people are sensitive in their reaction to the situations when themselves or others are treated unfairly (Baumert & Schmitt, 2016; Schmitt, 1996; Schmitt & Mohiyeddini, 1996; Schmitt et al., 1995). Justice sensitivity, which refers to the tendency to perceive and adversely respond to injustice, is essential for the mental health and subjective well-being of individuals (Baumert & Schmitt, 2016). Moreover, justice sensitivity has been taken as a potential risk factor for the emergence and maintenance of psychiatric disorders that have heritable origins in etiology, such as attention deficit hyperactivity disorder (Bondü & Elsner, 2015), depression (Bondü et al., 2017), anxiety (Bondü & Inerle, 2020), and borderline personality disorder (Lis et al., 2018).

However, whether the dispositional nature of justice sensitivity is more important than situational and societal factors for understanding justice principles and behavior is still disputed (Schmitt et al., 2005). That is, understanding the origin of justice sensitivity is vital to research on justice behavior. In the current work, our interest lies in assessing the extent to which genetic factors play a role in the inter-individual difference of justice sensitivity and whether different facets of justice sensitivity, namely, for oneself and others, share a common genetic basis.

Individual Differences in Justice Sensitivity

Recent theories assume that justice sensitivity involves a perceptual threshold for injustice, strong emotional responses to injustice, a tendency to ruminate about injustice, and a motivation to reestablish justice (Schmitt et al., 2005). Justice sensitivity divides the concern for justice into four facets according to the role that a person plays in an unjust situation, namely, as the victim, observer or bystander, passive beneficiary, or active perpetrator (Mikula et al., 1990). The victim is the person who feels unjustly treated, the observer is the one who perceives the incident without being directly involved, the beneficiary is the person who receives the benefit passively, and the perpetrator is the one who committed the critical action (Gollwitzer et al., 2005). These four facets of justice sensitivity can be further distinguished as justice concerns for the self and for others, such that victim sensitivity (VS) involves pro-self concerns for one's interest, whereas observer sensitivity (OS), beneficiary sensitivity (BS), and perpetrator sensitivity (PS)

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involve prosocial concerns for others and public well-being. VS is a mixture of self-related concerns and a sense of justice, while the remaining facets have no selfish component but indicate prosocial and genuine justice concerns (Thomas et al., 2011). The four facets of justice sensitivity also appear to overlap, with correlation coefficients ranging from 0.3 to 0.8 (Schmitt et al., 2010) because they share the concern for justice as a common element. Regarding the characteristic emotional responses to injustice, BS and PS share a clear similarity in terms of guilty reactions, whereas VS and OS induce outward-focused emotions, such as anger and moral outrage, respectively (Thomas et al., 2011; for a review, see Baumert & Schmitt, 2016).

Although individual justice sensitivity depends on the adopted perspectives, all four facets of justice sensitivity show dispositional stability concerning personality traits, behavioral consequences, and cultural values. For personality traits, the pro-self facet (VS) is associated primarily with self-related concerns, such as neuroticism, Machiavellianism, suspiciousness, paranoia, jealousy, and vengeance. By contrast, the prosocial facets (OS, BS, and PS) are associated primarily with other-related concerns, such as agreeableness, empathy, role-taking, and social responsibility (Schmitt et al., 2005, 2010). For behavioral consequences, OS, BS, and PS positively correlate with positive behavioral reciprocity and negatively with negative behavioral reciprocity. VS negatively correlates with positive behavioral reciprocity and positively correlates with negative behavioral reciprocity (Baumert et al., 2014). In particular, people with high (vs. low) OS contribute more to the public good regardless of past exploitation (Gollwitzer et al., 2009). Finally, for cultural values, recent cross-cultural studies reveal that variations in the prosocial facets of justice sensitivity have a stable relationship with collectivism at the country level and at the individual level (Maltese et al., 2020; Wu et al., 2014).

Origins of Justice Sensitivity

Previous observational research suggested that animals respond negatively to inequity. For example, chimpanzees responded with temper tantrums if they did not obtain what they desired, social canids refused to play with individuals who violated social rules, and ravens showed third party intervention against norm violations (Brosnan, 2006; Brosnan & De Waal, 2003). Developmental psychology and behavioral genetics suggest that human nature or genetic factors partly determine the individual differences in justice sensitivity. Development studies found that children as young as 2 years demonstrated preferences for fairness in ultimatum games (Li et al., 2016). A general predisposition towards just evaluations was also observed in 6- and 10-month-old infants who chose and looked longer at an individual who helped another than at one who hindered another (Hamlin et al., 2007; Warneken & Tomasello, 2006). In summary, the inequity response in animals and the appearance of fairness preference in the early stages of life suggest that intuitive reaction to injustice has deep biological roots. Behavioral genetics studies on twins further suggest that genes moderately contribute to the fairness-related decision-making behavior (Wallace et al., 2007; Wang et al., 2019) and morality-related traits, such as honesty–humility (Kandler et al., 2019).

However, from the perspective of socialization theory, environmental factors are also found to affect individual differences in justice sensitivity. For example, elder and highly educated people showed a lower level of pro-self justice sensitivity and a higher level of prosocial justice sensitivity than younger and lower educated people (Schmitt et al., 2010). In addition, compared with Western Germans (Capitalism-oriented), Eastern Germans (Socialism-oriented) showed higher levels of all facets of justice sensitivity (Schmitt et al., 2010). People in collectivist cultures, such as the Chinese, had high BS and comparable OS when compared with individualist Germans, Russians, and North Americans (Wu et al., 2014). Similarly, on prosocial facets of justice sensitivity, the collectivist Filipinos scored higher than the individualist Australians (Maltese et al., 2020).

Although ample experimental evidence has discussed the cultural and evolutionary origins of justice sensitivity, the relative social and genetic contributions have hitherto been left unexplored. The extent to which genetic and environmental factors contribute to the inter-individual differences in justice sensitivity is unknown. Besides, given that justice sensitivity consists of four components and is positively correlated with each other, whether common genetic or environmental factors that determine the phenotypic correlations between different facets of justice sensitivity exists is also unknown. Therefore, empirical investigation on the extent to which nature and nurture contribute to the course of human development in justice sensitivity is still necessary.

Present Research

To summarize, the current study aims to answer two questions. First, we investigate the extent to which genetic and environmental factors play a role in the inter-individual difference of justice sensitivity. Second, we investigate whether different facets of justice sensitivity share common genetic or environmental factors in terms of justice for self and others. Behavioral genetics, which pertains fundamentally to the study of human variations, could provide important clues as to the sources of individual differences (Plomin et al., 2013). Specifically, the twin methodology can be used to identify the extent to which individual differences in justice sensitivity are influenced by genetic and environmental factors and further examine the pairwise genetic and environmental correlations between multiple variables. Therefore, a twin study was conducted to examine the genetic and environmental bases of justice sensitivity and the genetic and environmental associations between different facets of justice sensitivity.

Method

Participants

A total of 244 same-sex twin pairs (133 females, aged 18–25, $M = 19.72$, $SD = 1.77$) were recruited to participate in this study, among which 151 pairs were monozygotic (MZ) and 93 pairs were dizygotic (DZ). The sample was based on the participant pool of the Beijing Twin Study (BeTwiSt), a longitudinal study that recruited reared-together twins from over 600 public schools in Beijing, China. Detailed information regarding the project and key findings are available in the studies by Chen et al. (2013) and Bi et al. (2019). The zygosity of the twin pairs in the present study was determined by DNA testing. The ethics committee provided approval for the study. We obtained written informed consent from each participant before commencing the study.

Measures

The Chinese version of the Justice Sensitivity Inventory was used to assess participants' sensitivity to injustice (Schmitt et al., 2010; Wu et al., 2014). Each subscale contains 10 items that measure a single facet: VS (e.g., "It bothers me when others receive something that ought to be mine"), OS (e.g., "It bothers me when someone gets something they do not deserve"), BS (e.g., "I ruminate for a long time about being treated nicer than others for no reason"), and PS (e.g., "It gets me down when I take something from someone else that I do not deserve"). Participants responded to the items on a 0–5 Likert scale (0 = *not at all*, 5 = *exactly*), with high scores indicating high sensitivity to injustice from the victim, observer, beneficiary, and perpetrator perspectives. Individual scores were obtained by averaging the items of the relevant subscale. The estimated reliabilities (Cronbach's α) of the four facets of justice sensitivity were .83 for VS, .90 for OS, .85 for BS, and .89 for PS, respectively.

Data Analysis

Additive genetic (A), shared environmental (C), and non-shared environmental (E) contributions to variance within a trait and covariance between traits can be estimated by employing quantitative genetic modeling (Plomin et al., 2013). A significant A suggests that genetic factors contribute significantly to the variation of the trait or covariance between traits. A shared environment contributes to the similarity of twins raised in the same family. The non-shared environment is unique to each individual, which also includes measurement error and thus cannot be omitted.

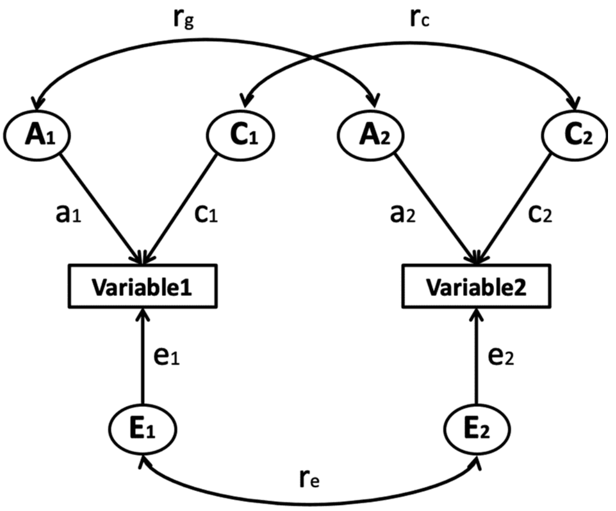

Figure 1. Path diagram illustrating bivariate genetic model-fitting. Measured variables are in rectangles. Latent factors A (genetic factors), C (shared environmental factors), and E (non-shared environmental factors plus measurement error) are in circles. r_g = genetic correlation; r_c = shared environmental correlation; r_e = non-shared environmental (plus measurement error) correlation.

Twins are perfectly correlated for sex and age in our study. Thus, variation associated with gender and age would inflate the correlation between twins. We separately regressed each facet of justice sensitivity onto sex and age and saved the standardized residuals for genetic analyses. Participants who scored ±3 *SD* beyond the mean value of the observed trait were excluded to avoid the effects of extreme values on genetic analyses. The numbers of excluded participants were three for VS, two for OS, one for BS, and four for PS, respectively. We used all available data, including those from several twin pairs that were not pairwise, to increase the statistical power of genetic model-fitting.

Univariate and multivariate models were implemented in the OpenMx package for R version 3.0.1 to estimate genetic and environmental effects. First, univariate models were used to partition the variance of each facet into genetic (A) and environmental (C and E) effects. For each facet, the full ACE model was examined first. Sub-models (AE, CE, and E) nested within the full model were then tested by removing one or two variance component(s). Next, a correlated factors model (Loehlin, 1996; Figure 1), wherein each variable was decomposed into ACE components separately, was used for the multivariate analyses of the correlated facets. The correlations of these components across

Table 1. Means, zero-order correlations among, and twin intraclass correlations for all measures

			Zero-order correlation			Twin correlations			
Measure	Mean	*SD*	VS	OS	BS	ICC_{MZ}	N_{MZ}	ICC_{DZ}	N_{DZ}
VS	2.71	0.71				.48***	150	−.10	91
OS	2.29	0.83	.37***			.42***	150	−.08	93
BS	2.82	0.79	.17***	.53***		.51***	151	.33*	92
PS	3.45	0.79	.08	.31***	.61***	.36***	150	.17	91

Note. VS = Victim Sensitivity; OS = Observer Sensitivity; BS = Beneficiary Sensitivity; PS = Perpetrator Sensitivity; ICC = Intraclass Correlation; MZ = Monozygotic Twins; DZ = Dizygotic Twins; *N* = Number of Twin Pairs. ****p* < .001, **p* < .05.

variables were also estimated. The full ACE model and all the sub-models were tested systematically.

We used three model fit indices: the change in chi-square ($\Delta\chi^2$), Akaike's Information Criterion (AIC; Akaike, 1987), and Bayesian Information Criterion (BIC; Raftery, 1995). Comparing the full model with a sub-model, a significant $\Delta\chi^2$ suggests that the nested model fits worse than the full model, and thus, the full model should be chosen; otherwise, the nested model with fewer parameters should be considered (Bollen, 1989; Kline, 1998). AIC and BIC are useful in model selection, with low values indicating better fit than high values. A BIC difference of 5 indicates "strong evidence" that one model is superior to another, whereas a difference of 10 indicates "conclusive evidence" (Raftery, 1993). Finally, after comprehensive consideration, the best-fitting model was chosen (Kline, 1998).

Statistical Power for Genetic Analyses

We calculated the minimum effect size that could be reliably observed in our sample to detect the statistical power of genetic or environmental effects in genetic analyses. We conducted the power analysis through the package "pwr" in R (Champely, 2018), which functions along the lines of Cohen (1988). Given a sample size of N = 241/243 (the total number of twin pairs; Table 1), level of significance at .10 (Verhulst, 2017), *df* at 1 (estimating one effect each time), and power at .80 conventionally, we can detect that the minimum effect size (*w*) was .16. In other words, if the standardized estimate of a path parameter in the genetic model (e.g., A in the univariate model or a1 in the multivariate model) was no less than .16, we would have sufficient power to examine the corresponding genetic or environmental effects. This type of method for estimating statistical power for genetic analyses has been successfully adopted by Luo et al. (2020).

Results

Table 1 shows the mean scores and standard deviations for each facet of justice sensitivity. Table 1 further shows that the pro-self facet of justice sensitivity (VS) was significantly and positively correlated with the prosocial facets of justice sensitivity (OS and BS). The correlations between each pair of prosocial facets (OS, BS, and PS) were also positively significant. The correlation between VS and PS was positive but not significant. The correlation pattern replicated the findings in previous studies. Typically, the correlation between BS and PS is the highest, while that between VS and PS is the lowest (Schmitt et al., 2010).

Univariate Model Fitting

For the four facets of justice sensitivity, MZ twin correlations were significantly higher than DZ correlations (Table 1), suggesting that genes substantially contribute to individual differences in terms of sensitivity to injustice. Thereafter, the heritability of each facet was examined by fitting a series of univariate models. In comparison with the ACE model, the E model fit significantly worse for each facet of justice sensitivity ($\Delta\chi^2$s $\geq$ 7.16, *p*s $\leq$ .03), the AE model fit equally well for VS, OS, and PS ($\Delta\chi^2$s = 0.00, *p*s = 1.00), and the AE and CE models each fit equally well for BS ($\Delta\chi^2$s $\leq$ 0.92, *p*s $\geq$ .34). Therefore, the nested model with fewer parameters (AE/CE) should be considered (Bollen, 1989; Kline, 1998). Finally, the AE model was considered more desirable because its AIC and BIC values were smaller than the CE model for each facet (Table 2). Table 2 shows that the univariate model fitting revealed moderate heritability for each facet (27% for VS, 21% for OS, 33% for BS, and 23% for PS) and large non-shared environmental effects plus measurement error (73% for VS, 79% for OS, 67% for BS, and 77% for PS). Importantly, the magnitude of all genetic and non-shared environmental effects was above the minimal effect size (.16) that we could detect with sufficient power.

Bivariate Model Fitting

Bivariate modeling analyses were conducted using correlated-factors models to determine the extent of covariation between each pair of justice sensitivity facets (OS–BS, OS–PS, BS–PS, VS–OS, and VS–BS) because of genetic and environmental factors.

Table 2. Univariate genetic model-fitting

						Change from full model					
Measure	Model	−2LL	*df*	AIC	BIC	$\Delta\chi^2$	Δdf	*p*	A	C	E
VS	ACE	1,026.88	481	64.88	−1,617.26				.27 (.02, .40)	.00 (.00, .19)	.73 (.60, .88)
	AE	1,026.88	482	62.88	−1,622.75	0.00	1	1.00	.27 (.12, .40)		.73 (.60, .88)
	CE	1,031.22	482	67.22	−1,618.42	4.33	1	.04		.18 (.06, .30)	.82 (.70, .94)
	E	1,039.58	483	73.57	−1,615.56	12.69	2	< .001			1.00 (1.00, 1.00)
OS	ACE	1,186.66	482	222.66	−1,462.97				.21 (.00, .36)	.00 (.00, .19)	.79 (.64, .94)
	AE	1,186.66	483	220.66	−1,468.47	0.00	1	1.00	.21 (.06, .36)		.79 (.64, .94)
	CE	1,189.70	483	223.70	−1,465.44	3.03	1	.08		.13 (.00, .25)	.87 (.75, 1.00)
	E	1,193.82	484	225.82	−1,466.81	7.16	2	.03			1.00 (1.00, 1.00)
BS	ACE	1,358.97	483	392.97	−1,296.16				.23 (.00, .45)	.09 (.00, .38)	.68 (.55, .82)
	AE	1,359.13	484	391.13	−1,301.50	0.16	1	.69	.33 (.19, .45)		.67 (.55, .81)
	CE	1,359.89	484	391.89	−1,300.74	0.92	1	.34		.28 (.16, .39)	.72 (.61, .84)
	E	1,380.04	485	410.04	−1,286.08	21.07	2	< .001			1.00 (1.00, 1.00)
PS	ACE	1,363.24	480	403.24	−1,275.40				.23 (.00, .37)	.00 (.00, .27)	.77 (.63, .93)
	AE	1,363.24	481	401.24	−1,280.90	0.00	1	1.00	.23 (.07, .37)		.77 (.63, .93)
	CE	1,364.58	481	402.58	−1,279.56	1.34	1	.25		.17 (.04, .29)	.83 (.71, .96)
	E	1,371.53	482	407.53	−1,278.11	8.29	2	.02			1.00 (1.00, 1.00)

Note. VS = Victim Sensitivity; OS = Observer Sensitivity; BS = Beneficiary Sensitivity; PS = Perpetrator Sensitivity; −2LL = Twice the Negative Log-Likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; $\Delta\chi^2$ = Change in Chi-Square; Δdf = Change in Degrees of Freedom (*df*); A = Proportion of Variance Due to Additive Genetic Effects; C = Proportion of Variance Due to Shared Environmental Effects; E = Proportion of Variance Due to Non-Shared Environmental Effects Plus Measurement Error. 95% confidence intervals are in parentheses. E, CE, and AE models are nested within the ACE model. The best-fitting model is italicized.

Observer Sensitivity and Beneficiary Sensitivity

We tested the full ACE model first and then the AE, CE, and E models (Table 3). Compared with the ACE model, the AE and CE models each fit equally well ($\Delta\chi^2$s $\leq$ 3.44, *p*s $\geq$ .33), but the E model fit significantly worse ($\Delta\chi^2$ = 27.80, p < .001). The AE model was deemed preferable because its AIC and BIC values were smaller than those of the CE model (Bollen, 1989; Kline, 1998; Raftery, 1993; see Figure 2A). In the AE model, genes that influenced OS also moderately influenced BS (r_g = .65), whereas non-shared environments plus measurement error that affected OS also had a significant effect on BS (r_e = .49).

Observer Sensitivity and Perpetrator Sensitivity

The same model analysis was conducted for OS and PS. Table 3 shows the AE model was optimal ($\Delta\chi^2$ = 0.55, p = .91). OS and PS shared moderate genetic (r_g = .58) and non-shared environmental (plus measurement error; r_e = .24) correlations (Figure 2B).

Beneficiary Sensitivity and Perpetrator Sensitivity

We conducted the same model analysis for BS and PS. Table 3 shows that the AE model provided the best fit for the data ($\Delta\chi^2$ = 1.24, p = .74). In the AE model, BS and PS shared moderate genetic (r_g = .50) and non-shared environmental (plus measurement error; r_e = .65) correlations (Figure 2C).

Victim Sensitivity and Observer Sensitivity

Table 3 shows that the AE model was optimal ($\Delta\chi^2$ = 0.00, p = 1.00). In the AE model, VS and OS shared modest genetic (r_g = .21) and non-shared environmental (plus measurement error; r_e = .42) correlations. Notably, the 95% confidence interval of the genetic correlation (–.45 to .58) included zero (Figure 2D). Thus, no sufficient evidence was found to support a genetic correlation between VS and OS.

Victim Sensitivity and Beneficiary Sensitivity

Table 3 shows that the AE model provided the best fit for the data ($\Delta\chi^2$ = 0.18, p = .98). In the AE model, VS and BS shared minimal genetic correlation (r_g = .07) and modest non-shared environmental correlation (plus measurement error; r_e = .21). Notably, the 95% confidence interval of the genetic correlation (–.35 to .41) included zero (Figure 2E). Thus, no sufficient evidence to support a genetic correlation between VS and BS was observed.

Trivariate Model Fitting

We further conducted a trivariate model fitting by using a correlated-factors model because the pairwise correlations between the three prosocial facets of justice sensitivity had significant behavioral correlations (Figure 3A) and significant genetic correlations (Figure 2) across the pairwise facets.

Table 3. Bivariate genetic model-fitting

						Change from full model		
Measure	Model	−2LL	*df*	AIC	BIC	$\Delta\chi^2$	Δdf	*p*
OS–BS	ACE	2,388.19	962	464.19	−2,900.09			
	AE	2,389.99	965	459.99	−2,914.78	1.80	3	.61
	CE	2,391.63	965	461.63	−2,913.14	3.44	3	.33
	E	2,415.99	968	479.99	−2,905.27	27.80	6	< .001
OS–PS	ACE	2,501.36	959	583.36	−2,770.42			
	AE	2,501.91	962	577.91	−2,786.36	0.55	3	.91
	CE	2,505.80	962	581.80	−2,782.48	4.44	3	.22
	E	2,516.49	965	586.49	−2,788.28	15.13	6	.02
BS–PS	ACE	2,494.58	960	574.58	−2,782.70			
	AE	2,495.82	963	569.82	−2,797.95	1.24	3	.74
	CE	2,496.24	963	570.24	−2,797.54	1.65	3	.65
	E	2,530.97	966	598.97	−2,779.29	36.39	6	< .001
VS–OS	ACE	2,138.61	960	218.61	−3,138.67			
	AE	2,138.61	963	212.61	−3,155.17	0.00	3	1.00
	CE	2,145.04	963	219.04	−3,148.73	6.43	3	.09
	E	2,161.72	966	229.72	−3,148.54	23.12	6	< .001
VS–BS	ACE	2,370.37	961	448.37	−2,912.41			
	AE	2,370.55	964	442.55	−2,928.72	0.18	3	.98
	CE	2,384.55	964	456.55	−2,914.72	14.18	3	< .001
	E	2,405.91	967	471.91	−2,909.85	35.54	6	< .001

Note. VS = Victim Sensitivity; OS = Observer Sensitivity; BS = Beneficiary Sensitivity; PS = Perpetrator Sensitivity; −2LL = Twice the Negative Log-Likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; $\Delta\chi^2$ = Change in Chi-Square; Δdf = Change in Degrees of Freedom (*df*); A = Additive Genetic Effects; C = Shared Environmental Effects; E = Non-Shared Environmental Effects Plus Measurement Error. E, CE, and AE models are nested within the ACE model. The best-fitting model is italicized.

We tested the full ACE model first and then the AE, CE, and E models. Table 4 shows that the correlated-factors model with only genetic and non-shared environmental components (i.e., AE model) provided the best fit for the data ($\Delta\chi^2 = 2.3$, $p = 0.89$). In the AE model, the three prosocial facets shared moderate genetic (r_g ranging from .51 to .65) and non-shared environmental (plus measurement error; r_e ranging from .24 to .64) correlations. Notably, the 95% confidence interval of the genetic correlation between OS and BS included zero (Figure 3B).

In summary, the prosocial facets of justice sensitivity correlated with each other in the genetic and environmental factors, and the magnitude of all genetic and non-shared environmental effects (plus measurement error) was above the minimal effect size (.16) that can be detected with sufficient power. However, the genetic correlations between the pro-self and prosocial facets of justice sensitivity were not significant. This analysis provides further evidence for the distinction between justice sensitivity for one's self and others.

Discussion

Justice is a fundamental concern of human societies and a critical component of morality. Individuals are motivated by considerations of justice, such as equity and fairness for themselves and others from an early age. Despite robust evidence that the desire for justice may be universal, numerous studies have revealed that individuals differ in their reactions to injustices when they or others are treated unfairly. Justice sensitivity has been proposed to be a risk factor that may contribute to the emergence and maintenance of several most common mental disorders (Bondü & Elsner, 2015; Bondü & Inerle, 2020; Bondü & Sahyazici-Knaak, 2017; Lis et al., 2018). With such consideration, we examined the genetic contribution to justice sensitivity variations and the genetic associations between different facets of justice sensitivity using twins. The current results identified modest genetic (21–33%) and large non-shared environmental contributions (plus measurement error; 67–79%) to justice sensitivity. The shared environmental contribution to justice sensitivity was minimal. More importantly, moderate genetic (.50–.65) and non-shared environmental (plus measurement error; .24–.65) correlations were found to be among the prosocial facets of justice sensitivity (OS, BS, and PS), whereas no significant genetic link was observed between the pro-self and prosocial facets. Thus, these findings provide novel evidence on the genetic basis of justice sensitivity and the associations between different facets of prosocial justice sensitivity.

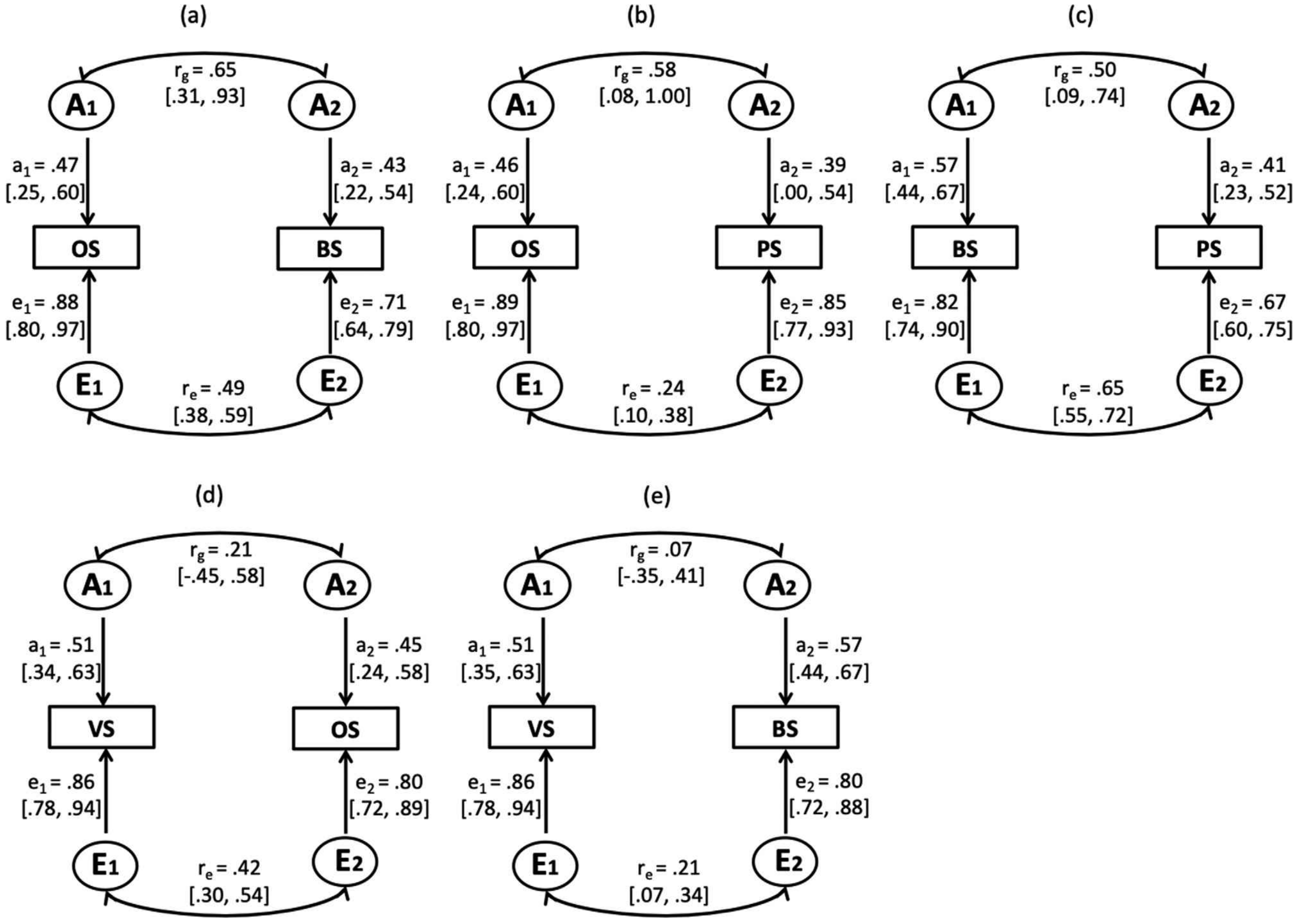

Figure 2. Best-fitting bivariate genetic models: (A) best-fitting model for OS and BS; (B) best-fitting model for OS and PS; (C) best-fitting model for BS and PS; (D) best-fitting model for VS and OS; (E) best-fitting model for VS and BS. VS = Victim Sensitivity; OS = Observer Sensitivity; BS = Beneficiary Sensitivity; PS = Perpetrator Sensitivity. Measured variables are in rectangles. Latent factors A (additive genetic factors) and E (non-shared environmental factors plus measurement error) are in circles. r_g = genetic correlation; r_e = non-shared environmental correlation (plus measurement error). All path estimates (95% confidence intervals), standardized but unsquared, are obtained from the best-fitting model.

The results support our hypothesis that the nurture and nature aspects play important roles in the individual differences in justice sensitivity. Studies have uncovered the heritability of numerous attitudes (Olson et al., 2001) and various aspects of social behavior (Ebstein et al., 2010), such as fairness-related decisions (Cesarini et al., 2008; Wallace et al., 2007; Wang et al., 2019). In the current study, the identified heritability of justice sensitivity has implications for understanding individual differences in sensitivity to injustice. Individuals differ in how readily they perceive and how strongly they react to injustice. Several systematic examinations have focused on individual differences in the emotions and behaviors that result from experiencing or witnessing injustice (Baumert & Schmitt, 2016; Schmitt, 1996; Schmitt et al., 2005, 2010). Based on such previous investigations, the present study moves a step forward by conducting a behavioral genetics study on twins and finds moderate justice sensitivity heritability and large non-shared environmental influence (plus measurement error) for all facets of justice sensitivity. These findings provide novel evidence that justice sensitivity is a fundamental trait with reliable genetic bases and environmental influences, thereby shedding light on the nature and nurture aspects of justice and morality.

A series of studies have documented the associations between the four facets of justice sensitivity (Gollwitzer et al., 2005; Schmitt et al., 2010). In line with the previous findings, we observed significant correlations for each paired facet of justice sensitivity except for VS and PS. More importantly, our findings offer biological evidence for the homogeneity of prosocial justice sensitivity for others. The associations between each pair of prosocial justice sensitivity (OS, BS, and PS) were found to be due in part to genetic factors, although non-shared environments (and measurement error) also play a role. This discovery implies an overlap of the genes and non-shared environments (and measurement error) that influence the three facets of prosocial justice sensitivity and provides evidence for the

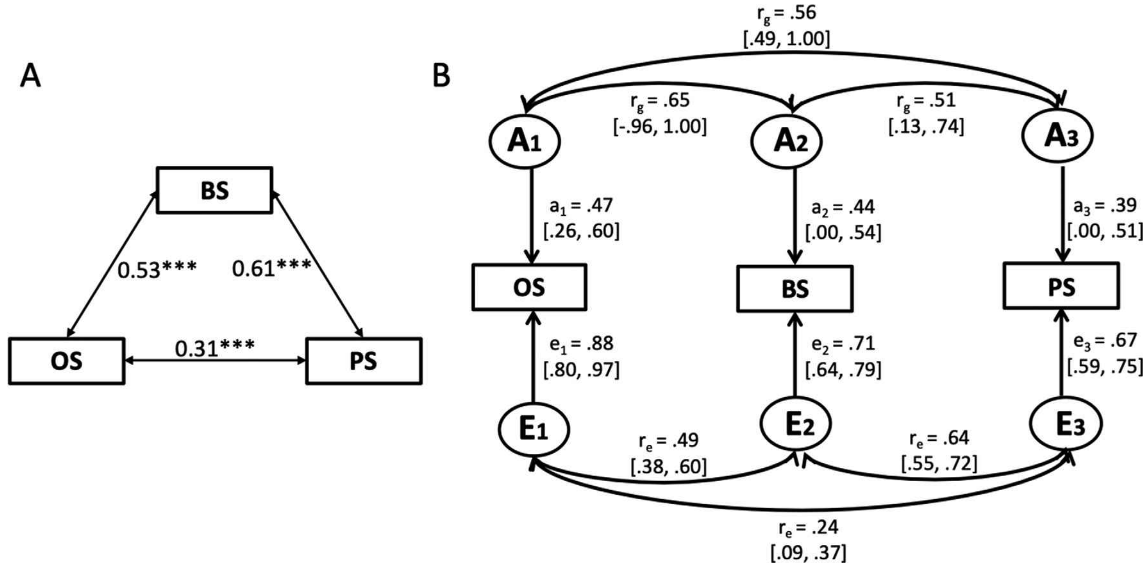

Figure 3. Behavioral and genetic correlations across the three prosocial facets. (A) Behavioral correlation coefficients between the three prosocial facets (***$p < .001$). (B) Best-fit trivariate genetic model for the three prosocial facets. Measured variables are in rectangles. Latent factors A (additive genetic factors) and E (non-shared environmental factors plus measurement error) are in circles. r_g = genetic correlation; r_e = non-shared environmental correlation (plus measurement error). All path estimates (95% confidence intervals), standardized but unsquared, are obtained from the best-fitting model.

Table 4. Trivariate genetic model-fitting

						Change from full model		
Measure	Model	−2LL	*df*	AIC	BIC	$\Delta\chi^2$	Δ*df*	*p*
OS–BS–PS	ACE	3,521.77	1,436	649.77	−4,372.17			
	AE	3,524.07	1,442	640.07	−4,402.85	2.30	6	.89
	CE	3,530.11	1,442	646.11	−4,396.81	8.34	6	.21
	E	3,566.74	1,448	670.74	−4,393.16	44.98	12	< .001

Note. OS = Observer Sensitivity; BS = Beneficiary Sensitivity; PS = Perpetrator Sensitivity; −2LL = Twice the Negative Log-Likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; $\Delta\chi^2$ = Change in Chi-Square; Δ*df* = Change in Degrees of Freedom (*df*); A = Additive Genetic Effects; C = Shared Environmental Effects; E = Non-Shared Environmental Effects Plus Measurement Error. E, CE, and AE models are nested within the ACE model. The best-fitting model is italicized.

inherent nature behind these links. Our findings also indicate the genetic distinctiveness between pro-self justice sensitivity (VS) and the three prosocial facets of justice sensitivity (OS, BS, and PS). No significant genetic link was observed between the pro-self and prosocial facets of justice sensitivity in our study. These findings are consistent with the previous view that individuals with the observer, beneficiary, and perpetrator sensitivities share a genuine concern for justice, whereas those with victim sensitivity have self-related concerns (Thomas et al., 2011). Previous studies have also shown that the pro-self facet (VS) is associated primarily with self-related concerns (such as jealousy) while the prosocial facets are associated primarily with others-related concerns (such as agreeableness; Schmitt et al., 2005, 2010). In addition, VS is negatively correlated with positive behavioral reciprocity and positively correlated with negative behavioral reciprocity, while the prosocial facets exhibited the exact opposite correlations (Baumert et al., 2014). People in collectivist cultures also displayed higher prosocial justice sensitivity as compared with people in individualist cultures (Maltese et al., 2020; Wu et al., 2014). Based on the above self–others distinction of justice sensitivity in terms of personality, behavior, and cultural values, our twin study provides novel evidence for the distinctiveness between pro-self and prosocial facets of justice sensitivity in terms of genetics. Overall, our research indicates that exploring the common genetic influences underlying different facets of justice sensitivity is important in understanding the links between pro-self and prosocial facets of justice sensitivity.

Concerning non-shared environmental effects, our study showed that non-shared environmental factors (plus measurement error) contribute to the variations in all the facets of justice sensitivity more so than genetic factors. Non-shared environmental factors are often related to individual-specific environmental factors and are not shared in a family. Previous literature suggests that, even identical twins living together, perceive and react to the same environments differently (Hanscombe et al., 2010). In addition, twins from the same family may also receive different education and have unique social networks. Thus, personal experiences and conditioning play a significant role in shaping individuals' sensitivity to injustice. Our study also showed that non-shared environmental factors (plus measurement error) contribute to the correlations between different facets of justice sensitivity (including correlations among the prosocial facets and correlations between the pro-self and prosocial facets). This discovery implies that some non-shared environmental factors influence all facets of justice sensitivity at the same time. This important finding suggests that concern for justice, which is common in the pro-self and prosocial components, is shaped by non-shared environmental factors, such as education and socialization outside the family and common social networks.

Shared environmental factors should also be considered in explaining the justice sensitivity variations. For BS and PS, the CE models are workable in our study, and the shared environmental factors were found to be predictable for BS (28%) and PS (17%). Thus, shared environmental factors often related to micro-level societies, such as family, also contribute to the variations of justice sensitivity to some extent. Although the family effect on BS and PS has not been tested, a few studies have suggested that parenting style (restrictive vs. nurturant) affects the development of inhibition-based moral orientations (Janoff-Bulman et al., 2014). Given that BS and PS are inhibition-based (Wu, 2014), for which people should not take advantage of others (as a beneficiary) or do evil (as a perpetrator), further studies are necessary to test the effects of restrictive parenting on the development of BS and PS. A more recent investigation revealed that compared with the upper class, the lower class (indicated by low family income) scored higher on prosocial justice sensitivity, especially for BS and PS (Wu et al., 2019).

This study has several limitations. First, the twin participants were young adults. Previous studies have found that older participants scored significantly higher on justice sensitivity for others and lower on justice sensitivity for self as compared with young adults (Schmitt et al., 2010). Thus, our young adult participants may not be a representative sample of the general population. Second, our study only included a Chinese sample. Recent cross-cultural studies compared justice sensitivity across countries in terms of collectivism–individualism and found that people in collectivist cultures scored higher on prosocial facets than those in individualist cultures (Maltese et al., 2020; Wu et al., 2014). Thus, our results from the Chinese sample may not necessarily be generalizable to other populations. Third, the Justice Sensitivity Inventory used in our study is a self-reported scale, which may be confounded by self-rater biases and socially desirable responses (Kandler et al., 2016; Verhulst et al., 2012). However, our finding that the pro-self facet is genetically distinct from all other facets may not be accounted for by the socially desirable responses. Future studies could adopt more objective measurement tools to validate our findings if possible. Fourth, the classic ACE model treated genetic, shared, and non-shared environmental effects as independent, which ignores the correlations or interactions between each other (Purcell, 2002). Such correlations or interactions are critical for gaining a more nuanced understanding of the origins of justice sensitivity. Future studies might include measures of environments to test for possible interactions or correlations with genes. We cannot separate the effects of non-shared environmental factors and measurement error in the genetic modeling analysis. Future researchers can control for the effect of measurement error by repeating the measures in twins and consequently boosting the power of genetic analyses (Cai et al., 2016). Lastly, our sample size was moderate, which led to wide confidence intervals for model parameters and limited statistical power to detect small genetic or environmental effects, especially shared environmental effects (Verhulst, 2017). Indeed, the magnitude of several shared environmental effects (Table 2) was below the minimal effect size (.16) in our study. Thus, future studies must investigate and further verify the heritability of justice sensitivity by extending the sample size and scope of participant pools.

Conclusion

Justice principles play a dominant role in social interaction, and justice sensitivity is important for human well-being and mental health. The origin of individual differences in justice sensitivity and the links between the different facets of justice sensitivity remains elusive. The present study is the first to partition genetic and environmental influences on justice sensitivity across the four facets (VS, OS, BS, and PS) using twin methodology and comprehensively examine the genetic associations between them. Overall, the results highlight the roles of additive genetic and non-shared environmental factors in shaping individuals' sensitivity to injustice. Genetic overlaps among prosocial concerns for others are also revealed. As the first genetic study on justice sensitivity, we believe our findings can shed light on the nature of justice sensitivity and provide additional evidence for understanding pro-self and prosocial facets of justice sensitivity.

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Conflict of Interest

The authors declare that they have no conflict of interest in this work.

Authorship

Zhou and Wu developed the research proposal, and Wang conducted the data analysis and wrote the first draft. All authors contributed to writing the full paper and its revisions.

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