Common Heritable Effects Underpin Concerns Over Norm Maintenance and In-Group Favoritism: Evidence From Genetic Analyses of Right-Wing Authoritarianism and Traditionalism

Gary J. Lewis[1] and Timothy C. Bates[2]
[1]University of York
[2]University of Edinburgh

Abstract

Research has shown that in-group favoritism is associated with concerns over the maintenance of social norms. Here we present two studies examining whether genetic factors underpin this association. A classical twin design was used to decompose phenotypic variance into genetic and environmental components in two studies. Study 1 used 812 pairs of adult U.S. twins from the nationally representative MIDUS II sample. Study 2 used 707 pairs of middle-age twins from the Minnesota Twin Registry. In-group favoritism was measured with scales tapping preferences for in-group (vs. out-group) individuals; norm concerns were measured with the Multidimensional Personality Questionnaire–Traditionalism (Study 1) and Right-Wing Authoritarianism (RWA; Study 2) scales. In Study 1, heritable effects underlying traditionalism were moderately (c. 35%) overlapping with the genetic variance underpinning in-group favoritism. In Study 2, heritable influences on RWA were entirely shared with the heritable effects on in-group favoritism. Moreover, we observed that Big Five Openness shared common genetic links to both RWA and in-group favoritism. These results suggest that, at the genetic level, in-group favoritism is linked with a system related to concern over normative social practices, which is, in turn, partially associated with trait Openness.

Studies of both implicit and explicit attitudes in diverse settings show that preferences for members of one's in-group, and negative sentiment toward out-group individuals, are widespread (Fiske, 2002; LeVine & Campbell, 1972; Sumner, 1907; Tajfel, Billig, Bundy, & Flament, 1971; Vanman, Saltz, Nathan, & Warren, 2004; Wittenbrink, Judd, & Park, 1997). While environmental factors are commonly believed to underpin in-group favoritism and out-group derogation, including parental rearing environment (Adorno, Frenkel-Brunswik, Levinson, & Sanford, 1950) or scarcity of key resources (Sherif, 1966), recent research shows that genetic factors also influence individual differences concerning in-group sentiment (Lewis & Bates, 2010; Orey & Park, 2012; Weber, Johnson, & Arceneaux, 2012). However, whether these heritable effects are specific for in-group bias or overlap with more general psychological systems is currently unclear. Here, using two independent, genetically informative samples, we test the hypothesis that genetic variation underlying in-group favoritism reflects more general differences in concerns for norm maintenance—the tendency to adopt prevailing norms and to be concerned when others violate these norms (Altemeyer, 1981; Duckitt, 2006). In addition, we examine the extent to which basic dimensions of personality—the Big Five traits—

overlap with heritable aspects of norm concerns and in-group favoritism (Study 2), in line with work positing that both of these constructs reflect basic personality traits (Ekehammar & Akrami, 2007; Sibley & Duckitt, 2008).

Intergroup Attitudes: The Role of Social Norms

Measures of intergroup attitudes necessarily assess hostile behavior and disregard for out-group individuals and positive behavior toward in-group members. Research into such attitudes must ultimately, however, account for such sentiment in terms of the traits, motivations, situations, and values that lead

to such attitudes and behavior (Allport, 1954; Altemeyer, 1981; Duckitt, 2006; Pratto, Sidanius, Stallworth, & Malle, 1994; Stephan & Stephan, 2000; Tajfel & Turner, 1979). Much of the extant research on the origins of intergroup attitudes has focused less on dispositional factors and more on contextual influences (such as resource competition; Sherif, 1966) or generalized conceptions of human social cognition (Hodson, 2009; Huddy, 2001). However, work over the last two decades or so (although also see Adorno et al., 1950; Allport, 1954) has begun to systematically develop the role of specific individual differences constructs as predictors of intergroup attitudes. Moreover, much of this work shares the common sentiment that concerns over the maintenance of social norms reflect important components in the pathway toward intergroup attitudes. Next, we introduce two major models in the field—right-wing authoritarianism and integrated threat theory—that bolster this view of social norm maintenance underpinning intergroup attitudes.

Altemeyer's Right-Wing Authoritarianism

Building from earlier work on the "authoritarian personality" (Adorno et al., 1950), Altemeyer (1981) noted that only three (of a hypothesized nine) aspects of the authoritarian personality were systematically correlated: authoritarian aggression, authoritarian submission, and conventionalism. As such, Altemeyer (1981) developed a new index of authoritarian personality—right-wing authoritarianism (RWA)—that collectively tapped the willingness to submit to authorities perceived as legitimate (authoritarian submission), the adherence to social norms (conventionalism), and the hostility and punitive sentiment toward those who do not adhere to social norms (authoritarian aggression). While some debate exists concerning the underlying psychometric structure of RWA (e.g. Mavor, Louis, & Sibley, 2010), most work to date has used a single scale score, and in this light RWA can be broadly understood as the common features of its parts: namely, concern over norms and their maintenance.

Norm Concerns and Intergroup Attitudes

Of specific interest here, a number of studies have linked RWA to a range of intergroup attitudes, spanning preferences for in-group individuals to derogation of out-group persons. For example, Gramzow & Gaertner (2005) observed that individuals high in RWA tended to show greater favoritism to a novel in-group. Similarly, Navarrete, Kurzban, Fessler, and Kirkpatrick (2004) found in a sample of U.S. citizens that high RWA predicted greater pro-American bias. Haddock, Zanna, and Esses (1993) noted that, among heterosexuals, RWA predicted negative attitudes toward homosexuals. McFarland (2010) reported a similar pattern of associations across five independent studies relating RWA to generalized prejudice (including attitudes concerning racism and sexism). Summa-

rizing findings in this literature, Sibley and Duckitt (2008) reported a meta-analysis of the association between RWA and prejudice in 10 samples totaling 2,919 subjects, noting an average correlation of .49 between the two constructs. Of further importance, links between RWA and prejudice appear to be specific to out-groups that threaten disruption to social norms (Duckitt, 2006; Duckitt & Sibley, 2010). A putative rationale for this empirical link between RWA and prejudice specific to cohesion-threatening out-groups is explained by Duckitt (2006) as follows: "RWA expresses threat-driven motivation to establish and maintain social or group security in the form of social control, order, cohesion, and stability. Consequently, persons high in RWA should dislike groups that seem to threaten societal or group security" (p. 686). RWA under this model, then, reflects a tendency to detect and reinforce normative social conventions. In turn, this tendency has been hypothesized to underlie the link from RWA to prejudice against out-groups, insomuch that such groups are perceived as challenges to prevailing social norms.

Further work in parallel literatures supports this general conclusion concerning links between intergroup attitudes and social norm violations. Integrated threat theory (ITT; Stephan & Stephan, 2000) argues that prejudice emerges through the perception of threat from an out-group. Physical threat is already predicted to increase prejudice according to realistic conflict theory (Sherif, 1966). ITT broadens the scope of threat, however, to include symbolic threat. This is defined by Stephan, Ybarra, and Rios Morrison (2009) as the threat that "tribes" with differing values pose to "the unified meaning system of the ingroup" (p. 43). ITT, then, predicts analogous links from value differences to prejudice, as proposed by Duckitt (2006) with regard to RWA.

Evidence for integrated threat theory comes from several sources. For instance, German participants who read a vignette about a (fictitious) immigrant group whose values were described as either similar to or dissimilar from those of the participants' in-group (Rohmann, Piontkowski, & van Randenborgh, 2008) showed an increased perception of symbolic threat in the dissimilar condition. Furthermore, Stephan, Ybarra, and Bachman (1999) reported that perceptions of differences in values and beliefs significantly predicted attitudes toward immigrant out-groups in student samples from Florida, Mexico, and Hawaii. Both the RWA and ITT models, then, indicate that prejudice emerges from concerns regarding prevailing social norms.

Genetic Studies of In-Group Favoritism and Norm Concerns

While work establishing the origins of intergroup attitudes has had a long history in the field (Adorno et al., 1950; Allport, 1954), only recently has research turned to the question of whether genetic factors influence such attitudes, perhaps because of prevailing views that social attitudes solely emerge

through processes of socialization (Charney, 2008; also see Alford, Funk, & Hibbing, 2005, for discussion of this point). Accordingly, research in this domain is still limited to a small set of studies; however, all such work to date has indicated that in-group favoritism is significantly influenced by heritable factors. For example, Lewis and Bates (2010) reported that in-group favoritism (identification with, and preferences for, individuals of one's own race, religion, and ethnicity) was underpinned by a common latent factor, which, in turn, was highly heritable (c. 50%). This result was recently replicated by Orey and Park (2012), who showed that ethnocentrism was significantly influenced by genetic factors, although to more modest levels (c. 20%). Of interest, neither of these studies found evidence for shared-environment effects on generalized in-group favoritism, in contrast to common conceptualizations concerning the origins of intergroup attitudes (Adorno et al., 1950), although in keeping with research on other psychological traits (Turkheimer, 2000).

Work examining the role of genetic influences on norm concerns and maintenance has also found evidence for heritable effects. Scarr and Weinberg (1981) observed that in biologically related family members, correlations on authoritarianism ranged from .34 to .46; in adoptive family members, these correlations were almost all nonsignificant (only 1 of 13 was significant), suggesting that genetic factors (vs. rearing environment) were of primary importance in determining levels of authoritarianism. Finkel and McGue (1997) found that traditionalism (from the Multidimensional Personality Questionnaire), tapping sentiment concerning adherence to social rules and conventions, contained significant heritable influences in a large sample of reared-together twins. Similarly, RWA was observed to have substantial heritable underpinnings (c. 50%; McCourt, Bouchard, Lykken, Tellegen, & Keyes, 1999).

STUDY 1

The argument outlined above suggests that in-group favoritism (at least in part) is linked with concerns over social norms. Accordingly, despite not explicitly reflecting in-group-centered attitudes and concerns, genetic factors associated with concerns for the maintenance of social norms may nevertheless show overlapping heritable effects with in-group favoritism. The plausibility of this hypothesis is further supported by work demonstrating that norm concerns are themselves heritable (Finkel & McGue, 1997; McCourt et al., 1999), and thus capable, in principle, of containing common heritable influences with in-group favoritism.

We tested this prediction using a nationally representative sample of adult U.S. twins analyzed with multivariate, multigroup structural equation modeling (see Methods for full details). This study design allowed us to not only examine the phenotypic correlation between in-group favoritism and sentiment toward social norms, but also to test whether these variables reflect shared genetic and/or environmental influences. Accordingly, here we were able to formally examine whether genetic variation influencing individual differences in normative concerns was common to the genetic variation underlying in-group favoritism.

Methods

Participants. Phenotypic data were available for 812 partial ($n = 473$) and complete ($n = 339$) pairs of twins contacted by the MacArthur Foundation Survey of Midlife Development in the United States (MIDUS II; see Brim, Ryff, & Kessler, 2004). Of the monozygotic (MZ) pairs, 149 were male ($M_{age} = 45$, $SD = 11.41$) and 163 were female ($M_{age} = 44$, $SD = 11.92$). Of the dizygotic (DZ) pairs, 106 were male ($M_{age} = 45$, $SD = 12.20$), 178 were female ($M_{age} = 46$, $SD = 11.97$), and 216 were opposite-sex pairs ($M_{age} = 46$, $SD = 11.76$). See Table 1 for full details on the number of complete pairs in each class of zygosity.

Measures. *In-group favoritism* was measured with nine items assessing strength of in-group favoritism in three domains (ethnic, racial, and religious) for each of three facets: degree of identification with the group, preference for affiliating with in-group members, and importance placed on marrying within the group (Lewis & Bates, 2010). For instance, race identification was assessed as "How closely do you identify with being a member of your racial group?" (1 = *Not at all closely*, 4 = *Very closely*). A composite score was formed as the sum of the items (Cronbach's $\alpha = .79$).

Table 1 Study 1: Univariate Modeling Results and Across-Zygosity Twin Pair Correlations for In-Group Favoritism and Traditionalism

	A	C	E	−2LL (EP)	MZ	DZ	MZm	MZf	DZm	DZf	DZos
Favoritism	.46 [.14, .62]	.07 [.00, .34]	.48 [.38, .58]	2754.53 (4)	.58 ($n = 149$)	.36 ($n = 190$)	.59 ($n = 64$)	.57 ($n = 85$)	.28 ($n = 35$)	.31 ($n = 75$)	.41 ($n = 80$)
Traditionalism	.20 [.00, .49]	.19 [.00, .41]	.61 [.50, .72]	2929.63 (4)	.44 ($n = 155$)	.31 ($n = 208$)	.39 ($n = 66$)	.47 ($n = 89$)	.30 ($n = 43$)	.40 ($n = 83$)	.26 ($n = 82$)

Note. A = additive genetic effects; C = shared-environment effects; E = unique-environment effects; −2LL = −2*log likelihood for the full ACE model; EP = estimated parameters; MZ = monozygotic pairs; DZ = dizygotic pairs; MZm = MZ male pairs; MZf = MZ female pairs; DZm = DZ male pairs; DZf = DZ female pairs; DZos = DZ opposite-sex pairs. A, C, and E parameters represent the standardized variance components for A, C, and E. The 95% confidence intervals for A, C, and E parameters, as well as sample sizes for twin correlations, are shown in brackets and parentheses, respectively.

Concern over norm maintenance was measured with the following three items from the Multidimensional Personality Questionnaire–Traditionalism scale (Patrick, Curtin, & Tellegen, 2002): "I am opposed to more censorship of books and movies because it would go against free speech" (reverse-scored), "People should observe moral laws more strictly than they do," and "I don't like to see religious authority overturned by so-called progress and logical reasoning" (1 = *False of me*, 4 = *True of me*). A composite score was formed as the sum of the items (Cronbach's α = .56).

Analysis. Prior to conducting the biometric analyses, both variables were residualized for the effects of age and sex (McGue & Bouchard, 1984), as is standard practice to avoid artificially inflating twin similarities simply because of shared sex and age. Models were estimated using full-information maximum likelihood in OpenMx 1.1 (Boker et al., 2010a, 2010b) running within R 2.13 (R Core Development Team, 2012).

The classical twin design typically partitions observed variation into three components: additive genetic influences (A), shared-environment influences (C), and unique-environment influences (E). Genetic effects are inferred when monozygotic twins are more similar than dizygotic twins, whereas shared-environment effects are inferred when MZ twin correlations are less than twice that of the DZ twins (Neale & Cardon, 1992). Unique-environment effects are inferred when MZ twin correlations are less than at unity, and thus this variance component also includes measurement error.

These inferences from the classical twin design rest upon specific assumptions that, if violated, may bias estimated parameters (Plomin, Defries, Knopik, & Neiderhiser, 2013). These assumptions can be tested and include the presence of equal environments across zygosities (which justifies treating the differences in twin correlations between the two zygosities as reflecting shared genetic rather than shared environmental influences) and a lack of assortative mating (which will cause the genetic sharing among DZ twins to vary above the assumed 50% default used in twin modeling). Assortative mating can be tested by using measured assortment on the trait in consideration (e.g., Kandler, Bleidorn, & Riemann, 2012). The equal environments assumption can be tested in several ways, such as by examining whether self-reported similarities in parental treatment are associated with increased similarity on the trait of interest (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Scarr & Carter-Saltzman 1979).

This design can be extended to the bivariate case such that not only can A, C, and E components for individuals traits be estimated (i.e., for traditionalism *or* in-group favoritism), but also correlations between these variance components for pairs of traits (i.e., for traditionalism *and* in-group favoritism). As such, genetic (or environmental) correlations can be derived that reflect the magnitude of shared heritable (or environmental) influences underlying a given pair of traits. It is important to emphasize that relationships occurring at the underlying genetic and environmental levels can differ greatly from the observed or phenotypic correlation, even differing in sign. That is, an observed phenotypic correlation of, say, .50 between two heritable variables may be entirely genetic in origin or involve no shared genetic effects at all. The genetic correlation, in turn, reflects the proportion of genes shared between two constructs. Thus, a genetic correlation of 1.0 implies that all the genes influencing trait A also influence trait B, and vice versa. Conversely, a genetic correlation of 0 implies that no genes affect more than one of the traits. Importantly, a genetic correlation of 1.0 does not imply that all of the phenotypic correlation is genetic and, indeed, is compatible with any proportion of the total phenotypic correlation being genetic in origin. Computationally, the genetic correlation is computed by pre- and post-multiplying the A matrix by the inverse of the standard deviation of **A** (i.e., the square root of $\mathbf{I} \times \mathbf{A}$, where **I** is an n row identity matrix):

$$\sqrt{I \times A}^{-1} A \sqrt{I \times A}^{-1}$$

Results

The phenotypic correlation between in-group favoritism and traditionalism (based on one individual in a pair taken at random) was .44 ($p < .001$). MZ correlations were higher than for DZ pairs on both measures, implying the presence of genetic influences on variation in each measure (see Table 1). Phenotypic variances and means were not significantly different across zygosity and twin order. Tests of sex differences (scalar and general sex-limitation effects) were not significant, and thus sexes were pooled for subsequent analyses.

We next examined the heritability of traditionalism and in-group favoritism in univariate models (see Table 1). In-group favoritism showed significant genetic influences and was most parsimoniously explained by a model containing only additive genetic and unique-environment effects (shared-environment effects could be removed without significantly worsening model fit: $\Delta\chi^2(1) = 0.20$, $p = .60$; but not additive genetic effects: $\Delta\chi^2(1) = 7.92$, $p < .001$): Additive genetic and unique-environment effects explained 53% and 47% of the variance in in-group favoritism, respectively. By contrast, the traditionalism measure was equally well explained by both AE and CE models: Additive genetic and shared-environment effects were indistinguishable. These effects could be dropped separately, $\Delta\chi^2(1) = 1.45$, $p = .23$, and $\Delta\chi^2(1) = 1.68$, $p = .20$, respectively, but not simultaneously, $\Delta\chi^2(2) = 47.74$, $p < .001$, and so both components were retained in the final model. The final model (ACE) explained 20%, 19%, and 61% of the phenotypic variance, respectively.

We next moved to a test of our core hypothesis, namely, that genetic influences on in-group favoritism would be shared with influences on traditionalism. The genetic correlation between traditionalism and in-group favoritism was estimated as $r = .59$. This path, however, could be removed from the model without significantly worsening fit, $\Delta\chi^2(1) = 1.89$,
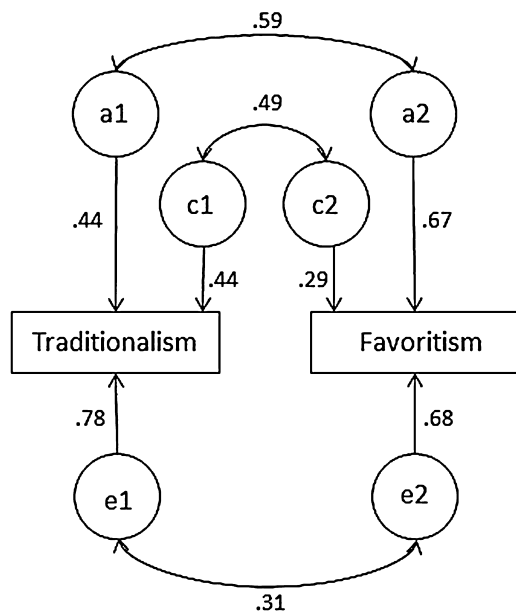
Figure I Study 1: Final model for traditionalism and in-group favoritism (parameter estimates are standardized). a = additive genetic effects; c = shared-environment effects; e = unique-environment effects. The −2 log likelihood for full model = 5,527.58 (estimated parameters = 11).

$p = .17$. This was also the case for the correlation between shared-environment effects, $r = .49$; $\Delta\chi^2(1) = 0.33$, $p = .57$. Of importance, however, dropping these paths simultaneously resulted in dramatic worsening of fit, $\Delta\chi^2(2) = 29.06$, $p < .001$, indicating that familial effects underpin the phenotypic correlation between traditionalism and in-group favoritism, but that additive genetic and shared-environment influences were not able to be formally distinguished in the current sample. Finally, we tested whether the shared unique environments accounted for a significant portion of the association between traditionalism and favoritism. This path was significant: Dropping the unique-environment correlation dramatically lowered model fit, $\Delta\chi^2(1) = 19.17$, $p < .001$. As such, we retained all three sources of covariation, which are presented in the final (unreduced) model (see Figure 1).[1]

Brief Discussion

Supporting previous work (Sibley & Duckitt, 2008), Study 1 showed that traditionalism and in-group favoritism were significantly associated at the phenotypic level. Both traditionalism and in-group favoritism were individually heritable, with evidence for shared-environment effects also underlying traditionalism. The multivariate results indicated a moderate-to-large genetic correlation between in-group favoritism and traditionalism, supporting our initial hypotheses, although it is noteworthy that this genetic association was not itself significant at the nominal level; of note, however, this association

could not be removed from the model when the shared-environment effects common to traditionalism and in-group favoritism were absent, indicating that familial influences underpin both of these measures. Accordingly, this finding of genetic links between traditionalism and in-group favoritism should be interpreted with caution. We also found evidence that unique-environment effects contributed to the overlap of traditionalism and favoritism.

STUDY 2

The findings of Study 1 provided support, albeit qualified (see below), for our initial hypotheses that heritable bases underlying concern for norm maintenance overlap with in-group favoritism. However, the traditionalism measure was only partially associated with the heritability of in-group favoritism ($r = .59$). This may reflect a theoretical need for additional genetic explanations of in-group favoritism. In addition, while a moderate-to-large genetic correlation between traditionalism and in-group favoritism was observed, this correlation was not significant at the nominal level (although it could not be removed from the model simultaneously with the equivalent shared-environment correlation). This finding may reflect limitations in power as afforded by sample size. Accordingly, we next sought to establish the veracity of this heritable link underlying norm concerns and in-group favoritism with an independent study sample, utilizing a more reliable and well-understood instrument of norm attitudes: right-wing authoritarianism (RWA; Altemeyer, 1981; Zakrisson, 2005).

In addition, the results of this study suggest additional interesting questions. In particular, if traditionalism and authoritarianism show, at least in part, heritable links with in-group favoritism, then does this common genetic basis reflect a specific mechanism reflecting conventionality, deference, and a dislike of deviance, or rather the action of more general traits, such as those indexed by the Big Five dimensions of personality? The Big Five personality domains are each known to be heritable (e.g., Bouchard, 2004) and, jointly, are thought to form a comprehensive high-level account of personality (Costa & McCrae, 1992), supporting the possibility that genetic covariation may exist between these personality variables and our measures of RWA and in-group favoritism. Moreover, Ekehammar and Akrami (2007) observed significant negative associations from facets of Openness and Agreeableness to in-group favoritism, and Duckitt and Sibley (2010) note that "RWA seems to be determined by the socialized belief that the social world is a dangerous and threatening place, *and* by the personality construct of social conformity (or Big Five low Openness and high Conscientiousness)" (p. 585, emphasis added; see also Sibley & Duckitt, 2008). Accordingly, we also conducted a further set of analyses to address the hypothesis that genetic variance in basic personality traits influences both RWA and in-group favoritism.

Methods

Participants. Phenotypic data were available for an independent U.S. sample of 707 partial ($n = 259$) and complete ($n = 448$) pairs of twins assessed for right-wing authoritarianism and in-group favoritism, among other measures. Of the monozygotic (MZ) pairs, 163 were male ($M_{age} = 56$, $SD = 2.44$) and 247 were female ($M_{age} = 56$, $SD = 2.50$). Of the dizygotic (DZ) pairs, 94 were male (mean age = 57, $SD = 2.61$), 203 were female ($M_{age} = 56$, $SD = 2.49$). No opposite-sex pairs were available for analysis. Only participants who self-reported as Caucasian and non-Muslim were used in the current analyses. See Table 2 for full details on the number of complete pairs in each class of zygosity.

Measures. *In-group favoritism* was measured by a series of "feeling thermometer" rating questions in which participants were asked to indicate how warm or cold they felt toward specific groups on a scale from 0 to 10. Participants rated four out-groups (African Americans, Hispanics, Muslims, and Asians) using this scale. In-group favoritism scores were created by subtracting warmth toward White individuals from each of the out-group scores to obtain a relative measure, and then summing the four values (Cronbach's $\alpha = .88$): Higher scores represent less warmth toward out-groups relative to one's warmth toward members of their own race. Feeling thermometers have been used as an explicit index of in-group bias in several studies (e.g., Hugenberg & Bodenhausen, 2004; Miller, Smith, & Mackie, 2004).

Right-wing authoritarianism scores were obtained using a short version of the Right-Wing Authoritarianism (RWA) Scale (Zakrisson, 2005). The short RWA Scale utilized in this study contains 15 items, including "The 'old-fashioned ways' and 'old-fashioned values' still show the best way to live" and "Facts show that we have to be harder against crime and sexual immorality, in order to uphold law and order." Participants were asked to rate themselves on each item using a 7-point Likert scale (1 = *strongly disagree*, 7 = *strongly agree*). Individuals' RWA score were created by summing the 15 items, reversing items where appropriate (Cronbach's $\alpha = .87$).

Big Five personality domain ratings were obtained using the Big Five Inventory (BFI; John, Donahue, & Kentle, 1991;

John, Naumann, & Soto, 2008). The version of the BFI utilized in this study contains two 8-item scales that measure Extraversion and Neuroticism, two 9-item scales that measure Agreeableness and Conscientiousness, and a 10-item scale for Openness. Participants rated themselves on each item using a 5-point Likert scale ranging from 1 (*Very uncharacteristic of myself*) to 5 (*Very characteristic of myself*).

Analysis. Prior to conducting the biometric analyses, all variables were residualized for the effects of age and sex (McGue & Bouchard, 1984). Models were estimated using full-information maximum likelihood in OpenMx 1.3.2 (Boker et al., 2010a, 2010b) running within R 2.15 (R Core Development Team, 2012).

In tests of genetic and environmental association between RWA and in-group favoritism, analyses were identical to those described in Study 1. Tests of whether heritable effects on RWA and in-group favoritism were overlapping with heritable effects underlying the Big Five measures of personality were performed using a multivariate Cholesky model with Extraversion, Agreeableness, Conscientiousness, Neuroticism, Openness, RWA, and in-group favoritism (entered in that order; see Figure 2). This model specifies as many latent factors as there are variables for each source of variance, with each subsequent factor having one fewer pathway than the preceding factor. Thus, for the first additive genetic latent factor loads on all of the *n* measured variables, the subsequent latent factors load on *n*-1, *n*-2 . . . n-*j* variables. In this way, each factor accounts for as much of the remaining variance as possible, until the last factor accounts only for residual variance (if any) in the last measured variable. This is repeated for shared-environment (C) and unique-environment factors (E). The multivariate Cholesky allows one to examine whether genetic and/or environmental covariance is present between multiple variables simultaneously.

Results

The phenotypic correlation between in-group favoritism and RWA was .29 ($p < .001$), based on one individual from each pair selected at random. MZ correlations were notably higher than for DZ pairs for both measures, implying the presence of

Table 2 Study 2: Univariate Modeling Results and Across-Zygosity Twin Pair Correlations for In-Group Favoritism and Right-Wing Authoritarianism

	A	C	E	−2LL (EP)	MZ	DZ	MZm	MZf	DZm	DZf
Favoritism	.19 [.00, .28]	.00 [.00, .21]	.81 [.72, .90]	3567.30 (4)	.22 ($n = 337$)	.08 ($n = 220$)	.27 ($n = 135$)	.17 ($n = 202$)	.03 ($n = 79$)	.10 ($n = 141$)
RWA	.49 [.22, .67]	.12 [.00, .36]	.39 [.32, .46]	3093.65 (4)	.61 ($n = 278$)	.36 ($n = 177$)	.69 ($n = 109$)	.56 ($n = 169$)	.43 ($n = 64$)	.32 ($n = 113$)

Note. A = additive genetic effects; C = shared-environment effects; E = unique-environment effects; −2LL = −2*log likelihood for the full ACE model; MZ = monozygotic pairs; DZ = dizygotic pairs; MZm = MZ male pairs; MZf = MZ female pairs; DZm = DZ male pairs; DZf = DZ female pairs; RWA = right-wing authoritarianism. The A, C, and E parameters represent the standardized variance components for A, C, and E. The 95% confidence intervals for A, C, and E parameters, as well as sample sizes for twin correlations, are shown in brackets and parentheses, respectively.
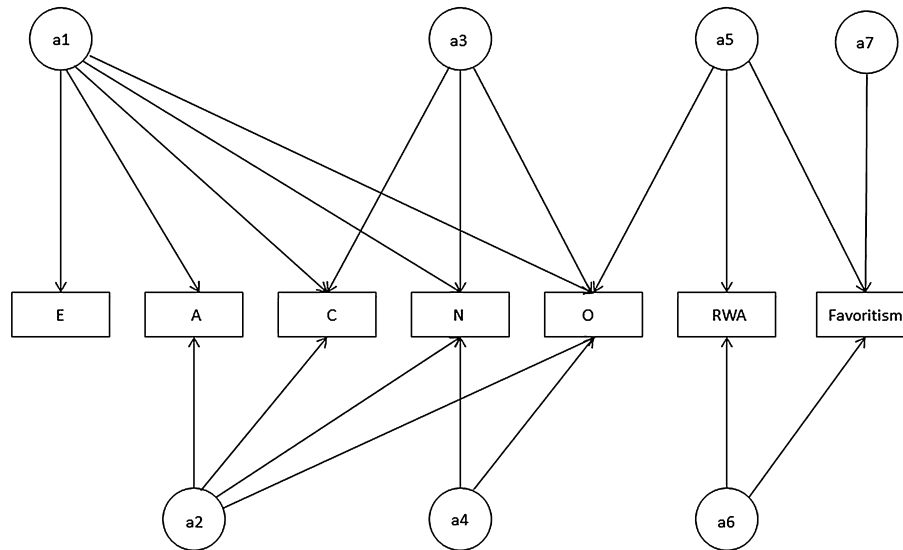
Figure 2 Figurative example of the Cholesky decomposition. Only additive genetic effects are displayed here so as to enhance clarity: shared- and unique-environment effects were also modeled in the analyses; a = additive genetic effects; E = Extraversion; A = Agreeableness; C = Conscientiousness; N = Neuroticism; O = Openness; RWA = right-wing authoritarianism; Favoritism = in-group favoritism.

genetic influences on variation in each trait (see Table 2). Phenotypic variances and means were not significantly different across zygosity and twin order. Scalar sex-limitation effects were not significant (male and female DZ pairs could be equated without significant loss of model fit for any of the variables: Opposite-sex pairs were not available to test for non-scalar sex limitation). Thus, males and females were pooled for subsequent analyses.

We next examined the heritability of each of our variables in univariate models (see Table 2). RWA showed significant genetic influences and was most parsimoniously explained by a model containing only additive genetic and unique-environment effects (shared-environment effects could be removed without significantly worsening model fit: $\Delta\chi^2(1) = .78$, $p = .38$; additive genetic effects were significant: $\Delta\chi^2(1) = 14.11$, $p < .001$): Additive genetic and unique-environment effects explained 61% and 39% of the variance, respectively, in RWA. In-group favoritism was equally well explained by AE and CE models. Additive genetic and shared-environment effects could each be dropped separately, $\Delta\chi^2 = 1.74$, $p = .19$, and $\Delta\chi^2 = 0$, $p = 1$, respectively, but not simultaneously, $\Delta\chi^2(2) = 16.78$, $p < .001$. The full model for in-group favoritism estimated A, C, and E components as explaining 19%, 0%, and 81% of the variance, respectively.

We next moved to a test of our first core hypothesis, namely, that genetic influences on in-group favoritism would be shared with genetic influences on RWA. In a bivariate model including in-group favoritism and RWA, the genetic correlation between traditionalism and in-group favoritism was estimated as 1.0, with a single latent genetic factor accounting for the heritability of both traits (i.e., the second latent genetic factor was estimated at zero). Moreover, the genetic correlation between RWA and in-group favoritism could not be dropped without significantly worsening fit, $\Delta\chi^2(1) = 11.27$, $p < .001$, formally indicating that genetic effects underlying RWA are common, with heritable variation underpinning in-group favoritism. This contrasted with the effect found for shared-environment effects; here, the correlation between in-group favoritism and RWA could be dropped with no significant change in fit, $\Delta\chi^2(1) = 2.21$, $p = .14$. Finally, we tested whether the unique-environment effects accounted for a significant portion of the association between traditionalism and favoritism. As in Study 1, removing the unique-environment correlation significantly lowered model fit, $\Delta\chi^2(1) = 9.13$, $p < .01$. The full model is presented in Figure 3.

Phenotypic and Biometric Associations: Big Five Traits, RWA, and In-Group Favoritism. We next tested whether Big Five personality domains showed phenotypic links to RWA and in-group favoritism. Two linear regression models were constructed, each using the entire sample (i.e., both individuals, where available, in each of the twin pairs to maximize sample size and power). The first was fitted with RWA as the dependent variable and the second with in-group favoritism; each of the Big Five domains was entered as an independent predictor. For RWA, significant effects were found for Extraversion ($\beta = .13$, $p < .01$), Conscientiousness ($\beta = .09$, $p < .01$), and Openness ($\beta = -.29$, $p < .01$). Neither Agreeableness nor Neuroticism showed significant effects ($\beta = .04$, $p = .24$, and $\beta = .03$, $p = .41$, respectively). For in-group favoritism, significant effects were found for Agreeableness ($\beta = -.08$, $p < .01$) and Openness ($\beta = -.20$, $p < .01$), with a marginal effect for Neuroticism ($\beta = .06$, $p = .06$). Neither Conscientiousness nor Extraversion showed significant effects ($\beta = .05$, $p = .12$, and
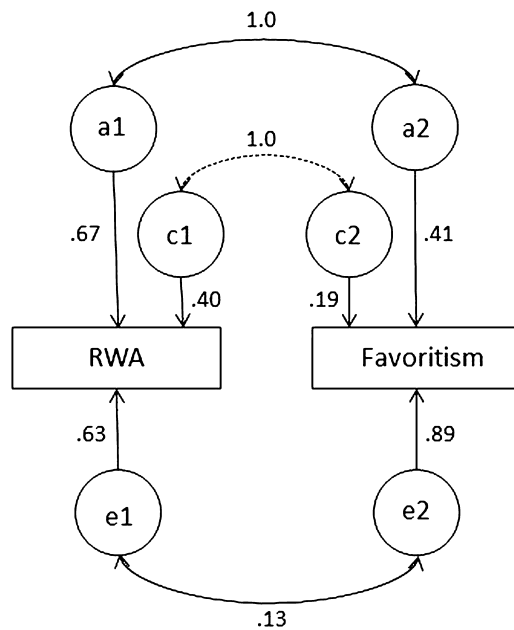
Figure 3 Study 2: Final model for RWA and in-group favoritism (parameter estimates are standardized). a = additive genetic effects; c = shared-environment effects; e = unique-environment effects; RWA = right-wing authoritarianism. The −2 log likelihood for full model = 6,565.09 (estimated parameters = 11); dashed line = $p > .05$.

$\beta = .04$, $p = .21$, respectively). The significant association between (low) Openness and both RWA and in-group favoritism supports previous work (Ekehammar & Akrami, 2007; Sibley & Duckitt, 2008). The association between Conscientiousness and RWA supports work by Sibley and Duckitt (2008), although it was not found by Ekehammar and Akrami (2007). These latter authors did, however, find significant support for higher Agreeableness in RWA, whereas we, like Sibley & Duckitt (2008), did not. The positive link between Extraversion and RWA found here is comparable to findings reported by Ekehammar, Akrami, Gylje, and Zakrisson (2004).

We next moved to a test of our genetic hypothesis; namely, that heritable bases underlying Big Five personality traits are significantly associated with the heritable bases underpinning RWA and in-group favoritism. To do this, we built a saturated Cholesky model, as described above. We first tested whether the shared-environment effects could be removed from the model without significantly worsening fit. Most of these paths were small (e.g., 18 of 27 were < .10) and in sum were not significant, $\Delta\chi^2(28) = 8.54$, $p = .99$. One shared-environment path, however (that to RWA), was unusually large (.39); accordingly, we tested this path separately. Removing the 27 smaller shared-environment paths did not, unsurprisingly, worsen fit, $\Delta\chi^2(27) = 5.43$, $p = .99$. The subsequent removal of the shared-environment path to RWA alone, though, caused a marginally significant decrement in fit, $\Delta\chi^2(1) = 3.11$, $p = .078$. Given the low power to detect shared-environment effects (Neale & Cardon, 1992), together with suggestive evi-

dence from previous studies for shared-environment effects on RWA (Bouchard, 2004), we allowed this effect to remain in subsequent analyses so as not to constrain the model into unlikely solutions. For the sake of completeness, however, we also present analyses following having dropped this path (see below).

Subsequent Analyses With Shared-Environment Effects Only on RWA. We next tested the specific path (a7) to in-group favoritism. This specific genetic influence on favoritism could be dropped without significant loss of fit, $\Delta\chi^2(1) = 1.57$, $p = .21$, suggesting here that, in line with bivariate results, heritable effects on in-group favoritism could be wholly accounted for by the other variables in the model. We then tested whether RWA and in-group favoritism could be wholly understood within the genetic space of the Big Five; however, removing the remaining specific genetic effects (a6) on RWA and favoritism resulted in a significant loss of fit, $\Delta\chi^2(2) = 13.08$, $p = .001$. Having established that heritable effects on RWA and favoritism could not be understood as isomorphic with Big Five traits, we next examined whether they could be set independent of the Big Five, and, if not, which specific Big Five traits showed genetic association with these social values. The genetic links from Openness to RWA and favoritism could not be dropped without significant loss of fit, $\Delta\chi^2(2) = 15.55$, $p < .001$. In contrast, the genetic links from Extraversion, Agreeableness, Conscientiousness, and Neuroticism latent variables to RWA and favoritism could each be removed without significantly reducing model fit, all $\Delta\chi^2(1) < 2.42$, $p > .12$, and, the net effect of losing these paths as a group was also not significant, $\Delta\chi^2(8) = 11.23$, $p = .19$. As such, these paths were removed from subsequent analyses. We tested no further paths.

Subsequent Analyses With No Shared Environment. As noted above, modest shared-environment effects on RWA are likely to be present (Bouchard, 2004), but we are underpowered to detect them in the current study. For this reason, we analysed the data taking into account their effect. However, in the interests of completeness and for comparison with these results with a model in which shared environment is specified to have zero effect, we next repeated the analyses having first set all shared-environment paths to zero. These analyses gave identical results for the role of personality on RWA and favoritism, but they differed in the genetic architecture required to account for variance in favoritism: Here a unique additive genetic path (a7) to in-group favoritism was required to adequately explain favoritism, $\Delta\chi^2(1) = 6.46$, $p = .01$.

These two analytical routes give rise to the important question regarding which model should be retained as our final model. Our preference lies with the model allowing a shared-environment effect on RWA, given the presence of shared-environment influences (e.g., Bouchard, 2004). Removing shared-environment effects prior to testing genetic paths will have constrained this shared-environment influence on RWA to
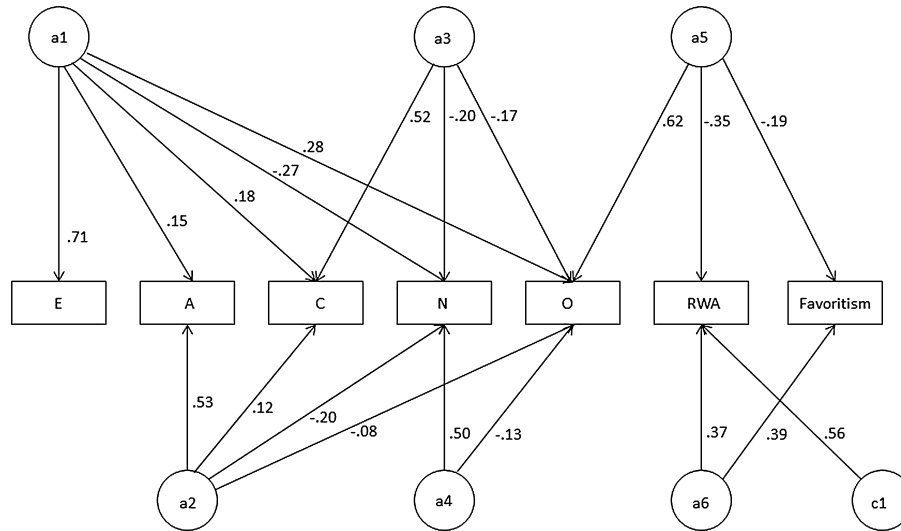
Figure 4 Study 2: Final model of additive genetic effects and shared-environment effects (standardized path coefficients) for Big Five personality traits, RWA, and in-group favoritism. a = additive genetic effects; c = shared-environment effects; E = Extraversion; A = Agreeableness; C = Conscientiousness; N = Neuroticism; O = Openness; RWA = right-wing authoritarianism; Favoritism = in-group favoritism. Squaring the path coefficients provides the proportion of trait variance accounted for by the latent factor; −2 log likelihood of the full (unreduced) model = 2,3283.23.

Table 3 Study 2: Final Model of Unique-Environment Effects (Standardized Path Coefficients) for Big Five Personality Traits, RWA, and In-Group Favoritism

	e1	e2	e3	e4	e5	e6	e7
E	.70						
A	.08	.83					
C	.13	.29	.76				
N	−.20	−.28	−.08	.69			
O	.23	.10	.12	.02	.64		
RWA	.02	.03	.07	.02	−.07	.64	
Favoritism	−.09	−.10	.03	.05	−.09	.12	.88

Note. e = unique-environment effects; E = Extraversion; A = Agreeableness; C = Conscientiousness; N = Neuroticism; O = Openness; RWA = right-wing authoritarianism; Favoritism = in-group favoritism.

Table 4 Partitioning of the Total Phenotypic Variance of RWA and In-Group Favoritism Into Genetic and Environmental Influences That Are Common With Personality, and Genetic and Environmental Influences That Are Specific to RWA and In-Group Favoritism

	Shared With Personality (%)			Unshared With Personality (%)		
	A	C	E	A	C	E
RWA	12	0	1	14	31	41
Favoritism	4	0	3	15	0	79

Note. RWA = right-wing authoritarianism; A = additive genetic effects; C = shared-environment effects; E = unique-environment effects.

Brief Discussion

Study 2 broadly replicated the findings of Study 1 in an independent sample of adult twins, using different measurement instruments to capture concerns over norm conformity (RWA) and in-group favoritism: Genetic effects underpinning RWA were identical to the heritable bases of in-group favoritism. This provides clear support for the central theory that maintenance of group norms and protocols is associated with in-group favoritism at a genetic level. Moreover, in line with the results of Study 1, we observed a significant overlap between the unique-environment effects on RWA and in-group favoritism.

One question that arises at this juncture concerns why favoritism was wholly accounted for by RWA, whereas only partially by traditionalism. One possibility is that RWA captures a broader set of attitudes concerning norm maintenance. Most notably, the measure of traditionalism used in Study 1 does not tap attitudes on aggression against norm transgressors

be explained solely via additive genetic paths. In turn, if RWA and favoritism share wholly overlapping genetic influences, but we force our model to explain this covariance with the RWA latent genetic effects actually containing both genetic and shared-environment influences, heritable effects on favoritism will not be able to be wholly explained by this RWA latent factor: And thus we will find evidence for a unique latent additive genetic effect on favoritism, but only as a consequence of decisions in our analytical steps. Accordingly, we favor our chosen final model (including a shared-environment path to RWA) as detailed in Figure 4 and Table 3. In Table 4, we detail the percentages of the total phenotypic variance in RWA and in-group favoritism that show common genetic and environmental influences with personality, as well as the percentage of genetic and environmental influences that are specific to RWA and in-group favoritism.

(RWA includes items such as "Our country needs a powerful leader, in order to destroy the radical and immoral currents prevailing in society today"), and this additional aspect may explain discrepancies between Studies 1 and 2. It is also possible that our different measures of favoritism across studies led to this differences; however, this seems less likely as both measures emphasized preferences for in-group members (rather than out-group derogation per se) and tapped fairly generalized aspects of favoritism (i.e., multiple-target out-groups).

The findings of Study 2 also supported phenotypic and genetic associations of Big Five personality traits and both RWA and in-group favoritism. At a phenotypic level, the results were similar to those reported by Sibley and Duckitt (2008), with significant links from both RWA and favoritism to (low) Openness. More modest links were observed between RWA and both Conscientiousness and Extraversion, and between in-group favoritism and (low) Agreeableness. Genetic factors influencing Openness were (in part) shared with the heritable variation underlying RWA and in-group favoritism. Notable, however, was the observation of significant independent genetic effects on RWA and in-group favoritism outside of the heritable bases of the Big Five: Analyses using more focused personality instruments, such as at the facet level of analysis, will now be valuable in determining the extent to which the heritable effects on RWA and favoritism reside outside the remit of the Big Five.

GENERAL DISCUSSION

The two studies presented here sought to test whether there are shared genetic influences on both concerns for norm maintenance and in-group favoritism, as well as whether this common genetic etiology is further shared with basic personality traits. In Study 1, we found a significant familial overlap between traditionalism and in-group favoritism: In this study, both genetic (.59) and shared-environment (.49) correlations were large, and fit decreased significantly if both (but neither on their own) were removed from the model. To gain further information about this common etiology, we conducted a second study (Study 2), using a different measure of concern for norm maintenance (right-wing authoritarianism: RWA) and in-group favoritism (an affective thermometer scale), again examining whether genetic links between these constructs were present. In this study, we observed that genetic effects underlying in-group favoritism were identical to those heritable influences on RWA. Jointly, then, Studies 1 and 2 were consistent with our predictions of a significant shared genetic etiology underpinning concerns for norms and in-group favoritism. In Study 2, we also extended these findings, demonstrating that Big Five personality traits exhibit modest but significant phenotypic and genetic influences on both RWA and in-group favoritism. We interpret this set of results, in sum, as indicating that in-group favoritism, both at the phenotypic and the genetic level, overlaps strongly with motivations for

adhering to social norms and ensuring that others do so also. Furthermore, this mechanism for norm adherence, in turn, appears to be a complex system, with its origins, in part, stemming from basic dimensions of personality, particularly Openness.

While these results indicate a shared genetic etiology underpins concerns over norm maintenance and in-group favoritism, the significant unique environmental overlap observed in both Studies 1 and 2 indicates that the commonality among these traits is not exclusively genetic in origin. These effects are intriguing because they suggest environmental exposure may underlie the association between norm concerns and in-group favoritism, although the current data cannot speak to the causal nature of this association. Further experimentation testing whether exposure to environments favoring the inculcation of norms increases in-group favoritism or vice versa is recommended.

These findings raise questions about the characteristics of the biological substrates that generate these attitudes and behaviors. Functional neuroimaging studies have provided some relevant insights on this issue. For instance, in both Black and White individuals, increases in amygdala activation have been reported in response to viewing faces of out-group versus in-group members (Hart et al., 2000; see also Cunningham et al., 2004). Of importance, however, Schreiber and Iacoboni (2012) report that amygdala activation may not represent the in-group/out-group distinction per se. These authors found that the same increases in amygdala activity could be prompted by viewing photographs of norm-violating in-group members. And the activation elicited by norm-violating in-group members exceeded that elicited by norm-adhering out-group members. These results support the current findings, suggesting that in-group favoritism may reflect concerns about appropriate social conduct rather than group membership per se, and indicate that variation in amygdala function (or neural circuits functionally linked to the amygdala) may reflect common genetic influences on norm adherence and in-group favoritism. This possibility should be examined in future work.

LIMITATIONS AND FUTURE RESEARCH DIRECTIONS

Specific recommendations for future research are apparent from the observations of the current studies. Firstly, while our two samples were large relative to much individual differences research, the detection of modest effects requires substantial sample sizes in twin designs (Visscher, 2004). Our inability, then, to discriminate between common genetic and shared-environment influences on traditionalism and in-group favoritism in Study 1 is indicative of a need for future work using larger samples, but perhaps also for more complex genetic relationships to be modeled, so as to allow a more detailed understanding of transmission mechanisms (Eaves, 2009).

Secondly, genetic correlations are subject to the same criticism as phenotypic correlations; namely, the direction of causality cannot be determined without additional experimental manipulation or contextual information. As such, while the current work supports a model in which genetic variation in norm concerns is primary to the genetic effects on in-group favoritism (as suggested by recent research; e.g., Duckitt, 2006), it is also possible that the direction of causality operates in reverse. Future work establishing the causal bases of the association between norm concerns and in-group favoritism will be of high value.

Thirdly, previous work has indicated that social dominance orientation (SDO) is linked to intergroup attitudes (Pratto et al., 1994), whereas other work implicates empathy and principled moral reasoning in (lower) levels of in-group favoritism (McFarland, 2010). Importantly, these traits can predict intergroup attitudes independently of RWA (McFarland, 2010). While we observed complete overlap between the genetic influence on norm maintenance and in-group favoritism in Study 2, the results of Study 1 indicated that residual genetic effects on in-group favoritism were evident even after accounting for genetic influences on traditionalism. As noted above, the additional coverage of the longer and more reliable measure of RWA (vs. traditionalism) may have accounted for this discrepancy between studies, although we cannot definitely rule out the possibility that the affective thermometer measure of in-group favoritism in Study 2 captured a narrower index of in-group favoritism. It is also possible that our less reliable measure of traditionalism in Study 1 contributed to these discrepancies across studies (although, notably, using a latent variable of traditionalism did not alter our results). Future work, then, on the genetic origins of in-group favoritism supplemented with a measure of SDO and a multidimensional assessment of in-group favoritism would be invaluable to further determining the genetic architecture of in-group/outgroup sentiment.

Fourthly, while the roles of the Big Five personality traits in explaining RWA and in-group favoritism were mostly modest, with a large effect found only for Openness, important variation in personality traits is evident at levels below the Big Five (DeYoung, Quilty, & Peterson, 2007), with recent work highlighting that specific subdomains of Big Five traits are predictive of relevant social attitudes, even when higher-order domains show nonsignificant effects (Hirsh, DeYoung, Xu, & Peterson, 2010). Moreover, higher-order traits beyond the remit of the Big Five have also shown links with social attitudes (Lee, Ashton, Ogunfowora, Bourdage, & Shin, 2010). Accordingly, further genetically informative research examining the contribution of facet-level personality constructs, as well as broader personality domains, is recommended.

Fifthly, our indices of in-group favoritism were explicit, self-report measures; however, much research has shown that implicit attitudes may not necessarily reflect explicit attitudes (Greenwald & Banaji, 1995). Future work that seeks to determine, firstly, whether heritable effects on explicit in-group attitudes are associated with such effects on implicit in-group attitudes and, secondly, whether heritable effects on implicit in-group attitudes overlap with the heritable bases of norm concerns will be valuable. In addition, multi-rater approaches (e.g., spouse, peer, parent) can also overcome potential biases in self-report measures, as demonstrated in recent twin research (Kandler et al., 2012).

Sixthly, although we suggest here that RWA and traditionalism index concerns over norm maintenance, this is not the only interpretation present in the literature: For instance, Altemeyer (1998) suggests that RWA is a core personality variable, whereas Akrami and Ekehammar (2006) argue that RWA is best understood as a surface personality variable.

Finally, the classical twin design used here rests upon certain assumptions, such as equal rearing environments across zygosity and the absence of gene-environment correlation and interaction (Plomin et al., 2013). The violation of these assumptions may bias parameter estimates. Additional studies are warranted, therefore, to test possible active, passive, or evocative gene-environment correlations and other more complex mediations of genetic effects across development (Plomin et al., 2013). In addition, measurement error will inflate estimates of unique-environment effects.

CONCLUSION

In summary, over two studies, utilizing two independent samples of adult twins, we demonstrated that concerns for norm maintenance—as reflected in right-wing authoritarianism and traditionalism—and in-group favoritism showed a largely overlapping genetic basis. An element of these shared genetic effects on norm concerns and favoritism was, in turn, common with the heritable bases of Openness. Future work should attempt to establish the neurobiological bases of these shared genetic influences, as well as the nature of the causal relations between these variables.

Note

1. Following the advice of an anonymous reviewer, we also analyzed our data using a latent factor of traditionalism. The results were very closely matched to those reported here for the traditionalism summed scale, with no substantive differences in any of the performed tests.

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