A common heritable factor influences prosocial obligations across multiple domains

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Although it has been shown that prosocial behaviour is heritable, it has not yet been established whether narrower aspects of pro-sociality are heritable, nor whether a common mechanism influences prosociality across its multiple domains. Here, we examine civic duty, work-place commitment and concern for the welfare of others with a study of prosocial obligations in 958 adult twin-pairs. Multivariate modelling indicated the existence of genetic factors underlying general prosocial obligations in females, with famil-ial effects (genetic and shared-environment effects were indistinguishable) influencing this general mechanism in males. At the domain-specific level, modest genetic effects were observed in females for civic and work obligations, with shared-environ-ment effects influencing welfare obligations. In males, genetic influences were observed for welfare obligation, with unique environments affecting work and civic duty.

1. INTRODUCTION

The ability to behave in a prosocial manner is a prerequi-site for large-scale social living. The presence of empathy and helping behaviour even in infancy [1] is suggestive of an innate capacity to behave in a prosocial manner. Furthermore, twin studies capable of decomposing behaviour into additive genetic, shared- or family environmental, and unique-environmental influences have shown heritable components for both empathy [2] and prosociality [3–5] (but see [6]) with evidence for cross-culture generality in these effects [4].

While a biological basis for prosociality has been established, important domains of prosociality, such as civic obligations, obligations in work [7] and redis-tribution of wealth/welfare obligation [8], have not previously been addressed in a genetically informative study. In particular, it is unclear whether these specific prosocial obligations are, first, individually heritable, and, second, are underpinned by a single, genetically influenced, mental faculty [9].

To address these issues, we report a twin study examining the genetic and environmental structure of self-reported prosocial obligations across the three

domains mentioned above. We hypothesized that a common mechanism underpinned each of the measu-red obligations, and that this common factor would be genetically influenced, in line with research demon-strating significant positive correlations between broad constellations of prosocial actions [10,11] and previous work demonstrating aspects of prosociality contain heritable variance [3–5].

2. MATERIAL AND METHODS

(a) Participants

Phenotypic data were available for 958 pairs of twins contacted by the MacArthur Foundation Survey of Midlife Development in the United States [12]. Of the monozygotic (MZ) pairs, 167 were male (mean age = 44.64 ± 11.32) and 194 were female (mean age = 43.69 ± 12.20). Of the dizygotic (DZ) pairs, 136 were male (mean age = 44.64 ± 12.42), 210 were female (mean age = 45.78 ± 12.61), and 251 were opposite-sex pairs (mean age = 45.86 ± 11.73). The excess of females over males is comparable to previous twin research [13].

(b) Prosocial obligation measures

Participants completed three scales measuring different forms of pro-social obligations: civic obligation (four items), work obligation (three items) and welfare obligation (three items) [7], with a response scale from 0 to 10. An example item assessing civic obli-gation was: '*how much obligation would you feel to testify in court about an accident you witnessed?*'; for work obligation: '*how much obli-gation would you feel to do more than most people would do on your kind of job?*'; and for welfare obligation: '*how much obligation would you feel to pay more for your healthcare so that everyone had access to healthcare?*' Cronbach's alpha [14] for the three prosocial domains was accepta-ble (civic, 0.78; work, 0.71; welfare, 0.81). For the purpose of examining an omnibus univariate model, the three prosocial obli-gations scales were summed to form a composite prosocial obligations measure. Prior to analysis, the effects of age and sex were regressed out, and standardized residuals were used in subsequent analyses [15].

(c) Analysis

All analyses used full-information maximum-likelihood modelling, and structural equation modelling was conducted using OpenMx [16,17] and R [18]. The modelling used to test our predictions is outlined graphically in figure 1. The prediction of heritable and/or environment effects specific to each of the three prosociality measures is shown in the lower portion of the figure, where each obli-gation is accounted for by a combination of additive genetic (A), shared- (or familial) environment (C), and unique-environment (E) effects. The upper portion of figure 1 shows the predicted latent mech-anism for domain-general prosociality, labelled here as 'prosociality mechanism'. This functions as a common pathway affecting all obli-gation measures, and through which genetic and environmental influences must be mediated. If the fit of this common pathway model does not differ significantly from that of the saturated model, this would provide support for a model of prosocial obligations as involving a general psychological mechanism influencing each of the three measured prosocial domains.

3. RESULTS

For each of the obligation domains, same-sex DZ twins were more highly correlated than were opposite-sex DZ twin pairs, suggestive of sex-limitation. Equating male and female DZ pairs, and DZ same-sex twin pairs to DZ opposite-sex pairs, did not significantly worsen model fit for any of the variables; however, to avoid biases in our model estimates, we analysed males and females separately.

Intra-class twin correlations are detailed in table 1. The full results of the univariate modelling for each domain are presented in table 2. For males across each obligation domain, including the general prosocial obli-gation scale, additive genetic and shared-environment effects were individually non-significant, although removing both paths simultaneously significantly wor-sened the model fit indicating familial influences on
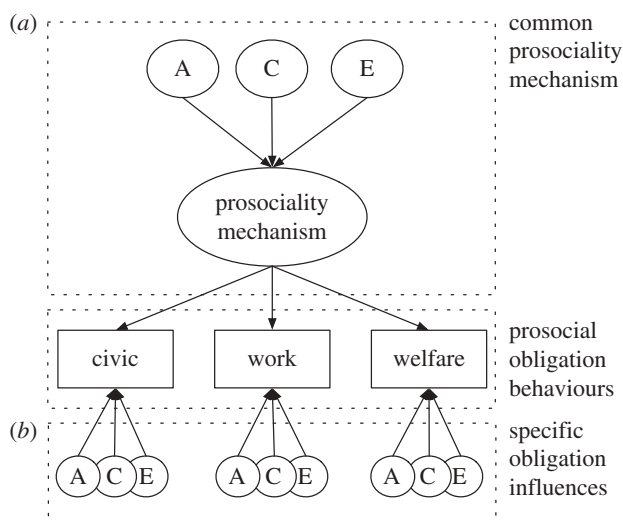
Figure 1. Common pathway model for prosocial obligations. The three measured prosocial obligations are shown in rectangles. Part (*a*) shows the putative common or general prosocial obligations mechanism. Part (*b*) represents putative genetic and environment influences for distinct prosocial obligations systems. A, additive genetic effects; C, common, or shared environment effects; E, unique-environment effects.

Table 1. Intra-class correlations for MZ, DZ same-sex, and DZ opposite-sex twin pairs for civic, work, welfare obligation and an omnibus summed measure of prosocial obligations.

	civic	work	welfare	prosociality
MZ male twin pairs	0.32	0.25	0.35	0.40
MZ female twin pairs	0.49	0.36	0.26	0.37
DZ male twin pairs	0.24	0.13	0.11	0.30
DZ female twin pairs	0.19	0.20	0.30	0.27
DZ opposite-sex twin pairs	0.06	−0.01	0.05	0.06

each trait. For females, additive genetic and unique-environment effects were significant for both civic and work obligation, with the third domain, welfare obligation, showing significant support only for unique-environmental influences (although dropping shared-environment effects reduced fit substantially, $p = 0.06$). For the general prosocial obligation measure, familial effects were observed: additive genetic and shared-environment effects were individually non-significant, but could not be dropped simultaneously without worsening model fit.

We next tested our full common-and-specific model of prosocial obligations. The phenotypic correlations between the three obligations variables were moderate-to-high ($0.41–0.68$). A confirmatory factor analysis indicated that neither a one-factor model nor a model with three uncorrelated obligation factors fitted the data well. By contrast, a hierarchical factor structure in which a super-ordinate 'prosocial obligations' factor loaded on the three obligations domains fitted well (see the electronic supplementary material). This indicated that both a common obligations factor and distinct obligations were present in the data.

Having established the existence of a common prosocial obligations factor, we moved to biometric analyses. We compared the fit of the full common pathway model to the saturated model. The full common pathway model was a better fit to the data, when compared with the saturated model, for both males and females (Akaike information criterion: 765.49 versus 767.90, and 1295.93 versus 1296.68, respectively). We next moved to tests of goodness-of-fit for nested common pathway models using the χ^2 test. For males, we could not drop all additive genetic and shared-environment effects simultaneously ($\Delta\chi_8^2 = 34.78, p < 0.01$); however, these effects were individually non-significant ($\Delta\chi_4^2 = 2.89, p = 0.58; \Delta\chi_4^2 = 1.08, p = 0.90$, respectively).

As such, we retained both of these sources of influence for further analyses. Nested tests indicated that the specific genetic effects on civic and work obligation, and the shared-environment effects on civic, work, and welfare obligation were non-significant, and so were removed from the model ($\Delta\chi_5^2 = 2.13, p = 0.83$). All other paths were significant and so we retained this reduced common-pathway model as our preferred final model for males (figure 2).

For females, the additive genetic effect on the common prosocial obligations factor was significant ($\Delta\chi_1^2 = 8.55, p < 0.01$); however, the shared-environment effect to the common factor could be dropped without significantly worsening fit ($\Delta\chi_1^2 = 0, p = 1$). At the domain-specific level, both additive genetic and shared-environment effects were non-significant ($\Delta\chi_3^2 = 2.13, p = 0.55; \Delta\chi_3^2 = 1.84, p = 0.61$, respectively); however, these influences could not be dropped simultaneously without significantly worsening fit ($\Delta\chi_6^2 = 57.62, p < 0.01$). Therefore, in line with the pattern of MZ–DZ intra-class correlations, we included genetic paths to civic and work obligation (reflecting the higher MZ to DZ correlations), and a shared-environment path to welfare obligation (reflecting the similar MZ to DZ correlations). The final model for females is presented in figure 2.

4. DISCUSSION

The data confirmed the significant role of genetic influences on prosocial obligations, supporting both common and specific mechanisms, and suggesting a distinct pattern of effects between the sexes. For the common prosocial obligations mechanism, additive genetic factors accounted for 48 per cent of the variance for females. In males, however, while familial effects on this common mechanism were apparent, the available power did not allow us to distinguish between additive genetic and shared-environment effects. These findings suggest that further work, perhaps with an extended twin design [19], will be required to tease apart possible mechanisms for these effects. Potential influences include both assortative-mating for prosociality, non-additive genetic effects [20] and, importantly, gene–environment interaction (G × E) [20] between the sexes (see below).
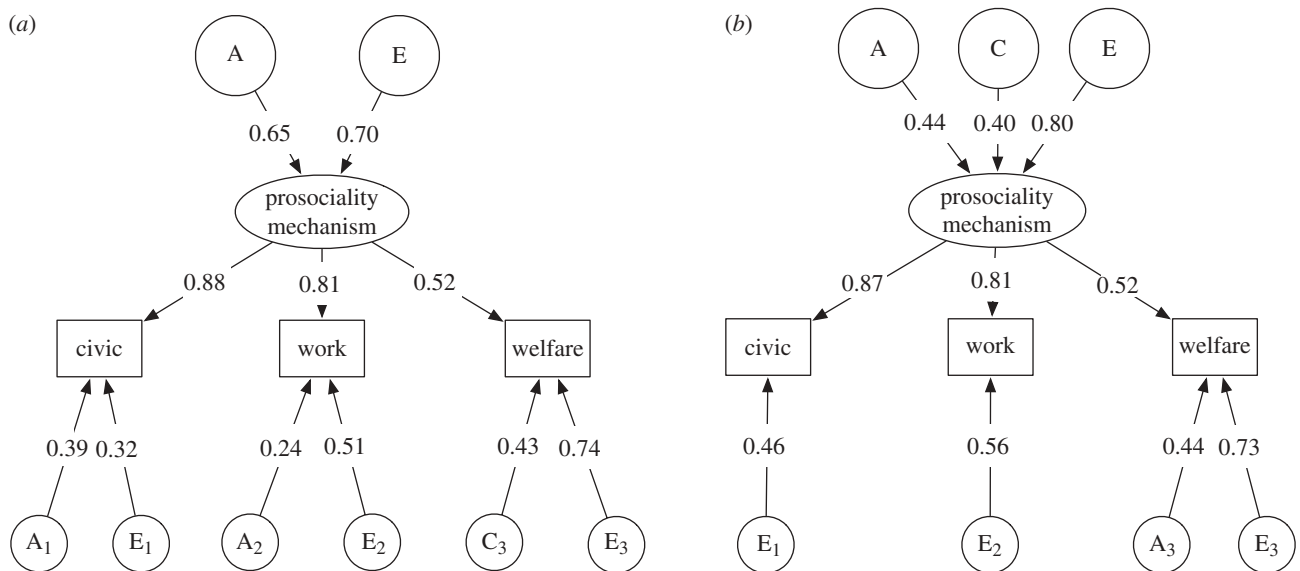
Figure 2. Final models of civic, work, and welfare prosocial obligations for (*a*) females and (*b*) males separately. Note: standardized path coefficients are shown, which when squared indicate the proportion of variance accounted for by that path. All paths are significant.

Table 2. Standardised univariate heritability estimates for civic, work, and welfare obligation, and an omnibus summed measure of prosocial obligations (95% CI in brackets).

	males			females		
	A	C	E	A	C	E
civic	0.14 (0–0.44)	0.17 (0–0.39)	0.69 (0.56–0.83)	0.50 (0.30–0.60)	0 (0–0.15)	0.50 (0.40–0.62)
work	0.15 (0–0.37)	0.08 (0–0.32)	0.77 (0.63–0.91)	0.38 (0–0.50)	0 (0–0.29)	0.62 (0.50–0.76)
welfare	0.34 (0–0.46)	0 (0–0.34)	0.66 (0.54–0.81)	0 (0–0.36)	0.28 (0–0.37)	0.72 (0.60–0.82)
general prosociality	0.23 (0–0.48)	0.13 (0–0.41)	0.64 (0.52–0.79)	0.32 (0–0.50)	0.06 (0–0.33)	0.62 (0.50–0.77)

At the domain-specific level, for males, welfare obligation showed a significant additive genetic component (with the other traits driven by unique-environment effects). For females, civic and work obligations showed significant additive genetic effects, with shared-environment influences underlying welfare obligations. The origin of these specific genetic influences on prosocial obligations is an open question. It may be that selection for stable division of work [21], civil conflicts [22] and welfare behaviours such as obligate food sharing [8] have been important in shaping specific adaptations linked to in-group cooperation.

Unique-environment effects were often large. While these effects partly reflect measurement error, they also potentially reflect gene–environment interaction (G × E) [20]. Given the likely differential family and social constraints placed on males and females enforcing compliance versus rewarding independence [23], it seems likely that genetic differences related to prosocial obligations may interact with these differentially experienced social environments. This view would be consistent with contingency models of prosocial obligations supported by research on the moderation of prosocial contributions by social exclusion [24] and knowledge of prosociality levels in the group [25].

In summary, the present data indicate that a common factor underlies prosocial obligations across three important social domains. The results also highlight important avenues for additional study in line with the contrasting observation of moderate-to-large additive genetic factors underlying this general prosocial obligations factor in females, with modest-to-moderate familial factors influencing general prosocial obligations in males. Further research is required to explore these possible gender differences and transmission modes, as well as the possible effects of contingency upon obligations.

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