How genes influence personality: Evidence from multi-facet twin analyses of the HEXACO dimensions

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ABSTRACT

Much of the variation in human personality is described by five to six dimensions. Less clear, however, is whether these dimensions are underpinned by similarly coherent and unified biological bases. Using the HEXACO traits, we tested whether genetic covariance within each dimension was underpinned by a single common genetic factor using three distinct classes of multivariate model. For each of the six factors, only a single common genetic basis was required to explain genetic covariation among the facets, along with facet-specific genetic effects. These findings provide support for personality models positing that the major dimensions of personality are each underpinned by a unified and coherent biological architecture.

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1. Introduction

The psychometric structure of personality has been a topic of enduring interest for some decades (John, Naumann, & Soto, 2008). As a result of this research, it is now uncontroversial to assert that a small number of latent factors – often five (Costa & McCrae, 1992a) or, less frequently, three (Eysenck & Eysenck, 1975), or six (Lee & Ashton, 2004) – account for the bulk of reliable variance in a wide spectrum of traits and behaviors. However, alongside this descriptive research, a parallel debate has focussed on whether these domains reflect unitary underlying biological systems (McCrae & Costa, 1999), or are instead better understood as convenient heuristics, valid only at the phenotypic level (Jang, Livesley, Angleitner, Riemann, & Vernon, 2002; Paunonen & Jackson, 1996; Saucier & Goldberg, 1996). Note, this latter perspective does not necessarily suggest that heritable effects on personality are absent; rather, it posits that heritable effects do not form a unitary underlying genetic architecture.

While much has been published on this topic (e.g. Costa & McCrae, 1998), tests of the unitary basis of personality domains have largely taken place at the phenotypic level. This taxonomic approach has helped to advance the field by providing a common language for the structure of basic traits (John et al., 2008). However, because this work operates at the phenotypic level,

unresolved questions concerning underlying etiological bases still exist. A powerful alternative to this approach is to use genetically informative data directed specifically at testing the underlying structure of personality (Bates & Lewis, 2012), although little research to date has adopted this strategy in order to address this issue. Moreover, the few papers published to date have provided mixed results. Accordingly, in the current study, our goal was to test whether each of the basic domains of personality – indexed here by the HEXACO six-factor model of traits – reflect a single underlying genetic basis (i.e. genes that influence all of the facets of the respective domain), as well as the nature of additional genetic factors required to account for the heritable multivariate or facet-level structure present beneath each of the major domains. We next introduce the key research to date in this field.

1.1. Genetic architecture of personality: previous research

Much work has been conducted using genetically informative designs to investigate the etiology of personality (Bouchard, 2004). Of note, however, here we are interested specifically in multivariate genetic analyses that offer a window to whether items and/or facets of core personality domains show a common genetic basis: univariate analyses, the more common approach in this literature to date (e.g. Bouchard, 2004), while undoubtedly useful for answering certain other questions, cannot provide insight to the underlying architecture across multiple facets. As noted by Heath, Eaves, and Martin (1989), analyzing scales of aggregated items confounds the genetic and environmental influences that

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are specific to subsets of these items with influences that may be shared by all items.

The first study (of which we are aware) to meet this requirement of incorporating genetic multivariate analyses at the item- or, at least, facet-level of personality was reported by Heath et al. (1989). These authors examined the items of the Eysenck Personality Questionnaire's (Eysenck & Eysenck, 1975) extraversion, neuroticism, and psychoticism scales in adolescents, and tested whether the items forming each of these dimensions showed evidence for common genetic effects within each domain. For both extraversion and neuroticism, Heath et al. (1989) found evidence for a common genetic influence. For psychoticism, evidence was found for a more complicated genetic architecture, suggesting that this dimension was comprised of at least two distinct heritable factors, consistent with psychometric work suggesting this dimension is better parsed into distinct domains of agreeableness and conscientiousness (Costa & McCrae, 1992b). This first study, then, supported a coherent genetic basis for two of three major personality domains, and indicated the power of the method to detect erroneous aggregations of distinct traits. It is important to note, however, that Heath et al. (1989) did not compare competing models in exploring the nature of this general factor – as discussed more fully below – limiting the conclusions that could be drawn from such analyses.

Jang et al. (2002) subsequently reported a study deploying similar techniques to those used by Heath et al. (1989), but this time using NEO-PI-R data, and thus providing full coverage of the Five-Factor personality space. In addition, and of importance, these authors compared two distinct classes of multivariate model, allowing them to contrast differing accounts of the genetic architecture of each of the five factors. The first class of model, the common pathway model, constrains all common genetic and environmental variance though a single pathway (Kendler, Heath,

Martin, & Eaves, 1987; see Fig. 1). The second class of model, the independent pathway model, also constrains genetic and environmental covariation to be explained via a single common pathway, but instead of requiring all sources of this covariance to be channelled through a single mediating latent factor, this model allows for independent general effects of genes, shared-environments, and unique-environments (also see Fig. 1). Jang et al. (2002) found that, while evidence for common genetic effects across all facets was present for each of the five factors, the common pathway model nevertheless provided a comparatively poor fit in each case. Moreover, Jang et al. (2002) also reported the existence of a second, independent genetic factor loading on between three and five of the facets (depending on which domain was under analysis). This observation led Jang et al. (2002) to note that the "*the present results suggest that higher-order traits such as "neuroticism" do not exist as veridical psychological entities per se, but rather they exist as useful heuristic devices that describe pleiotropic effects and the common influence of environmental factors on sets of individual facets*" (p. 99).

Subsequently, Johnson and Krueger (2004) reported on a new sample, again using Big Five measures, but based on a 25-item instrument (five adjectives per domain). In this study, somewhat more nuanced results were reported. The common pathway model provided the best fit for neuroticism and extraversion. In contrast, agreeableness, conscientiousness, and openness showed a complex genetic architecture, with covariation between the domain items not fully captured by a single factor: Indeed, these domains were not even well described by the independent pathway model, supportive of the observation by Jang et al. (2002) that the genetic architecture of personality is complex.

Finally, and most recently, Briley and Tucker-Drob (2012) used 440-items from the California Personality Inventory to generate NEO facets in a sample of 800 adolescent twin pairs. For extraversion
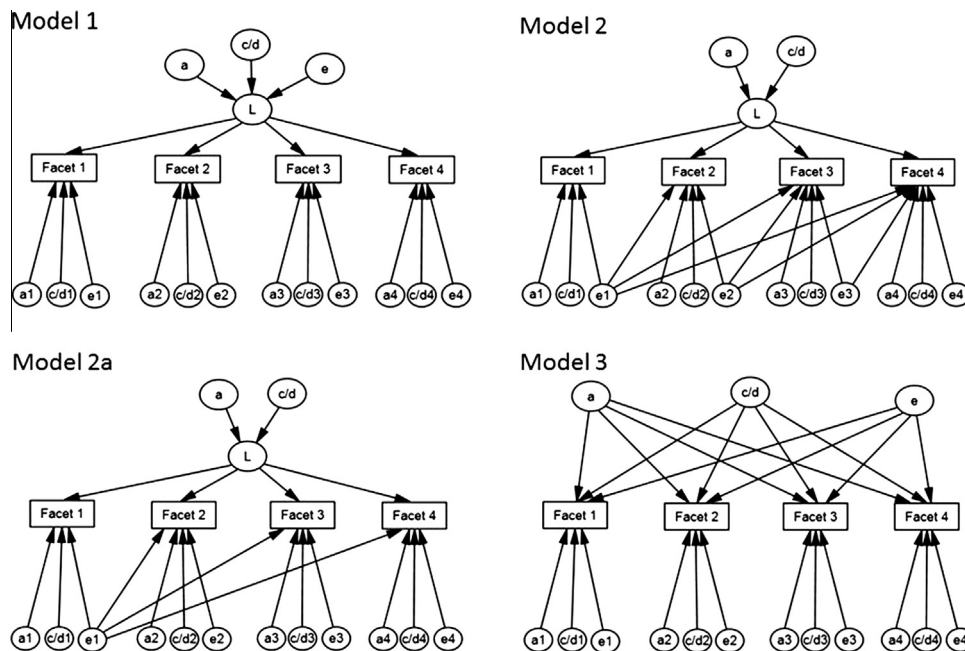

Fig. 1. Graphical representation of the theoretical models. *Note:* Model 1 details a common pathway model, which requires common influences of additive genetic (a), shared-environment (c) or dominance genetic (d) effects, and nonshared-environment (e) effects on HEXACO personality facets to act on a single common latent factor (L); Model 2 requires common influences of additive genetic (a) and dominance genetic (d) effects on HEXACO personality facets to act on a single common latent factor, and also allows a common nonshared-environment factor to explain covariation independent of this common genetic pathway; Model 2a is similar to Model 2 but allows additional nonshared-environmental paths to explain covariation; Model 3 details an independent pathway model, which also models common influences of additive genetic (a), shared-environment (c) or dominance genetic (d) effects, and nonshared-environment (e) effects on HEXACO personality facets, although these common effects are allowed to differ for genetic and environmental influences. Because estimation of C and D cannot be performed simultaneously with information solely from MZ and DZ twins, only one of these variance components was included for an given model (see text for further details).

and openness, a common pathway model fit best. For agreeableness, neuroticism, and conscientiousness, however, a common pathway model was rejected in favor of an independent pathway model. However, as results from additional models (e.g. the Cholesky decomposition) were not reported, it is unclear from this study whether the genetic covariation of the facets within each domain were well described by a single common or independent factor, or whether further genetic factors (as predicted to be the case by Jang et al. (2002) and Johnson and Krueger (2004)) would achieve a still better fit.

1.2. The current study

While these previous studies have provided important insights into the genetic architecture of personality, primarily demonstrating the existence of (at least) one common genetic basis for each of the major Big Five traits, clearly further research is needed. Specifically, the architecture of these common genetic bases is not consistent across the studies and thus additional study is required to determine the underlying biological structure of personality. For example, Johnson and Krueger (2004) report that common pathway models provided preferred fits for neuroticism and extraversion only, whereas Briley and Tucker-Drob (2012) find evidence for a common pathway interpretation of extraversion and openness, but not for neuroticism. And Jang et al. (2002) found no evidence for common pathway models providing best fit for any of the Big Five domains, instead concluding that the genetic covariation within domains is more complex than can be captured though a single unifying factor.

Some important limitations are also apparent from this previous work. Firstly, the limited construct breadth apparent in Johnson and Krueger's (2004) work – for example, neuroticism was defined by five highly similar items – may have meant that the full range of the construct was not tapped, and so reducing the insights that can be drawn from claims that these analyses tested the genetic unity of the construct space. Secondly, while dominance effects (i.e. non-additive genetic influences) are noted to underlie personality traits (Keller, Coventry, Heath, & Martin, 2005), of the previously-noted studies only Heath et al. (1989) and Briley and Tucker-Drob (2012) assessed the dominance genetic architecture, and so mixed results may have emerged from conflating these sources of variance into a single component. Finally, although not a criticism of previous work *per se*, no study (of which we are aware) has extended this form of multivariate analysis to the HEXACO traits. Accordingly, it is unknown whether the additional factor of honesty/humility (Ashton & Lee, 2007) reflects a coherent underlying genetic architecture. It is important to briefly note at this point that while the HEXACO traits are often highly similar to their namesake within the Big Five framework, specific distinctions are apparent beyond the simple addition of honesty/humility. Most prominently, HEXACO agreeableness omits aspects such as sympathy and sentimentality, as well as the modesty and humility aspects that define honesty/humility, and in the process arguably capturing a construct more akin to the label of the factor than achieved with Big Five agreeableness (Ashton & Lee, 2007). Similarly, while HEXACO emotionality shares features with Big Five neuroticism, it includes sentimentality and excludes anger, which contribute to Big Five neuroticism.

These mixed results, along with the noted methodological limitations, motivated the current study. Our goal was straightforward: We sought to establish the genetic coherence of the six factors defined by the HEXACO model of personality. To achieve this goal we used a large sample of middle-aged (mostly female) adult UK twin pairs, to whom the HEXACO 60-item personality instrument had previously been administered. Data from these twin pairs were analyzed using multi-group, multivariate structural equation modeling in order to test a series of genetically informative models concerning the underlying genetic architecture of the HEXACO traits.

To this end, we were faced with the question of exactly which models best reflected current theories in the field concerning the underlying genetic architecture of personality. Johnson and Krueger (2004) have suggested that the common-pathway model, which constrains common covariance to the indicators to be explained by a single latent factor, which in turn is influenced by genetic and environmental factors, is the best reflection of the claims made by personality trait theories. Somewhat provocatively then, using this model (as noted above), these authors reported no unitary basis to at least some of the core domains of personality. We, however, feel this model unfairly reflects the predictions of personality system theory (McCrae & Costa, 1999). In contrast, we suggest that a mixed model that constrains additive and dominance genetic effects (when dominance effects are observed to contribute to trait variation) through a common pathway, as well as a common unique-environmental factor would best reflect the predictions from personality system theory. This position would seem to follow the clear distinction between basic tendencies and characteristic adaptations made by personality system theory (McCrae & Costa, 1999), with the former conceptualized as the biological backbone of personality and (largely) impermeable to systematic environmental influences, and the latter conceptualized as the aspects of the broader personality space shaped by personal experience and cultural factors. We do note, however, that unique-environment effects may not be limited to the level of characteristic adaptations, but also operate at the level of basic tendencies, perhaps via influences on processes during brain development that serve to lay the foundations for basic tendencies.

With these expectations in mind, then, we included models that can be viewed conceptually as lying in an intermediate position between the independent pathway and common pathway model designs. In this combined model, we constrained additive and dominance genetic effects to explain common phenotypic variation via a single common pathway (as with the common pathway model), but allowed the unique-environment effects to have a common factor, acting directly on the traits (as in the independent pathway model). We allowed this unique-environment structure to either take the form of full triangular decomposition so as to not reject models based on local misspecification concerning unique-environments given our specific interests in the genetic architecture, or to possess a sole common factor with additional specific effects to each of the personality facets (see Fig. 1).

2. Methods

2.1. Participants

The current study sample was drawn from a subset of the TwinsUK study, which is an on-going study following monozygotic (MZ) and dizygotic (DZ) twins in the UK (Moayyeri, Hammond, Hart, & Spector, 2013). Mean age was 61 years (SD = 12.84). Twin zygosity was determined using self-rating measure of similarity and genotyping when uncertainty was present (Ooki, Yamada, Asaka, & Hayakawa, 1990). The current sample consisted of the following number of complete twin pairs: MZ male pairs: $n = 36$ (unpaired singletons: $n = 128$); MZ female pairs: $n = 474$ (unpaired singletons: $n = 378$); DZ male pairs: $n = 30$ (unpaired singletons: $n = 127$); DZ female pairs: $n = 408$ (unpaired singletons: $n = 569$). No DZ opposite-sex pairs were available for analysis. The sample

contains considerably more females than males because the initial phenotypes of interest (e.g. osteoporosis) to the TwinsUK registry show greater prevalence in females and so this demographic was targeted.[1] More recent data collections have started to include male participants (Spector & Williams, 2006).

2.2. Measures

Participants were administered the 60-item HEXACO Personality Inventory (HEXACO-60; Ashton & Lee, 2009). The HEXACO-60 assesses six personality dimensions (10 items per dimension), with each of these dimensions reflected in four facets (2–3 items per facet): honesty-humility (sincerity, fairness, greed avoidance, and modesty), emotionality (fearfulness, anxiety, dependence, and sentimentality), extraversion (social self-esteem, social boldness, sociability, and liveliness), agreeableness (forgiveness, gentleness, flexibility, and patience), conscientiousness (organization, diligence, perfectionism, and prudence), and openness to experience (aesthetic appreciation, inquisitiveness, creativity, and unconventionality). Participants responded to self-reflective statements on this questionnaire using a 5-point Likert scale (1 = strongly disagree, to 5 = strongly agree) and facets were constructed as the mean score of the items (either two or three) that comprised the scale, reversing where appropriate.

2.3. Analyses

The classical twin design partitions observed variation into three latent components: Additive genetic influences (A), shared-environmental influences (C; environmental influences fostering similarities within twin pairs), dominance genetic influences (D: non-additive gene effects), and unique-environmental influences (E; environmental influences serving to make individuals within a twin pair less similar). Genetic effects are inferred when monozygotic (MZ) twins are more similar than dizygotic (DZ) twins, shared-environment effects are inferred when MZ twin correlations are less than twice that of the DZ twins, and dominance genetic effects are inferred when MZ twin correlations are more than twice that of the DZ twins. Unique-environment effects are inferred when MZ twins are correlated less than at unity for a given trait, and this variance component thus also contains measurement error. While these heuristics provide an instructive guide to the pattern of relative genetic and environmental effects, modern approaches typically utilize a multi-group structural equation modeling framework, which facilitates formal tests of parameter significance, as well as for the estimation of parameters in multivariate models (Neale, 2003), such as in the current study.

Prior to analyses, the effects of age and sex were controlled, and standardized residuals were used in subsequent analyses (McGue & Bouchard, 1984). A classical twin design was used. We used structural equation modeling to model the covariance of identical twins in terms of additive genetic effects, shared-environment effects or dominance genetic effects (as necessary), and unique-environment effects. These models were estimated by full-information maximum likelihood analysis using OpenMx (Boker et al., 2010, 2013).

3. Results

Descriptive statistics for all measures (for one individual in a twin pair selected at random) and twin correlations are detailed in Table 1. For all of the 24 measures, MZ twin correlations

exceeded DZ twin correlations indicating the presence of genetic effects on trait variation. All MZ correlations were less than 1.0 indicating that all measures contained unique-environmental influences (which also include measurement error). Evidence for shared-environment effects was limited: DZ twin correlations were less than half the MZ twin correlations for 20 of the 24 facets indicating possible non-additive genetic influences.

3.1. Multivariate twin analyses

We next built a series of multivariate models reflecting the major competing perspectives concerning the genetic architecture of personality, as detailed in Fig. 1. We compared the fit of each these models for each of the HEXACO personality dimensions. We modeled shared-environment effects for openness, in line with some of the DZ correlations observed to be greater than half the MZ correlations. Because of the omission of dominance effects for this domain, we did not test all of the theoretical models detailed in Fig. 1: Instead, we limited our analyses to Models 1 and 3. Dominance genetic effects were modeled for the other dimensions.

The fit statistics for each of the fitted models are detailed in Tables 2–7. Because the majority of our models were non-nested we used the Akaike Information Criterion (AIC) to adjudicate between our models. Our findings indicate the following: for honesty/humility, emotionality, and agreeableness, Model 2a – the model with a common pathway for additive and dominance genetic effects, and a triangular decomposition for unique-environment effects – provided the most parsimonious fit to the data. For conscientiousness, the independent pathway model was retained as the final model. Finally, for extraversion and openness the full Cholesky decomposition (the baseline model) was favored.

In order to not reject models with a common genetic factor on grounds of local misspecification we examined whether modest alterations to these models at the level of specific genetic and environmental influences (i.e. the parameters in the lower half of the common and independent pathway models: see Fig. 1) led to improved fit for extraversion and openness. In each case we noted that minor modifications led to substantial improvements in model fit such that it became apparent that common genetic factors were evident in each case. For extraversion, a common pathway model that allowed the specific A, D, and E latent factors for liveliness to also load on social esteem produced a model that did not fit significantly worse than the Cholesky (see Table 4). For openness, an independent pathway model that allowed the specific E factor for inquisitiveness to also load on unconventionality produced a model that did not fit significantly worse than the Cholesky (see Table 7). We retained these modified models as our favored final models, although we also note that modifications of this kind should be acknowledged as exploratory.

In a final set of steps we individually tested each of the A, C/D, and E paths for significance. In the case of A and C/D, a number of these paths were not individually significant, despite often showing moderately-sized parameter estimates; however, simultaneously dropping non-significant A and C/D paths that loaded on the same measured or latent variable often led to dramatic worsening in fit. Such an observation suggests that while power may not allow us to distinguish between these two sources of variance for a given variable, significant effects are in fact present. As such, we report all parameters in our final models and note which were individually significant and which were significant only when removed in tandem with the corresponding A or C/D path. The final model for each of HEXACO dimensions is detailed in Figs. 2–7.

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[1] Analyses reflect all available information, but our results were unchanged when using only female twin pairs.

Table 1
Descriptive statistics and twin correlations for HEXACO facets.

Facet	α	Means (SD)			Twin correlations	
		All	MZ	DZ	MZ	DZ
Honesty/humility						
Sincerity	.43	3.80 (.69)	3.79 (.70)	3.82 (.69)	.24	.09
Fairness	.55	4.27 (.77)	4.27 (.79)	4.25 (.74)	.34	.11
Greed avoidance	.49	3.37 (.81)	3.37 (.82)	3.37 (.80)	.32	.09
Modesty	.60	4.03 (.69)	4.05 (.69)	4.02 (.69)	.27	.09
Emotionality						
Fearfulness	.56	2.93 (.77)	2.88 (.77)	2.98 (.76)	.35	.17
Anxiety	.63	3.34 (.87)	3.34 (.84)	3.35 (.90)	.41	.07
Dependence	.53	3.07 (.82)	3.09 (.83)	3.05 (.80)	.31	.14
Sentimentality	.50	3.55 (.65)	3.55 (.65)	3.55 (.65)	.40	.16
Extraversion						
Social self-esteem	.64	3.81 (.71)	3.83 (.71)	3.79 (.70)	.38	.17
Social boldness	.67	3.06 (.78)	3.09 (.77)	3.04 (.79)	.45	.11
Sociability	.43	3.43 (.73)	3.44 (.75)	3.43 (.71)	.32	.07
Liveliness	.46	3.46 (.71)	3.44 (.71)	3.49 (.70)	.29	.14
Agreeableness						
Forgiveness	.74	3.18 (.87)	3.18 (.88)	3.19 (.86)	.35	.15
Gentleness	.50	3.28 (.64)	3.25 (.64)	3.30 (.64)	.32	.12
Flexibility	.39	3.34 (.62)	3.31 (.62)	3.37 (.62)	.25	.10
Patience	.60	3.66 (.76)	3.62 (.76)	3.70 (.76)	.25	.03
Conscientiousness						
Organization	.49	3.89 (.77)	3.94 (.76)	3.84 (.77)	.21	.11
Diligence	.38	3.83 (.67)	3.84 (.66)	3.82 (.68)	.26	.14
Perfectionism	.54	3.54 (.67)	3.56 (.66)	3.52 (.67)	.27	.11
Prudence	.55	3.51 (.64)	3.52 (.65)	3.49 (.63)	.33	.11
Openness						
Aesthetic appreciation	.59	3.54 (.97)	3.52 (.95)	3.55 (.99)	.49	.25
Inquisitiveness	.52	3.43 (.85)	3.42 (.85)	3.44 (.85)	.41	.19
Creativity	.62	3.31 (.83)	3.26 (.84)	3.36 (.81)	.51	.14
Unconventionality	.53	3.16 (.64)	3.12 (.64)	3.19 (.64)	.40	.22

Note. SD = standard deviation; α = Cronbach's alpha for scale scores collapsed across sex and zygosity; MZ = monozygotic; DZ = dizygotic; Means and α were derived from one randomly chosen member from each twin pair.

Table 2
Fit statistics for all models tested with honesty/humility facets.

Model	−2LL	df	AIC
Honesty/humility			
Baseline model	33152.57	11,989	9174.57
Model 1: CP	33173.81	12,001	9171.81
Model 2: CPAD/common E	33207.59	11,999	9209.59
Model 2a: CPAD/full lower E	**33154.12**	**11,996**	**9162.12**
Model 3: IP	33157.19	11,995	9167.20

Note. ADE model fitted; CP = common pathway model; CPAD = common pathway model for A and D effects only; IP = independent pathway model; A = additive genetic effects, D = dominance genetic effects, E = nonshared-environment effects; Final model is bolded.

Table 3
Fit statistics for all models tested with emotionality facets.

Model	−2LL	df	AIC
Emotionality			
Baseline model	32139.04	11,836	8467.04
Model 1: CP	32170.21	11,848	8474.21
Model 2: CPAD/common E	32190.86	11,846	8498.86
Model 2a: CPAD/full lower E	**32147.92**	**11,843**	**8461.92**
Model 3: IP	32147.48	11,842	8463.49

Note. ADE model fitted; CP = common pathway model; CPAD = common pathway model for A and D effects only; IP = independent pathway model; A = additive genetic effects, D = dominance genetic effects, E = nonshared-environment effects; Final model is bolded.

Table 4
Fit statistics for all models tested with extraversion facets.

Model	−2LL	df	AIC
Extraversion			
Baseline model	31749.64	11,974	7801.64
Model 1: CP	31903.90	11,986	7931.90
Model 1a: modified CP	**31763.09**	**11,983**	**7797.09**
Model 2: CPAD/common E	31939.4	11,984	7971.40
Model 2a: CPAD/full lower E	31776.40	11,981	7814.40
Model 3: IP	31774.93	11,980	7814.93

Note. ADE model fitted; CP = common pathway model; CPAD = common pathway model for A and D effects only; IP = independent pathway model; A = additive genetic effects, D = dominance genetic effects, E = nonshared-environment effects; Final model is bolded.

Table 5
Fit statistics for all models tested with agreeableness facets.

Model	−2LL	df	AIC
Agreeableness			
Baseline model	32091.05	11,893	8305.05
Model 1: CP	32115.81	11,905	8305.81
Model 2: CPAD/common E	32229.95	11,903	8423.95
Model 2a: CPAD/full lower E	**32094.03**	**11,900**	**8294.03**
Model 3: IP	32093.08	11,899	8295.08

Note. ADE model fitted; CP = common pathway model; CPAD = common pathway model for A and D effects only; IP = independent pathway model; A = additive genetic effects, D = dominance genetic effects, E = nonshared-environment effects; Final model is bolded.

Table 6
Fit statistics for all models tested with conscientiousness facets.

Model	−2LL	df	AIC
Conscientiousness			
Baseline model	32526.55	11,950	8626.55
Model 1: CP	32591.27	11,962	8667.27
Model 2: CPAD/common E	32595.82	11,960	8675.82
Model 2a: CPAD/full lower E	32535.32	11,957	8621.32
Model 3: IP	**32531.26**	**11,956**	**8619.26**

Note. ADE model fitted; CP = common pathway model; CPAD = common pathway model for A and D effects only; IP = independent pathway model; A = additive genetic effects; D = dominance genetic effects; E = nonshared-environment effects; Final model is bolded.

Table 7
Fit statistics for all models tested with openness facets.

Model	−2LL	df	AIC
Openness			
Baseline model	31491.12	11,913	7665.12
Model 1: CP	31557.28	11,925	7707.28
Model 2: CPAD/common E	–		–
Model 2a: CPAD/full lower E	–		–
Model 3: IP	31506.73	11,919	7668.73
Model 3a: modified IP	**31499.93**	**11,918**	**7663.93**

Note. ACE model fitted; CP = common pathway model; IP = independent pathway model; A = additive genetic effects, C = shared-environment effects, E = nonshared-environment effects; Model 2 and 2a were not tested for these facets as D effects were not modeled; Final model is bolded.

4. Discussion

The current study tested competing models concerning the genetic architecture of HEXACO personality traits with the goal of determining whether a unitary underlying genetic factor was sufficient to explain the genetic covariation between domain facets. For each of the domains, genetic covariation was adequately represented by a single common genetic factor, with no need to specify further localised genetic covariation in order to provide a well-fitting model. These results are consistent with models of personality that posit that basic dimensions of personality reflect a coherent underlying biological architecture that broadly mirrors the phenotypic architecture (McCrae & Costa, 1999).

Our findings show both convergence and divergence from previous work in the field. For example, genetic covariation within the facets of neuroticism, extraversion, and openness each was noted to be well explained with a single underlying common genetic factor, a result consistent with most other work in the literature (Briley & Tucker-Drob, 2012; Heath et al., 1989; Johnson & Krueger, 2004), although Briley and Tucker-Drob's (2012) results only converge with our findings for extraversion and neuroticism. Genetic covariation underpinning the facets of agreeableness and conscientiousness was also found here to be adequately explained by a model with a single common genetic factor, an observation in line with work from Briley and Tucker-Drob (2012), but not that of either Jang et al. (2002) or Johnson and Krueger (2004), who reported that a more complex genetic architecture was required to fully explain these traits. Finally, and novel to this study, we observed that honesty-humility was adequately explained by a single common genetic factor.

The fact that this pattern of results – specifically for agreeableness and conscientiousness – does not cleanly reflect prior work in the field raises questions as to why such differences may have arisen. We suggest the following reasons as possible explanations. Firstly, we used the HEXACO six-factor instrument, which possesses a somewhat different architecture to Big Five (Ashton & Lee, 2007). The inclusion of the sixth factor of honesty-humility necessarily alters the structure of agreeableness and neuroticism/emotionality, as elements of these constructs move to define honesty-humility. This may explain why we see a single common genetic factor explaining genetic covariance for agreeableness, whereas Jang et al. (2002) and Johnson and Krueger (2004) did not: our measure of agreeableness may simply have been a purer construct by not including the variance associated with honesty/humility and neuroticism. Secondly, we examined additional structural models not used before in this literature, and which may have provided a more realistic test of the genetic architecture of personality.

Our study was not without specific limitations and thus recommendations for future work are warranted. First, our sample largely consisted of middle-aged females, and as such future studies of this kind on males, or at least samples including comparable numbers of males to females, would be useful. Second, because we only possessed data in twins we were constrained in the number of variance components (ACE or ADE) that we could simultaneously model. Models incorporating additional genetic relatedness data (e.g.
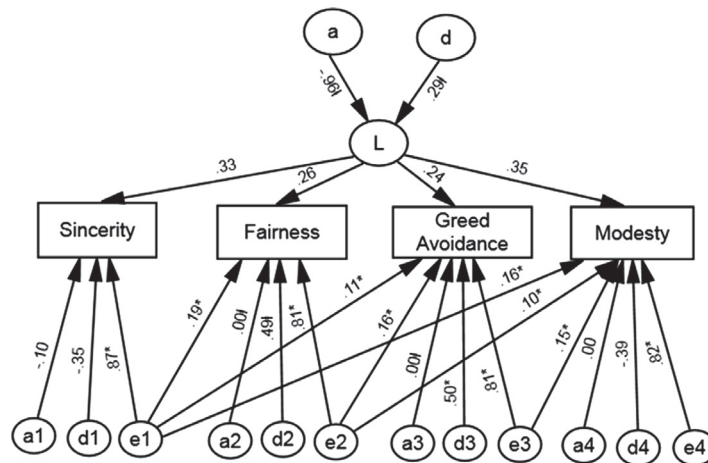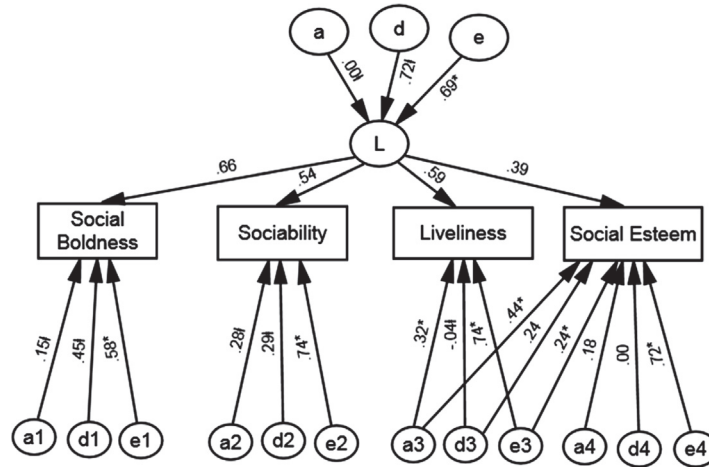

Note. * p < .05; ‡ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.

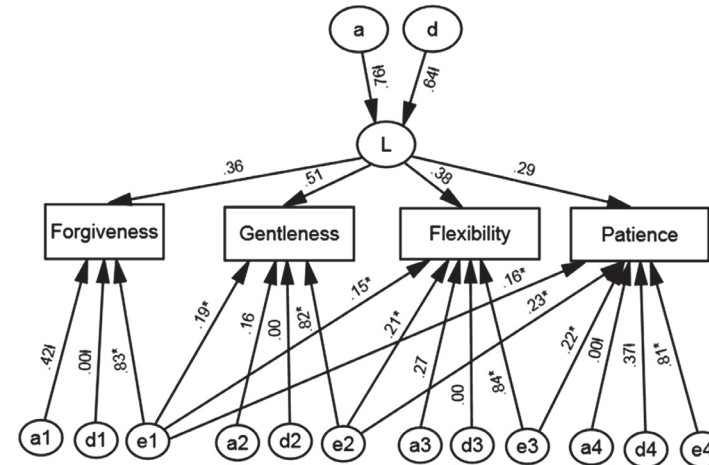**Fig. 2.** Final model for honesty-humility.
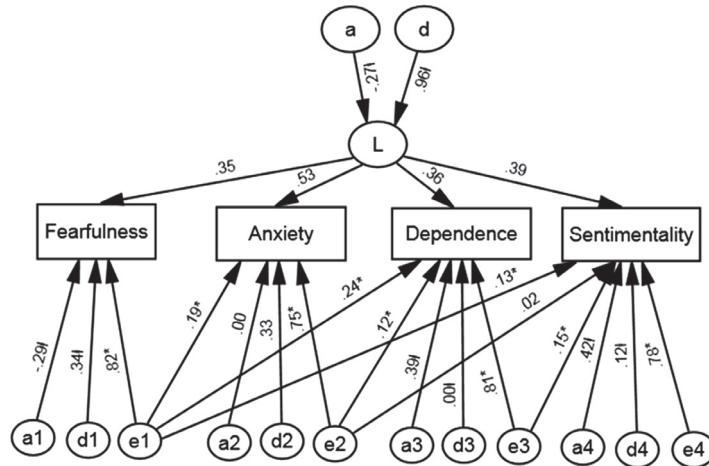
Note. * p < .05; ǂ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.

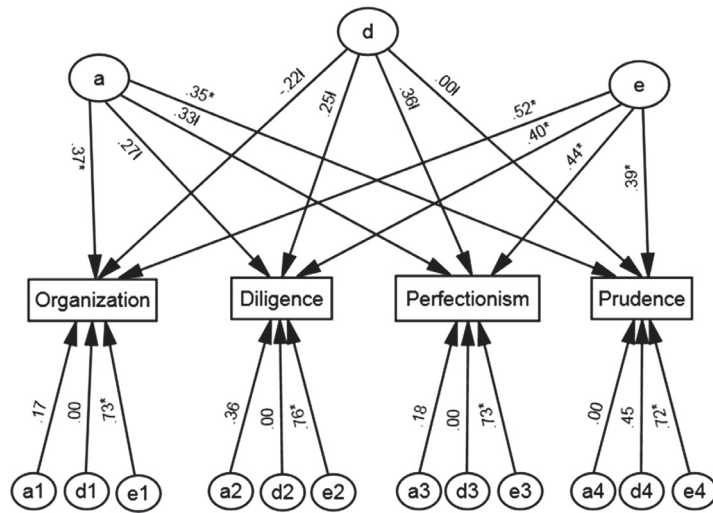**Fig. 3.** Final model for emotionality.

Note. * p < .05; ǂ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.
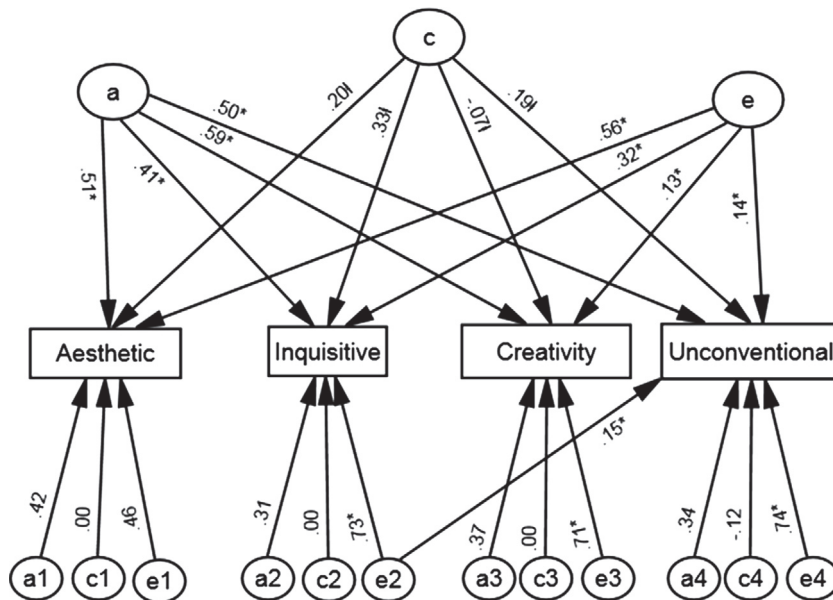
Fig. 4. Final model for extraversion.

Note. * p < .05; ǂ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.

Fig. 5. Final model for agreeableness.

Note. * p < .05; ‡ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.

Fig. 6. Final model for conscientiousness.

Note. * p < .05; ‡ p < .05 when the corresponding a or c/d path is simultaneously constrained to zero.

Fig. 7. Final model for openness.

parents, offspring) provide the capability to more comprehensively assess the genetic architecture of personality (Eaves, Last, Young, & Martin, 1978; Nance & Corey, 1976). Finally, the Cronbach's alpha values for a number of our facets were relatively low. This is not unusual for scales comprised of 2 or 3 items (such as the scales used in the current study) as Cronbach's alpha increases, in part, as a function of scale length (Cortina, 1993); however, this may have made it more difficult to detect additional unique genetic influences. Nonetheless, given our focus here was specifically on the common genetic architecture of the HEXACO traits this limitation is of only modest concern.

In conclusion, these results indicate that for all of the HEXACO traits only a single underlying common genetic factor is needed

to account for genetic covariation among each dimension's facets, although genetic effects specific to the facets was also apparent. This suggests that dimension-scores are valid targets for the search for genes affecting personality, and that models which do not include a role for generalist genes within personality dimensions must be updated.

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