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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.

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

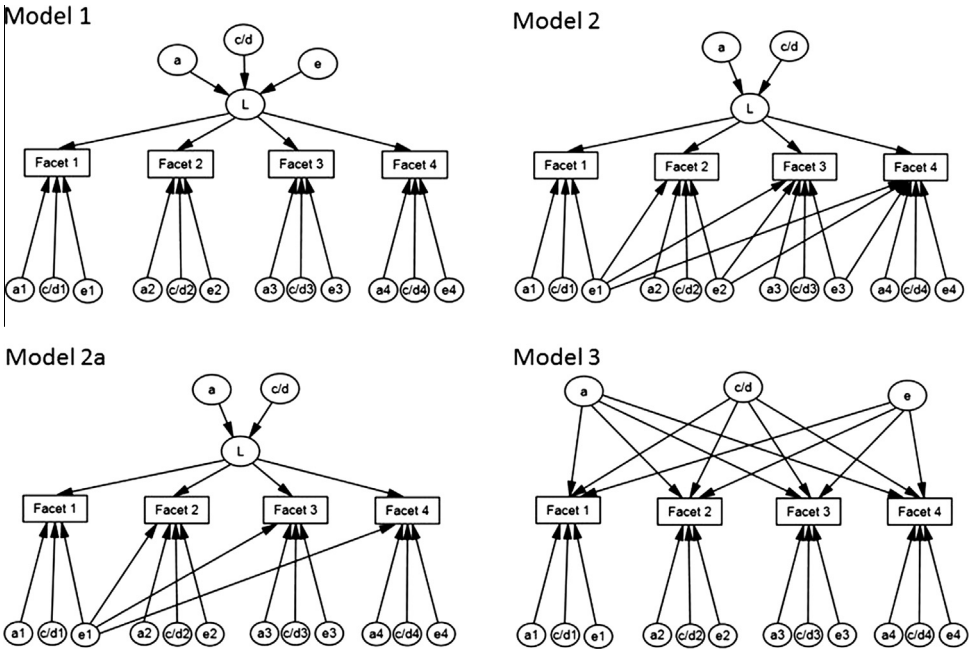

Fig. 1. Graphical representation of the theoretical models. *Note:* Model 1 details a common pathway model, which requires common influences of additive genetic (a), shared-environment (c) or dominance genetic (d) effects, and nonshared-environment (e) effects on HEXACO personality facets to act on a 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).