Psychological Methods
2013, Vol. 18, No. 3, 406–433

1082-989X/13/$12.00 DOI: 10.1037/a0032755

Can Genetics Help Psychometrics? Improving Dimensionality Assessment Through Genetic Factor Modeling

Sanja Franić
Vrije Universiteit Amsterdam

Conor V. Dolan and Denny Borsboom
University of Amsterdam

James J. Hudziak
University of Vermont

Catherina E. M. van Beijsterveldt and Dorret I. Boomsma
Vrije Universiteit Amsterdam

In the present article, we discuss the role that quantitative genetic methodology may play in assessing and understanding the dimensionality of psychological (psychometric) instruments. Specifically, we study the relationship between the observed covariance structures, on the one hand, and the underlying genetic and environmental influences giving rise to such structures, on the other. We note that this relationship may be such that it hampers obtaining a clear estimate of dimensionality using standard tools for dimensionality assessment alone. One situation in which dimensionality assessment may be impeded is that in which genetic and environmental influences, of which the observed covariance structure is a function, differ from each other in structure and dimensionality. We demonstrate that in such situations settling dimensionality issues may be problematic, and propose using quantitative genetic modeling to uncover the (possibly different) dimensionalities of the underlying genetic and environmental structures. We illustrate using simulations and an empirical example on childhood internalizing problems.

Keywords: dimensionality, latent variable model, genetic covariance structure modeling, common pathway model, independent pathway model

It could be argued that all psychometric modeling starts and ends with the assessment of dimensionality, that is, with the determination of the number of latent psychological attributes that are measured through a set of indicators (e.g., questionnaire items, subtest scores). Psychometrics starts with dimensionality assessment because some idea of how many attributes one intends to measure, however implicit, guides the test construction and item selection process, as well as the psychometric models one subsequently entertains as viable candidate models for the data. Ideally, it also ends with dimensionality assessment in that when the fog clears and validity issues begin to be settled, a picture emerges of which psychological attributes are measured by the test items; clearly, this question cannot be answered without simultaneously resolving the dimensionality issue.

The importance of dimensionality assessment, however, extends beyond purely psychometric issues pertaining to test construction, as dimensionality assessment impacts the research questions that psychologists pose and, as a result, the answers they obtain. For instance, via identification of item clusters, dimensionality assessment steers the allocation of items to subscales. This not only determines which subtest scores are analyzed in empirical data analysis, but also significantly influences the interpretation of latent variables hypothesized in psychological research. This interpretation may in turn result in revisions of theory concerning the nature of the psychological construct under consideration. In this way, procedures aimed at determining dimensionality play a central role in psychology; not just in the development of psychological tests, but also in the revision of interpretations of psychological constructs, and thus in the development of psychological theory (Cronbach & Meehl, 1955; Gorsuch, 1983; Haig, 2005a, 2005b; Mulaik, 1987; Rummel, 1970).

The most widely used, and in this sense most important, way of investigating dimensionality is through the statistical method of exploratory factor analysis (EFA) and related models (e.g., principal component analysis; Lawley & Maxwell, 1971). The influence of this method pervades many different areas in psychology. For instance, EFA has played an important role in the development of the five-factor model of personality (Costa & McCrae, 1985; Goldberg, 1990), the theory of childhood psychopathology associated with the Child Behavior Checklist (CBCL; Achenbach, 1966, 1991), and the Cattell–Horn–Carroll model of the structure of cognitive abilities (Carroll, 2003; Cattell, 1941; Horn, 1965). Many other examples could be listed, as EFA is one of the most widely used statistical techniques in the psychological science (Fabrigar, Wegener, MacCallum, & Strahan, 1999). In the past

This article was published Online First July 8, 2013.

Sanja Franić, Department of Biological Psychology, Vrije Universiteit Amsterdam, Amsterdam, the Netherlands; Conor V. Dolan and Denny Borsboom, Department of Psychological Methods, University of Amsterdam, Amsterdam, the Netherlands; James J. Hudziak, Department of Psychiatry and Medicine, University of Vermont; Catherina E. M. van Beijsterveldt and Dorret I. Boomsma, Department of Biological Psychology, Vrije Universiteit Amsterdam.

Correspondence concerning this article should be addressed to Sanja Franić, Department of Biological Psychology, Vrije Universiteit Amsterdam, Van der Boechorststraat 1, 1081 BT Amsterdam, the Netherlands. E-mail: s.franic@vu.nl

decades, confirmatory methods, such as item response theory modeling and confirmatory factor analysis (CFA), have been added to the repertoire for dimensionality assessment, and a good deal of work has gone into the development of heuristics to facilitate the process (Fabrigar et al., 1999; Henson & Roberts, 2006; Zwick & Velicer, 1982, 1986).

Notwithstanding the availability of these statistical tools, the evaluation of dimensionality remains difficult. For instance, in the area of cognitive abilities research, there is currently a lack of consensus on whether the *g* factor (general intelligence) can be equated with some of the more specific common factors, such as working memory or fluid reasoning (e.g., Ackerman, Beier, & Boyle, 2005; Matzke, Dolan, & Molenaar, 2010). Given the lack of sufficiently elaborate theory, research relies heavily on the intercorrelations among common factors as a source of information concerning dimensionality, conditional on the specified factor structure. Similar issues arise in psychopathology research, where some of the most prominent debates concern the origin of covariation between symptoms of two or more disorders (e.g., Angold, Costello, & Erkanli, 1999; Cramer, Waldorp, van der Maas, & Borsboom, 2010; Lilienfeld, Waldman, & Israel, 1994). For instance, the co-occurrence of symptoms of anxiety and depression is typically subject to many different explanations, ranging from those that view the disorders as different points on the same continuum to those conceptualizing them as empirically and conceptually distinct phenomena (Clark, 1989).

It would thus appear that EFA and related methods, which work purely on the observed covariation between the items,[1] do not always have sufficient resolution to firmly clinch dimensionality issues. However, it is not entirely clear why dimensionality assessment is so difficult. In the light of work done in the field of quantitative genetics (e.g., Boomsma & Molenaar, 1986; Martin & Eaves, 1977), we propose that one of the possible reasons underlying this difficulty is that item covariation, upon which EFA and related methods work, may be the result of genetic and environmental influences that differ from each other in dimensionality and structure. In the current article, we study the relationship between item covariance structures, on the one hand, and the underlying genetic and environmental covariance influences giving rise to such structures, on the other. This relationship, as we will show, may be such that it hampers obtaining a clear phenotypic dimensionality (i.e., dimensionality assessed on the basis of observed item covariation only). Incorporating genetic information in item analysis may yield a deeper understanding of the number of latent variables measured through the test scores. This provides important insights and research opportunities in the context of dimensionality assessment.

The structure of this article is as follows. We first introduce genetic factor modeling as applied in the classical twin design, and note that the genetic and environmental influences underlying the observed item covariation do not necessarily resemble each other in structure. This fact, in turn, may have implications for dimensionality assessment. We illustrate using (a) a simulation study and (b) an empirical example on childhood internalizing psychopathology. Before addressing these issues, however, it is necessary to cover the basics of the genetic factor model as applied in the classical twin design.

Genetic Covariance Structure Modeling and the Twin Design

Genetic covariance structure modeling (Martin & Eaves, 1977) is the application of structural equation modeling (Bollen, 1989; Kline, 2005) to data collected in genetically informative samples, such as siblings or adoptees (Boomsma, Busjahn, & Peltonen, 2002; Franić, Dolan, Borsboom, & Boomsma, 2012; Neale & Cardon, 1992). The fact that the samples are genetically informative (i.e., they consist of relatives whose average degree of genetic resemblance is known based on quantitative genetic theory; Falconer & Mackay, 1996) makes it possible to assess the relative contributions that genetic and environmental factors make to individual differences in observed traits (i.e., phenotypes). This is done by modeling genetic and environmental effects as contributions of latent variables to individual differences in observed traits, and estimating these contributions as regression coefficients in the linear regression of the observed traits on the latent genetic and environmental variables. The genetic and environmental latent variables themselves represent the effects of many unidentified influences: The genetic factors represent the effects of an unknown number of genes (polygenes), and the environmental factors correspond to effects of a potentially large number of unmeasured environmental influences. Measured genotypic and environmental information may also be included in the analyses (Cherny, 2008; Medland & Neale, 2010), but we do not consider this possibility in the present article.

Identification in genetic covariance structure modeling is achieved by using the information on the average degree of genetic resemblance between relatives in specifying the model. For instance, in the classical twin design the sample consists of monozygotic (MZ) and dizygotic (DZ) twin pairs. DZ twins share on average 50% of their segregating genes, whereas MZ twins share nearly their entire genome (Falconer & Mackay, 1996; van Dongen, Draisma, Martin, & Boomsma, 2012). The observed (i.e., phenotypic) covariance structure is typically modeled as a function of latent factors representing three sources of individual differences: additive genetic (A), shared environmental (C), and individual-specific environmental (E) sources.[2] Additive genetic influences are modeled by one or more A factors, which represent the total additive effects of genes relevant to the phenotypes. Based on quantitative genetic theory (Falconer & Mackay, 1996), the A factors are known to correlate 1 across MZ twins and .5 across DZ twins. Environmental influences affecting a phenotype in family members in an identical way, thereby increasing their similarity beyond what is expected based on genetic resemblance alone, are modeled by one or more C factors. Therefore, by definition, the C factors correlate unity across twins, regardless of zygosity. All environmental influences causing the observed trait to differ in two family members are modeled by one or more E factors. These influ-

[1] Or on the estimated covariation between latent distributions assumed to underlie discrete items.

[2] In addition, the trait may be influenced by nonadditive genetic effects (D). Unlike additive genetic effects, which result from additive action of genes, nonadditive genetic effects represent interactive effects of genes on the trait of interest. These will not be modeled in the present article, as the classical twin design does not allow for simultaneous estimation of A, D, C, and E effects. In our empirical example, we performed a series of univariate analyses with the results showing most of the items in our data set to conform better to an ACE than to an ADE model.

ences include environmental events to which each family member is uniquely exposed (e.g., two members of a twin pair engaging in different extracurricular activities), as well as events to which multiple family members are exposed but are affected by in a different way (e.g., both twins may be exposed to parental divorce, but the divorce may affect the trait of interest in each of the twins differently). Thus, by definition, the E factors correlate 0 across twins.

The twin design relies on several further assumptions, which include the equal environment assumption (i.e., it is assumed that MZ and DZ twins are equally correlated in their exposure to environmental factors of etiological relevance to the trait under study), equality of variance in MZ and DZ twin pairs, and absence of genotype–environment interaction (i.e., of dependency of genetic effects on the environment and vice versa), of genotype–environment correlation (i.e., of nonrandom placement of genotypes in the range of available environments), of rater bias, and of recruitment bias (e.g., Dolan, 1992; Lykken, McGue, & Tellegen, 1987; Martin & Wilson, 1982; Neale, Eaves, Kendler, & Hewitt, 1989; Stoolmiller, 1999). The presence of these phenomena does not hamper the approach, but requires them to be modeled explicitly (see, e.g., Derks, Dolan, & Boomsma, 2006). For other assumptions of the twin model, see, for example, Derks et al. (2006); Falconer and Mackay (1996); Lykken, McGue, Bouchard, and Tellegen (1990); Martin, Boomsma, and Machin (1997); Plomin, Defries, McClearn, and McGuffin (2008); and Purcell (2002).

Figure 1 depicts two examples of the particular multivariate twin model relevant to the present article. Within a given model two identical parts are specified, one for each twin. These parts relate the observed phenotypic variables to the latent common variables. For each twin, the covariation in item scores is specified to be a function of the twins' A, C, and E factors. The A, C, and E factors are correlated 1, 1, and 0 in the MZ twins and .5, 1, and 0 in the DZ twins, respectively. Note that the correlations between the A, C, and E factors within an individual are assumed to be 0, as are the cross-correlations between Twin 1 and Twin 2. Subsequently, the data are analyzed in a multigroup analysis of MZ and DZ covariance matrices. The expected covariance structure in a multivariate twin model is thus

$$\begin{pmatrix} \boldsymbol{\Sigma}_{11} & \boldsymbol{\Sigma}_{12} \\ \boldsymbol{\Sigma}_{21} & \boldsymbol{\Sigma}_{22} \end{pmatrix} = \begin{pmatrix} \boldsymbol{\Sigma}_{A} + \boldsymbol{\Sigma}_{C} + \boldsymbol{\Sigma}_{E} & r_{A}\boldsymbol{\Sigma}_{A} + \boldsymbol{\Sigma}_{C} \\ r_{A}\boldsymbol{\Sigma}_{A} + \boldsymbol{\Sigma}_{C} & \boldsymbol{\Sigma}_{A} + \boldsymbol{\Sigma}_{C} + \boldsymbol{\Sigma}_{E} \end{pmatrix}, \quad (1)$$

where, given p phenotypes (i.e., observed traits, indicators) per individual, $\boldsymbol{\Sigma}_{11}$ ($\boldsymbol{\Sigma}_{22}$) is the $p \times p$ covariance matrix of Twin 1 (Twin 2), $\boldsymbol{\Sigma}_{12}$ is the Twin 1 − Twin 2 $p \times p$ covariance matrix, and $\boldsymbol{\Sigma}_{A}$, $\boldsymbol{\Sigma}_{C}$, and $\boldsymbol{\Sigma}_{E}$ are the additive genetic, shared environmental, and unique environmental $p \times p$ covariance matrices, respectively. The coefficient r_{A} is the additive genetic twin correlation (1 for MZ twins, .5 for DZ twins). The $\boldsymbol{\Sigma}_{A}$, $\boldsymbol{\Sigma}_{C}$, and $\boldsymbol{\Sigma}_{E}$ matrices may be subject to further modeling, as depicted in Figure 1. Although the $\boldsymbol{\Sigma}_{A}$, $\boldsymbol{\Sigma}_{C}$, and $\boldsymbol{\Sigma}_{E}$ covariance matrices may be subjected to any kind of a covariance structure model (see Boomsma & Molenaar, 1987; Eaves, Long, & Heath, 1986; Neale & Cardon, 1992), we focus on the type of model depicted in Figure 1.

The first model in Figure 1 is a *common pathway model* (Kendler, Heath, Martin, & Eaves, 1987), also known as the psycho-

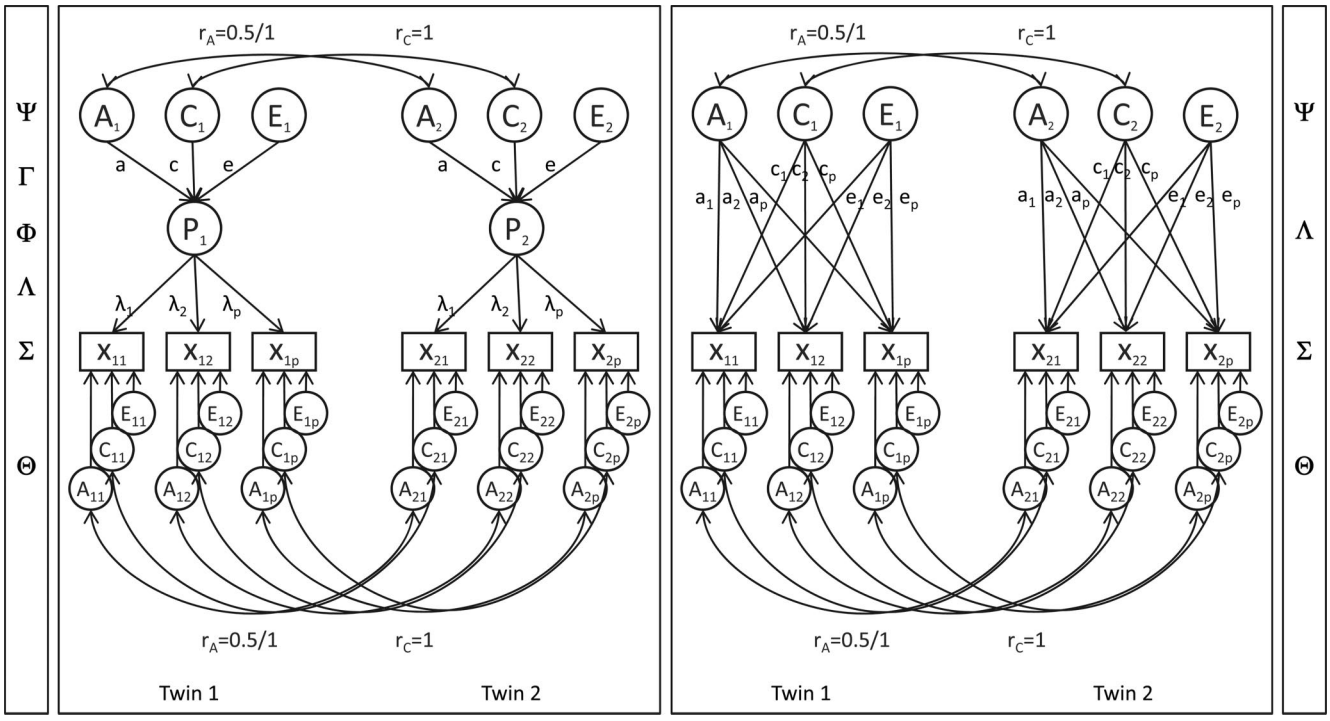


Figure 1. A common pathway (left) and an independent pathway (right) genetic factor model. Matrix names on the sides correspond to notation in the text. Note: As indicated by the notation, the a, c, e, and λ parameters are subject to equality constraints over Twin 1 and Twin 2. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor.

metric factor model (McArdle & Goldsmith, 1990). In common pathway models, all the A, C, and E influences on item covariation are mediated by a latent variable, henceforth referred to as the *psychometric factor* (factors P_1 and P_2 in Figure 1). P_1 and P_2 may be viewed as phenotypic latent factors (i.e., latent factors obtained in factor analysis as typically applied in psychological research; e.g., "neuroticism" or "g"). In common pathway models, the psychometric factor acts as a mediator of the genetic and environmental effects.

The second model is the *independent pathway model* (Kendler et al., 1987), also known as the biometric factor model (McArdle & Goldsmith, 1990). An example of this model is depicted in the right panel of Figure 1. In independent pathway models, there is no phenotypic latent variable that mediates genetic and environmental effects on the item responses. Rather, the A, C, and E factors influence item responses directly. In terms of the phenotypic covariance matrix of item responses, we can convey the common and the independent pathway model as follows:

$$\boldsymbol{\Sigma}_{11} = \boldsymbol{\Sigma}_{22} = \boldsymbol{\Lambda}\boldsymbol{\Phi}\boldsymbol{\Lambda}^t + \boldsymbol{\Theta}_{cp} = \boldsymbol{\Lambda}\left(\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C + \boldsymbol{\Phi}_E\right)\boldsymbol{\Lambda}^t + \boldsymbol{\Theta}_{cp} \quad (2)$$

$$= \boldsymbol{\Lambda}\left(\boldsymbol{\Gamma}_A\boldsymbol{\Psi}_A\boldsymbol{\Gamma}_A^t + \boldsymbol{\Gamma}_C\boldsymbol{\Psi}_C\boldsymbol{\Gamma}_C^t + \boldsymbol{\Gamma}_E\boldsymbol{\Psi}_E\boldsymbol{\Gamma}_E^t\right)\boldsymbol{\Lambda}^t + \Theta_{cp},$$

$$\boldsymbol{\Sigma}_{21} = \boldsymbol{\Sigma}_{12} = \boldsymbol{\Lambda}\left(r_A\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C\right)\boldsymbol{\Lambda}^t + \boldsymbol{\Theta}_{cp21}$$

$$= \Lambda\left(r_A\boldsymbol{\Gamma}_A\boldsymbol{\Psi}_A\boldsymbol{\Gamma}_A^t + \boldsymbol{\Gamma}_C\boldsymbol{\Psi}_C\boldsymbol{\Gamma}_C^t\right)\boldsymbol{\Lambda}^t + \boldsymbol{\Theta}_{cp21},$$

$$\boldsymbol{\Sigma}_{11} = \boldsymbol{\Sigma}_{22} = \boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Lambda}_E\boldsymbol{\Psi}_E\boldsymbol{\Lambda}_E^t + \boldsymbol{\Theta}_{ip}, \quad (3)$$

$$\boldsymbol{\Sigma}_{21} = \boldsymbol{\Sigma}_{12} = r_A\boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Theta}_{ip21},$$

respectively. Here $\boldsymbol{\Lambda}$ (in the common pathway model) and $\boldsymbol{\Lambda}_A$, $\boldsymbol{\Lambda}_C$, and $\boldsymbol{\Lambda}_E$ (in the independent pathway model) matrices contain the loadings of the indicators on the psychometric factor and on the biometric (A, C, and E) factors, respectively, and $\boldsymbol{\Psi}_A$, $\boldsymbol{\Psi}_C$, and $\boldsymbol{\Psi}_E$ are the covariance matrices of the A, C, and E factors. In the common pathway model, the covariance matrix of the psychometric factor, $\boldsymbol{\Phi}$, equals $\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C + \boldsymbol{\Phi}_E$, that is, $\boldsymbol{\Gamma}_A\boldsymbol{\Psi}_A\boldsymbol{\Gamma}_A{}^t + \boldsymbol{\Gamma}_C\boldsymbol{\Psi}_C\boldsymbol{\Gamma}_C{}^t + \boldsymbol{\Gamma}_E\boldsymbol{\Psi}_E\boldsymbol{\Gamma}_E{}^t$, where $\boldsymbol{\Gamma}_A$, $\boldsymbol{\Gamma}_C$, and $\boldsymbol{\Gamma}_E$ are the vectors of factor loadings $\boldsymbol{\Gamma}_A$ = [a], $\boldsymbol{\Gamma}_C$ = [c], $\boldsymbol{\Gamma}_E$ = [e]. Note that in both models the diagonal matrices $\boldsymbol{\Theta}$ (denoted $\boldsymbol{\Theta}_{cp}$ and $\boldsymbol{\Theta}_{ip}$, as they may vary over the models) contain the residuals of the items in the model and $\boldsymbol{\Theta}_{cp21}$ and $\boldsymbol{\Theta}_{ip21}$ matrices contain the Twin 1 − Twin 2 covariance among the residuals. The residual matrices may be subjected to their own decomposition, that is, $\boldsymbol{\Theta} = \boldsymbol{\Theta}_A + \boldsymbol{\Theta}_C + \boldsymbol{\Theta}_E$ and $\boldsymbol{\Theta}_{21} = r_A\boldsymbol{\Theta}_A + \boldsymbol{\Theta}_C$ (Neale & Cardon, 1992), as depicted in Figure 1.[3] It is immediately clear from Figure 1 that the common pathway model differs from the independent pathway model in the presence of the psychometric factors P_1 and P_2. As we explain next, this difference can have important implications for dimensionality assessment.

Phenotypic Latent Variable Model and the Common Pathway Model

In the present article, we distinguish between genetic factor models (introduced above) and phenotypic factor models. By *phenotypic factor model* we refer to the factor model as usually formulated and applied in psychological research. The term *phenotypic* is used because the model is applied only to the observed (i.e., phenotypic) covariation; no genetic information is used. The eight-factor cross-informant model of the CBCL (Achenbach, 1966) and the five-factor model of personality (McCrae & Costa, 1999; McCrae & John, 1992) are examples of a phenotypic factor model.

The common pathway model bears a number of similarities to the phenotypic factor model. Notably, both the phenotypic factor model and the common pathway model are based on the assumption that all covariation in item responses is attributable to one or more latent variables. In phenotypic factor modeling, this is formulated as the requirement of measurement invariance: influences of all external variables affecting covariation in item responses run only via the latent variable (Mellenbergh, 1989; Meredith, 1993). Likewise, in common pathway modeling one assumes that all the A, C, and E influences on item covariation run only via the psychometric factor. That is, there are no direct effects of A, C, and E on the items.[4]

The assumption of full mediation of external influences by a latent variable has strong implications. For instance, different external variables affecting a set of item responses via the same latent variable exert the same magnitude of influence relative to each other on all the items that depend on that latent variable. For instance, if an A and a C variable affect a set of items via the same psychometric factor, the magnitude of influence exerted by the variable A on any individual item will be a scalar multiple of the magnitude of influence exerted by the variable C on that item, and this scalar multiple (k) will be a constant across all the items depending on this psychometric factor. This can be seen from the regression equations describing the common pathway model, for example (in terms of the symbols used in Figure 1),

$$x_{11} = \lambda_1\left(aA_1 + cC_1 + eE_1\right) + \varepsilon_{11} = \lambda_1 aA_1 + \lambda_1 cC_1 + \lambda_1 eE_1 + \varepsilon_{11}, \quad (4)$$

$$x_{12} = \lambda_2\left(aA_1 + cC_1 + eE_1\right) + \varepsilon_{12} = \lambda_2 aA_1 + \lambda_2 cC_1 + \lambda_2 eE_1 + \varepsilon_{12},$$

etc. (note that $\varepsilon_{11} = A_{11} + C_{11} + E_{11}$ in Figure 1, etc.). In contrast, the independent pathway model imposes no proportionality constraints on the factor loadings, for example,

$$x_{11} = a_1A_1 + c_1C_1 + e_1E_1 + \varepsilon_{11}, \quad (5)$$

$$x_{12} = a_2A_1 + c_2C_1 + e_2E_1 + \varepsilon_{12},$$

etc. Specifically, letting k denote a positive constant, we note that the introduction of the constraints $a_1/a_2 = c_1/c_2 = e_1/e_2 = k$ renders Equation 4 and Equation 5 equivalent (Yung, Thissen, & McLeod, 1999). Thus, the common pathway model makes explicit an assumption of the phenotypic latent variable model concerning the sources of item covariation—all influences on item covariation run via the phenotypic latent variable. This means, barring cases of model equivalence, that *a latent variable model cannot hold unless the corresponding common pathway model holds*. Because any given latent variable hypothesis implies a corresponding common pathway model, a refutation of that common pathway model constitutes evidence against the latent variable hypothesis.

[3] A more detailed account of the residual decomposition is provided in Appendix A.

[4] As such, the common pathway model may be interpreted as a MIMIC model (Jöreskog & Goldberger, 1975), as the causal influences of A, C, and E factors on the observed responses are mediated by the phenotypic factor. However, in this case the multiple causes are latent rather than observed variables as in the Jöreskog and Goldberger (1975) case.

For this reason, one may test the latent variable hypothesis by comparing the fit of a common pathway model to that of a corresponding independent pathway model. Specifically, if a model in which all the A, C, and E factors exert direct influence on the phenotype fits the data statistically better than a model in which these influences are mediated by a phenotypic latent variable, this would provide evidence against the hypothesis that the effects on the observed item covariation are completely mediated by the phenotypic latent variable. In that case the latent factors employed in the phenotypic factor model are no more than an amalgamation of the direct influences of the A, C, and E factors on the observed item responses. This would have implications for the substantive interpretation of such factors as well-defined, causal entities that produce the observed item covariation (e.g., Borsboom, Mellenbergh, & van Heerden, 2003; Haig, 2005a, 2005b).[5] If, on the other hand, an independent pathway model does not fit the data better than the corresponding common pathway model, this would provide support for the structure employed in the common pathway model and substantiation for the corresponding phenotypic latent variable hypothesis. Comparison of an independent pathway model and a common pathway model may be conducted using a likelihood ratio test, because, as shown above, a common pathway model can be derived from an independent pathway model by imposing appropriate proportionality constraints on the factor loadings (i.e., the models are nested).

The logic underlying the present approach is essentially the same as that involved in measurement invariance research and multiple indicators multiple causes (MIMIC) modeling: The latent variable is required to screen off the effects of genetic and environmental factors (in Pearl, 2000, terminology, the latent variable *d* separates genes and environment from the item responses). However, what makes the genetic case special is that the A, C, and E factors (a) plausibly determine the variance of the latent variable completely and (b) can be highly structured by applying standard genetic theory to genetically informative data. This allows for unique possibilities to investigate hypotheses on the origins of structures seen in the correlations among psychometric items. To demonstrate that the proposed methodology works under realistic conditions, we next provide a simulation example.

Simulation Study

To illustrate the relationship between the observed association structures and the underlying genetic and environmental structures, we simulated several data sets. In each data set, a different pattern of genetic and environmental effects gives rise to the observations. These patterns depart progressively from the ideal situation of a common pathway model. As we will show, such departures lead to psychometrically indeterminate covariation structures, in the sense that standard psychometric research practices would not (and in fact could not) converge on correct assessments of the underlying dimensionality. However, we also show that attending to genetic information, present in the widely available twin data sets, allows one to resolve the psychometric puzzle accurately (i.e., to better understand the dimensionality of the data set).

In total, four data sets were simulated. In the first data set (Data Set 0) the data are consistent with a common pathway model. In the three subsequent data sets (Data Sets 1–3), the assumption of the common pathway model concerning the proportionality of the genetic and environmental effects on the items is violated to an increasing extent. This was achieved by manipulating the dimensionalities of the latent A, C, and E structures (i.e., the order of the covariance matrices $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$). Figure 2 outlines the general structure of this simulation.

Each of the four data sets comprises 12 continuous normally distributed variables per individual (24 variables per twin pair), for 1,000 MZ and 1,000 DZ twin pairs. We used exact data simulation (i.e., the simulated data fitted the generating model exactly; e.g., van der Sluis, Dolan, Neale, & Posthuma, 2008). We limit the current presentation to a single set of parameter values (given in Table 1),[6] which we do not vary over the four simulations. The manipulation involves only (a) varying the dimensions of the $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$ covariance matrices (and the dimensions of the corresponding $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices) and (b) varying the patterns of factor loadings within the $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices. However, all simulations were performed with five sets of parameter values, and our conclusions were found to be invariant.[7] The simulation script provided in Appendix B may be used to verify the generality of our inferences. In the following text, we will first review the four generating models. Subsequently, we present the results of dimensionality assessment for the four data sets.

Models

The baseline model (Model 0, depicted in the first panel of Figure 2) is a common pathway model. The expected phenotypic covariance structure ($\mathbf{\Sigma}_{CP}$) under this model is

$$\begin{pmatrix} \mathbf{\Sigma}_{11} & \mathbf{\Sigma}_{12} \\ \mathbf{\Sigma}_{21} & \mathbf{\Sigma}_{22} \end{pmatrix} = \begin{pmatrix} \mathbf{\Lambda}(\mathbf{\Phi}_A + \mathbf{\Phi}_C + \mathbf{\Phi}_E)\mathbf{\Lambda}^t + \mathbf{\Theta}_{cp} & \mathbf{\Lambda}(r_A\mathbf{\Phi}_A + \mathbf{\Phi}_C)\mathbf{\Lambda}^t + \mathbf{\Theta}_{cp21} \\ \mathbf{\Lambda}(r_A\mathbf{\Phi}_A + \mathbf{\Phi}_C)\mathbf{\Lambda}^t + \mathbf{\Theta}_{cp21} & \mathbf{\Lambda}(\mathbf{\Phi}_A + \mathbf{\Phi}_C + \mathbf{\Phi}_E)\mathbf{\Lambda}^t + \mathbf{\Theta}_{cp} \end{pmatrix},$$

where $\mathbf{\Sigma}_{11}$ ($\mathbf{\Sigma}_{22}$) is the 12 × 12 phenotypic covariance matrix of Twin 1 (Twin 2); $\mathbf{\Sigma}_{12}$ is the 12 × 12 Twin 1 − Twin 2 phenotypic covariance matrix; $\mathbf{\Lambda}$ is a vector containing the loadings of the indicators on the psychometric factor; $\mathbf{\Phi}_A$, $\mathbf{\Phi}_C$, and $\mathbf{\Phi}_E$ are the A, C, and E variance components of the psychometric factor, respectively; coefficient r_A is the additive genetic twin correlation (1 for MZ twins, .5 for DZ twins); $\mathbf{\Theta}_{cp}$ is a diagonal matrix containing the residuals of the items; and $\mathbf{\Theta}_{cp21}$ and $\mathbf{\Theta}_{ip21}$ are matrices containing the Twin 1 − Twin 2 covariance among the residuals. In the present case, the variance of each of the items in $\mathbf{\Sigma}_{11}$ ($\mathbf{\Sigma}_{22}$) is 1, and the correlations between the indicators range from .12 to .62.

The model above may also be expressed in terms of parameters of an independent pathway model, as presented in Table 1. In this independent pathway model, the expected covariance structure ($\mathbf{\Sigma}_{IP}$) is:

[5] This would, however, not diminish the usefulness of phenotypic latent variables as a means of summarizing data or their utility as predictors. In addition, the specific reasons for rejecting the common pathway model may be local (due only to a subset of observed variables), and thus the violation may be accommodated by the addition of parameters or by the removal of offending variables.

[6] The table does not detail the parameters of the ACE model for the residuals given our focus on dimensionality assessment; these are given in Appendix A and the simulation script (Appendix B).

[7] Details on the five sets of parameter values may be obtained from the first author.

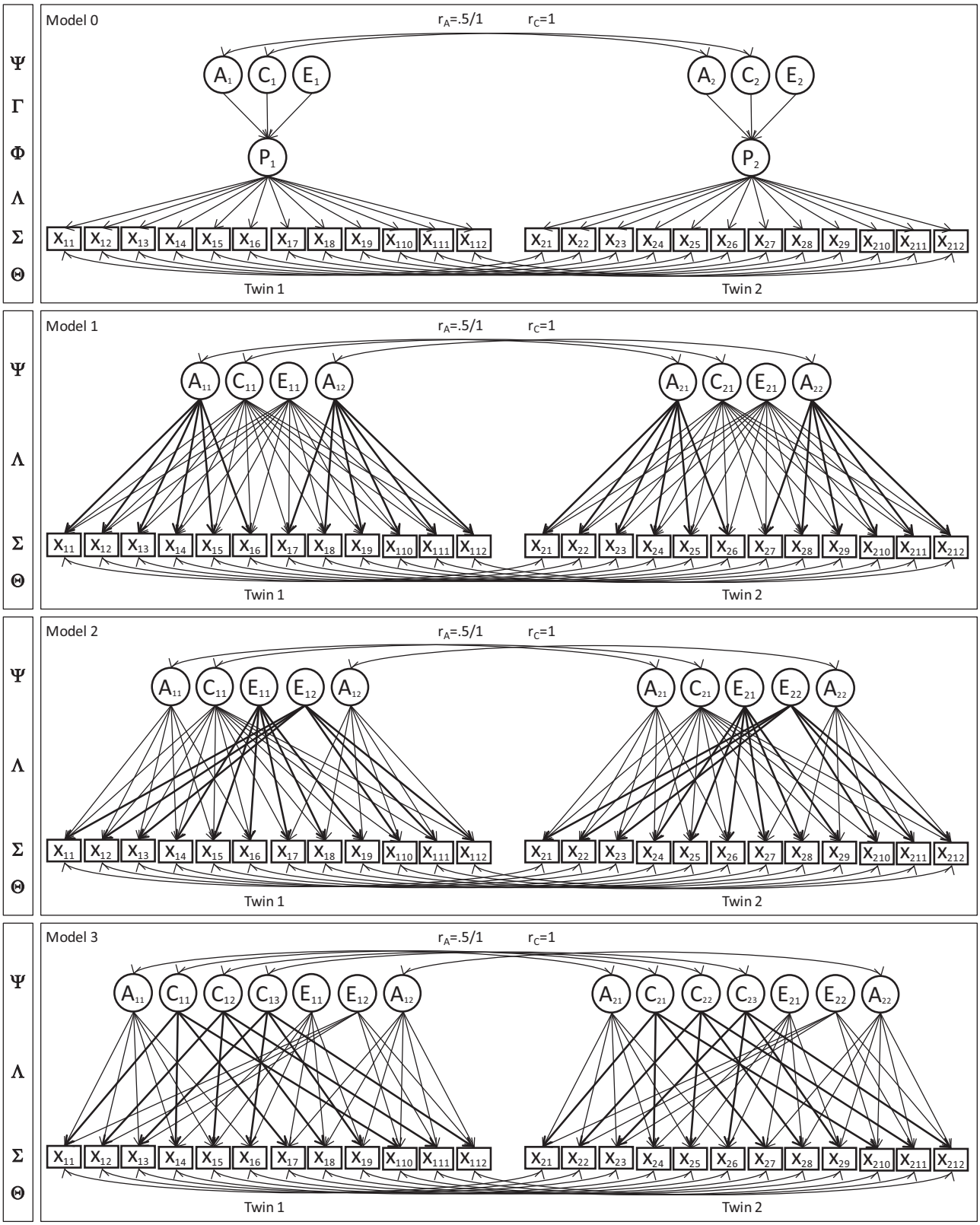


Figure 2. Path diagrams of Models 0–3. Matrix names on the left correspond to notation in the text. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor.

Table 1
Parameters of Models 0–3

Model 0 in terms of common pathway parameters

$\boldsymbol{\Lambda}^t$ = [✓.1, ✓.15, ✓.2, ✓.25, ✓.3, ✓.35, ✓.4, ✓.45, ✓.5, ✓.55, ✓.6, ✓.65]
$\boldsymbol{\Gamma}_A$ = [✓.5], $\boldsymbol{\Gamma}_C$ = [✓.3], $\boldsymbol{\Gamma}_E$ = [✓.2]
$\boldsymbol{\Psi}_A = \boldsymbol{\Psi}_C = \boldsymbol{\Psi}_E$ = [1]
$\boldsymbol{\Theta}_{cp} = \mathbf{I} - \text{diag}[\boldsymbol{\Lambda}(\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C + \boldsymbol{\Phi}_E)\boldsymbol{\Lambda}^t] = \mathbf{I} - \text{diag}[\boldsymbol{\Lambda}(\boldsymbol{\Gamma}_A\boldsymbol{\Psi}_A\boldsymbol{\Gamma}_A{}^t + \boldsymbol{\Gamma}_C\boldsymbol{\Psi}_C\boldsymbol{\Gamma}_C{}^t + \boldsymbol{\Gamma}_E\boldsymbol{\Psi}_E\boldsymbol{\Gamma}_E{}^t)\boldsymbol{\Lambda}^t]$
= diag(.9, .85, .8, .75, .7, .65, .6, .55, .5, .45, .4, .35)
$\boldsymbol{\Theta}_{cp21mz} = .8^*\mathbf{I} - \text{diag}(\boldsymbol{\Lambda}(\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C)\boldsymbol{\Lambda}^t)$
= diag(.72, .68, .64, .6, .56, .52, .48, .44, .4, .36, .32, .28)
$\boldsymbol{\Theta}_{cp21dz} = .55^*\mathbf{I} - \text{diag}(\boldsymbol{\Lambda}(.5\boldsymbol{\Phi}_A + \boldsymbol{\Phi}_C)\boldsymbol{\Lambda}^t)$
= diag(.495, .4675, .44, .4125, .385, .3575, .33, .3025, .275, .2475, .22, .1925)

Model 0 in terms of independent pathway parameters

$\boldsymbol{\Lambda}_A{}^t = \boldsymbol{\Gamma}_A\boldsymbol{\Lambda}^t$ = [✓.05, ✓.075, ✓.1, ✓.125, ✓.15, ✓.175, ✓.2, ✓.225, ✓.25, ✓.275, ✓.3, ✓.325]
$\boldsymbol{\Lambda}_C{}^t = \boldsymbol{\Gamma}_C\boldsymbol{\Lambda}^t$ = [✓.03, ✓.045, ✓.06, ✓.075, ✓.08, ✓.105, ✓.120, ✓.135, ✓.150, ✓.165, ✓.180, ✓.195]
$\boldsymbol{\Lambda}_E{}^t = \boldsymbol{\Gamma}_E\boldsymbol{\Lambda}^t$ = [✓.02, ✓.03, ✓.04, ✓.05, ✓.06, ✓.07, ✓.08, ✓.09, ✓.10, ✓.11, ✓.12, ✓.13]
$\boldsymbol{\Psi}_A = \boldsymbol{\Psi}_C = \boldsymbol{\Psi}_E$ = [1], $\boldsymbol{\Theta}_{ip} = \boldsymbol{\Theta}_{cp}$, $\boldsymbol{\Theta}_{ip21mz} = \boldsymbol{\Theta}_{cp21mz}$, $\boldsymbol{\Theta}_{ip21dz} = \boldsymbol{\Theta}_{cp21dz}$

Model 1

$$\boldsymbol{\Psi}_A = \text{diag}(1, 1)$$
$$\boldsymbol{\Lambda}_A^t = \begin{pmatrix} \surd.05 & \surd.075 & \surd.1 & \surd.125 & \surd.15 & \surd.175 & & & & & & \\ & & & & & & \surd.2 & \surd.225 & \surd.25 & \surd.275 & \surd.3 & \surd.325 \end{pmatrix}$$

Model 2

$$\boldsymbol{\Psi}_E = \text{diag}(1,\ 1)$$
$$\boldsymbol{\Lambda}_E^t = \begin{pmatrix} \surd.02 & \surd.03 & \surd.04 & & & & & & & \surd.11 & \surd.12 & \surd.13 \\ & & & \surd.05 & \surd.06 & \surd.07 & \surd.08 & \surd.09 & \surd.10 & & & \end{pmatrix}$$

Model 3

$$\boldsymbol{\Psi}_C = \text{diag}(1,\ 1,\ 1)$$
$$\boldsymbol{\Lambda}_C^t = \begin{pmatrix} \surd.03 & & & \surd.075 & & & \surd.120 & & & \surd.165 & & \\ & \surd.045 & & & \surd.08 & & & \surd.135 & & & \surd.180 & \\ & & \surd.06 & & & \surd.105 & & & \surd.150 & & & \surd.195 \end{pmatrix}$$

Note. The models are conveyed in terms of parameters that differ from the preceding model. For instance, the parameter matrices not listed under Model 1 ($\boldsymbol{\Lambda}_C$, $\boldsymbol{\Lambda}_E$, $\boldsymbol{\Psi}_C$, $\boldsymbol{\Psi}_E$, $\boldsymbol{\Theta}_{ip}$, and $\boldsymbol{\Theta}_{ip21}$) equal those in Model 0. In addition, the factor loading parameters are conveyed in terms of square roots, as this gives straightforward information on the proportion of variance explained (e.g., a factor loading of ✓.1 indicates $\surd.1^2 = .1$ explained variance). $\boldsymbol{\Lambda}$ = vector containing the loadings of the indicators on the psychometric factor; $\boldsymbol{\Gamma}_A$, $\boldsymbol{\Gamma}_C$, $\boldsymbol{\Gamma}_E$ = vectors of factor loadings of the psychometric factor on the A, C, and E factors; $\boldsymbol{\Psi}_A$, $\boldsymbol{\Psi}_C$, $\boldsymbol{\Psi}_E$ = covariance matrices of the A, C, and E factors; $\boldsymbol{\Theta}_{cp} = \boldsymbol{\Theta}_{ip}$ = 12 × 12 diagonal matrix containing the residual item variances; $\boldsymbol{\Theta}_{cp21mz} = \boldsymbol{\Theta}_{ip21mz}$ = 12 × 12 diagonal matrix of Twin 1 − Twin 2 covariances among monozygotic twins; $\boldsymbol{\Theta}_{cp21dz} = \boldsymbol{\Theta}_{ip21dz}$ = 12 × 12 diagonal matrix of Twin 1 − Twin 2 covariances among dizygotic twins; $\boldsymbol{\Lambda}_A$, $\boldsymbol{\Lambda}_C$, $\boldsymbol{\Lambda}_E$ = matrices containing direct factor loadings of the items on the A, C, and E factors.

$$\begin{pmatrix} \boldsymbol{\Sigma}_{11} & \boldsymbol{\Sigma}_{12} \\ \boldsymbol{\Sigma}_{21} & \boldsymbol{\Sigma}_{22} \end{pmatrix} = \begin{pmatrix} \boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Lambda}_E\boldsymbol{\Psi}_E\boldsymbol{\Lambda}_E^t + \boldsymbol{\Theta}_{ip} & r_A\boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Theta}_{ip21} \\ r_A\boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Theta}_{ip21} & \boldsymbol{\Lambda}_A\boldsymbol{\Psi}_A\boldsymbol{\Lambda}_A^t + \boldsymbol{\Lambda}_C\boldsymbol{\Psi}_C\boldsymbol{\Lambda}_C^t + \boldsymbol{\Lambda}_E\boldsymbol{\Psi}_E\boldsymbol{\Lambda}_E^t + \boldsymbol{\Theta}_{ip} \end{pmatrix}.$$

Here $\boldsymbol{\Lambda}_A$, $\boldsymbol{\Lambda}_C$, and $\boldsymbol{\Lambda}_E$ vectors contain the loadings of the indicators on the A, C, and E factors, respectively, and the residual matrices $\boldsymbol{\Theta}_{ip}$, $\boldsymbol{\Theta}_{ip21mz}$, and $\boldsymbol{\Theta}_{ip21dz}$ are equal to those in the common pathway model. In the case of the present model (Model 0), $\boldsymbol{\Sigma}_{CP} = \boldsymbol{\Sigma}_{IP}$. Note that the independent pathway factor loading parameters above are fully consistent with a common pathway model; that is, the elements of $\boldsymbol{\Lambda}_A$, $\boldsymbol{\Lambda}_C$, and $\boldsymbol{\Lambda}_E$ matrices satisfy the proportionality constraint $a_i/a_{i+1} = c_i/c_{i+1} = e_i/e_{i+1} = k$, where $i = 1, \ldots 11$ and k is a constant). Taking these parameter values as a point of departure, we specify the three subsequent models.

In Model 1, the additive genetic influences on the items are represented by two orthogonal A factors per twin. Note that this model (depicted in the second panel of Figure 2) may alternatively be represented as a common pathway model with two phenotypic factors per twin, each factor being a function of its own A, C, and E factor (where the two A factors are uncorrelated and the two C

factors, as well as the two E factors, correlate unity). In this sense, the model does not represent a severe violation of the common pathway structure.

In Model 2 (depicted in the third panel of Figure 2), the structure employed in Model 1 is further altered, by increasing the dimensionality of the E structure. This model represents a more severe violation of the common pathway structure, as the items here no longer cluster identically with regard to A and E influences (i.e., the patterns of factor loadings in the $\mathbf{\Lambda}_A$ and $\mathbf{\Lambda}_E$ matrices differ from each other). For instance, sets of items that form a unidimensional structure with respect to additive genetic influences are two-dimensional with respect to unique environmental influences. In Model 3 (fourth panel of Figure 2), the common pathway structure is further violated by increasing the dimensionality of the C structure. Here, the clustering of the items is markedly different with regard to the A, C, and E influences; thus, the observed dimensionality is a function of A, C, and E influences that severely violate the common pathway structure.

Analyses

The analyses of the data sets consisted of two parts. In the first part, the aim was to examine the effect that the violations of the common pathway structure had on the phenotypic dimensionality estimates. To this end, the dimensionality of the data sets was assessed using EFA. The phenotypic latent factors obtained in the EFA were subsequently used as a basis for specifying confirmatory genetic factor common pathway models. As in standard genetic research, here we decomposed the variation in the latent factors obtained in the phenotypic EFA into genetic and environmental components. In the second part, the aim was to obtain a clearer indication of the data-generating mechanism by disposing of the hypotheses concerning the number of latent variables in the model, and applying independent pathway modeling in a purely exploratory manner, to uncover the (possibly different) structures of the A, C, and E influences. Specifically, we used EFA to determine the possibly different dimensionalities of the covariance matrices $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$, in terms of the latent covariance matrices $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$ (see Equations 1 and 3). Here the dimensionality of the observed covariance matrix is a function of the A, C, and E covariance structures, which may differ in dimensionality, and in no way satisfy the common pathway model. The advantage of this is that it provides an insight into the dimensionality of the phenotypic structure that does not assume, but does not exclude, the common pathway model. The analyses were performed using Mplus (Muthén & Muthén, 2007a), Mx (Neale, 2000), and R (R Development Core Team, 2009).[8] In evaluating model fit, we used the comparative fit index (CFI), the Tucker–Lewis index (TLI), and the root-mean-square error of approximation (RMSEA).

Table 2
Fit Measures Obtained in Phenotypic Exploratory Factor Analysis of Data Sets 1–3

Factor	χ^2	*df*	*p*	RMSEA
Data Set 1				
1f	325.3	54	0	.0003
2f	0	43	1	0
3f	0	33	1	0
4f	0	24	1	0
Data Set 2				
1f	470.0	54	0	.0004
2f	133.6	43	1	.0002
3f	0	33	1	0
4f	0	24	1	0
Data Set 3				
1f	543.8	54	0	.0004
2f	328.5	43	0	.0004
3f	196.8	33	0	.0004
4f	89.5	24	0	.0003
5f	0	16	1	0

Note. RMSEA = root-mean-square error of approximation.

Table 3
Factor Correlations Obtained in Phenotypic Exploratory Factor Analysis (EFA) of Data Sets 1–3

Factor	1f	2f	3f	4f	5f
Two-factor EFA solution Data Set 1					
1f	1				
2f	.04	1			
Three-factor EFA solution Data Set 2					
1f	1				
2f	.24	1			
3f	−.68	.16	1		
Five-factor EFA solution Data Set 3					
1f	1				
2f	.09	1			
3f	−.06	.09	1		
4f	.04	.04	.23	1	
5f	−.02	−.01	.26	.25	1

Results

Given that Model 0 has a unidimensional structure and was used only as a baseline model from which parameter values were derived, we limit the presentation to the results obtained in analyses of Data Sets 1–3.

Data Set 1. Seeing as Model 1 can be viewed as a two-factor common pathway model in which the two C factors, as well as the two E factors, correlate unity, one can simply accommodate the violation of the one-factor common pathway structure by fitting a two-factor model. The phenotypic EFA results, a summary of which is provided in Tables 2–4 (see also Figure 3), reflect this: A two-factor EFA solution provides a perfect fit to the data, as do a two-factor common pathway model ($\chi^2 = 0$, $df = 581$, $p = 1$, RMSEA = 0, CFI = 1, TLI = 1) and a two-A, two-C, two-E independent pathway model ($\chi^2 = 0$, $df = 508$, $p = 1$, RMSEA = 0, CFI = 1, TLI = 1) based on this two-factor EFA solution. Note that perfect fit is associated with chi-square values of 0 because we

[8] All scripts may be obtained from the first author upon request. We alternated between Mplus and Mx because Mplus estimates the polychoric correlations very efficiently, whereas Mx's matrix-based syntax is very convenient in fitting models involving high-dimensional Cholesky decompositions. R was used for its data simulation features.

Table 4
Promax-Rotated Factor Loadings Obtained in Phenotypic Exploratory Factor Analysis of Data Sets 1–3

	Data Set 1		Data Set 2			Data Set 3					Data Set 2			
Factor	P1	P2	P1	P2	P3	P1	P2	P3	P4	P5	P1	P2	P3	P4
x1		**.309**		.191	**.253**					**.289**				**.459**
x2		**.378**		.234	**.310**		−.167		**.281**	.108		**.285**	.215	
x3		**.437**		.270	**.358**	.267	−.209	**.309**		.127		**.329**	.249	
x4		**.489**		**.507**	.121	−.208	.249	.227		**.358**		**.507**		
x5		**.535**		**.555**	.132	−.157		.243	.429			**.555**		
x6		**.578**		**.600**	.143			**.562**				**.600**		
x7	**.633**		**.521**	.170			**.639**			.297	**.596**	.107		
x8	**.671**		**.553**	.181			.402		**.449**	−.109	**.632**	.113		
x9	**.708**		**.583**	.190		.311	**.398**	.367		−.113	**.666**	.12		
x10	**.742**		**.798**	−.105	.352	.362	.365	−.175		**.495**	**.737**		.51	
x11	**.775**		**.833**	−.109	.367	**.478**		−.191	.474		**.770**		.533	
x12	**.807**		**.867**	−.114	.382	**.782**		.209			**.801**		.554	
Prop var	.263	.107	.251	.102	.063	.102	.084	.065	.058	.050				
Prop cum var	.263	.371	.251	.353	.416	.102	.186	.250	.309	.359				

Note. Highest factor loadings for each item appear in bold. Prop (cum) var = proportion of (cumulative) variance explained.

used exact data simulation. The parameter estimates obtained in genetic factor modeling indicate that C and E are unidimensional (the correlations between the two C factors in both the common and the independent pathway model are 1, as are the correlations between the two E factors), whereas A may be represented by two orthogonal factors. The structure depicted in the second panel of Figure 2 therefore need not preclude accurate dimensionality assessment. However, one might consider situations in which the data-generating structure is less consistent with the common pathway model; in the following examples we consider more severe violations of the common pathway structure.

Data Set 2. In Model 2, the $\boldsymbol{\Sigma}_A$ and $\boldsymbol{\Sigma}_E$ matrices are both two-dimensional, but the items cluster differently with regard to A and E influences (e.g., clusters of items that form a unidimensional structure with respect to additive genetic influences are two-dimensional with respect to unique environmental influences). Note that the data-generating structure may still be accommodated by a common pathway model with four phenotypic factors, each affecting three items. However, as common pathway analyses are confirmatory in nature and predicated on the results of phenotypic analyses, we first investigated whether phenotypic EFA correctly indicated the number of phenotypic factors needed to account for the observed covariance structure.

The results of the EFA are shown in Table 2. Here, both a one-factor solution and a two-factor solution were clearly rejected by the chi-square statistic, but in a three-factor solution both the chi-square and the RMSEA indicated a perfect fit ($\chi^2 = 0$, $df = 33$, $p = 1$, RMSEA = 0, CFI = 1, TLI = 1). In the four-factor solution the same was the case, although the model (based on promax rotation; presented in Table 4) does not correspond to the data-generating structure. Moreover, in the four-factor solution none of the items appear to be best represented by the third factor, and only one item loads substantially (factor loading above $\sqrt{.025}$) on the fourth factor. Considering the fit statistics and the factor structure given in Tables 2 and 4, it appears that in the standard situation of dimensionality assessment the three-factor solution would represent a compelling choice.

On the basis of this three-factor EFA solution (detailed in Table 4), we specified a three-factor common pathway model and a corresponding independent pathway model, depicted in Figure 4. In both of these models, the phenotypic covariation in Twin 1 (Twin 2) is a function of three mutually correlated A (C, E) factors (i.e., $\boldsymbol{\Psi}_A$, $\boldsymbol{\Psi}_C$, and $\boldsymbol{\Psi}_E$ are 3×3 matrices with freely estimated off-diagonal elements). Although inclusion of cross-loadings improves model fit, we specify simple structure models given our focus on dimensionality assessment. For the common pathway model, the fit measures were $\chi^2(577) = 2158$, $p < .001$, RMSEA = .052, CFI = .944, TLI = .944, and for the independent pathway model, $\chi^2(507) = 1148$, $p < .001$, RMSEA = .036, CFI = .976, TLI = .974. Additional analyses showed that inclusion of cross-loadings (as indicated by the EFA solution) improves model fit for both the common pathway model and the independent pathway model; however, even then, the parameter estimates remain somewhat biased. Thus, even if one assumed the presence of cross-loadings, these models are still unable to precisely convey the actual A, C, and E effects on the items. If

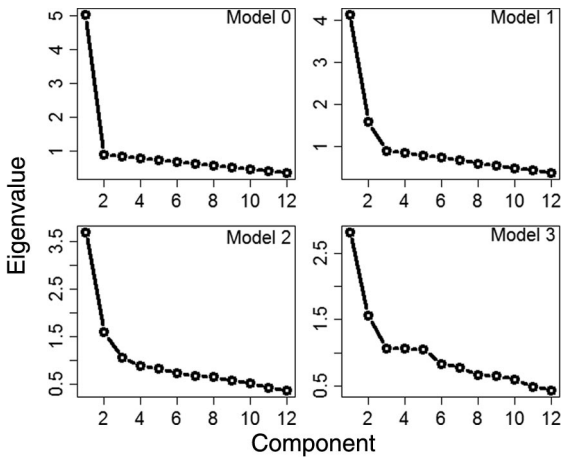


Figure 3. Eigenvalues of the phenotypic covariance matrices for Models 0–3.

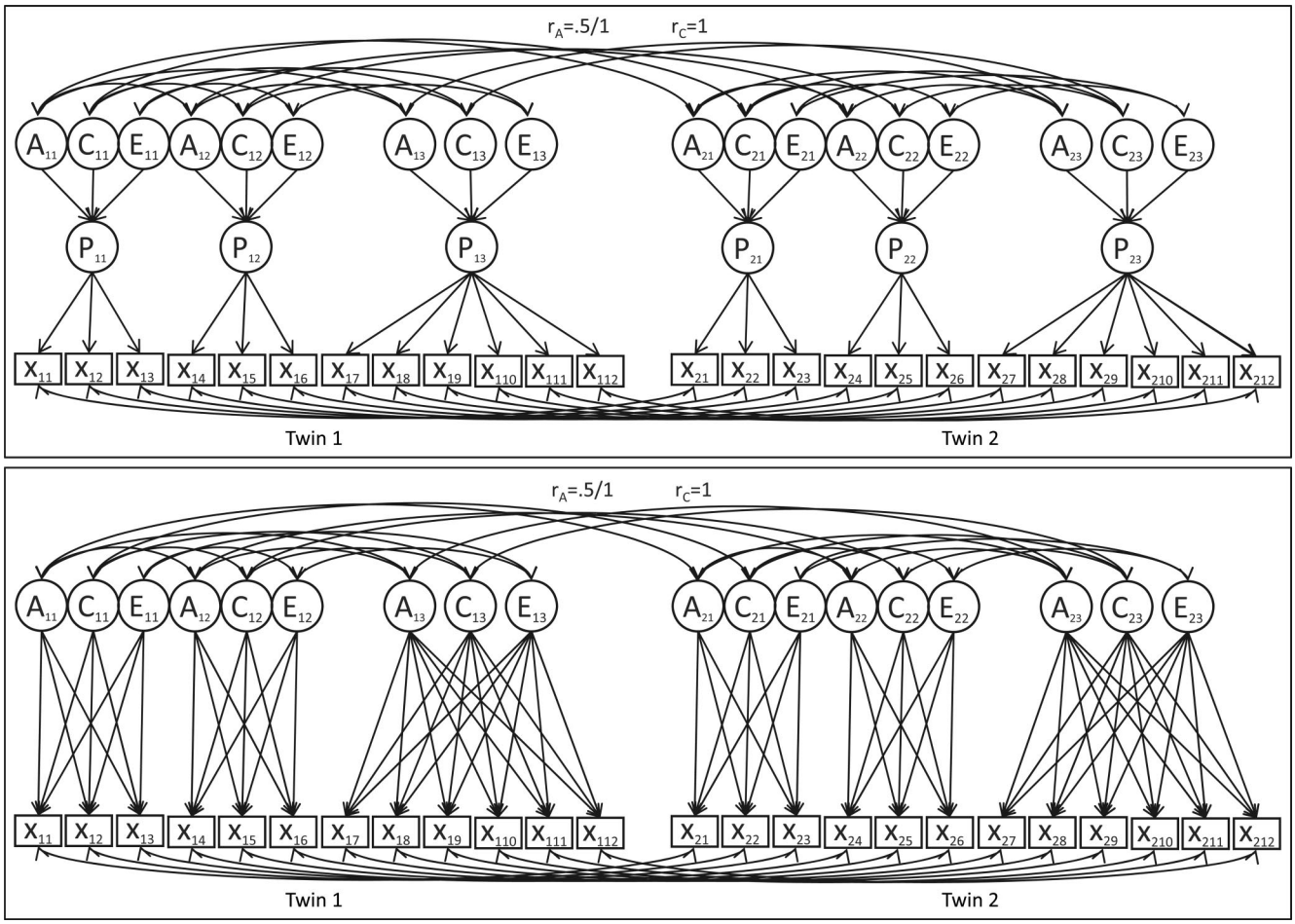


Figure 4. A common pathway (upper panel) and an independent pathway (lower panel) model based on the phenotypic exploratory factor analysis of Data Set 2. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor.

one considers the generating model (third panel in Figure 2), it is clear why this is the case: a model that assumes equal clustering of items with regard to A, C, and E effects (as does any model based on phenotypic factor analysis) cannot adequately describe the present data-generating mechanism. Although we detail only the results based on the three-factor EFA solution, none of the EFA solutions presented in Table 2 correctly convey the genetic and environmental effects on the

Table 5
Fit Statistics Obtained in Exploratory Factor Analysis of the Σ_A, Σ_C, and Σ_E Matrices in Data Sets 2 and 3

	Data Set 2				Data Set 3			
Factor	χ^2	*df*	*p*	RMSEA	χ^2	*df*	*p*	RMSEA
A								
1f	519.7	54	0	.0004	519.7	54	0	.0004
2f	0	43	1	0	0	43	1	0
C								
1f	0	54	1	0	1157.7	54	0	.0006
2f	0	43	1	0	495	43	0	.0005
3f	0	33	1	0	0	33	1	0
E								
1f	1302.9	54	0	.0007	1302.9	54	0	.0007
2f	0	43	1	0	0	43	1	0

Note. RMSEA = root-mean-square error of approximation.

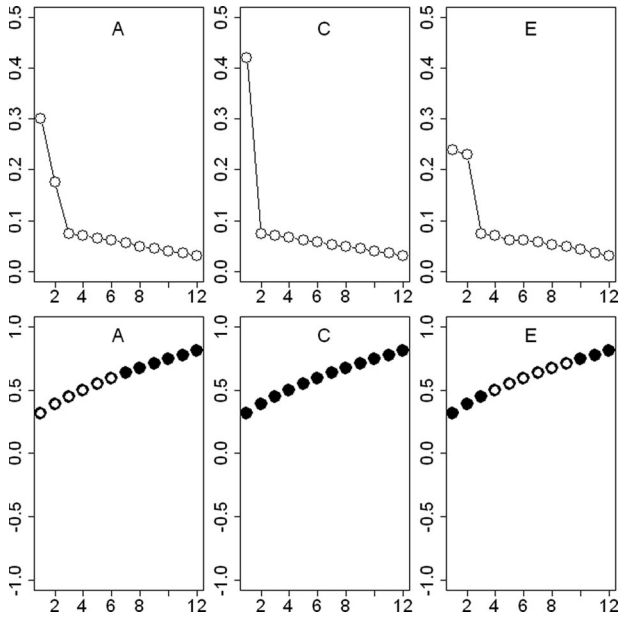


Figure 5. Data Set 2: Normalized eigenvalues for the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ matrices (upper panel) and factor loadings obtained in the exploratory factor analysis solutions with two A, one C, and two E factors (lower panel). Shading codes for different latent factors. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor.

items. It is interesting to note that the misspecification of the phenotypic models (in the sense that none accurately represented the data-generating structure) was not evident in the fit measures associated with the models; the fit measures associated with all but the one-factor EFA solution indicated an excellent fit.

The second part of the analyses was aimed at directly addressing the dimensionality issue, without reference to the phenotypic factor structure. To this end, the A, C, and E components of the observed covariance structure were estimated from the data, and the dimensionality of each of those components was separately evaluated using EFA. The A, C, and E covariance components (i.e., the 12 × 12 $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ matrices) may be estimated from twin data by fitting the model given in Equation 1. These analyses were carried out in Mx (Neale, 2000). Subsequently, each of the three covariance matrices was subjected to EFA. As we do not assume any phenotypic model and make no predictions about the dimensionalities of the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ covariance components, this approach is purely exploratory.

The results of the EFA are given in Table 5 and Figure 5. As apparent from both the table and the scree plots in the figure, the results correctly indicate the order of the $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$ matrices to be 2, 1, and 2, respectively. The estimated factor loadings of the corresponding EFA solutions with two A, one C, and two E factors, shown in the lower panel of Figure 5, correspond exactly to the parameters of the generating model.

Data Set 3. In Model 3, the A, C, and E structures differ appreciably from one another. The results of phenotypic EFA, shown in Tables 2–4, indicate that a model with five phenotypic factors provides an adequate description of the data. However, as evident from Table 4, the pattern of factor loadings in this model is inconsistent with a simple structure; thus, deciding on the number of actual latent dimensions underlying the data and the nature of the factors is complicated. Given that none of the EFA solutions in Table 2 can correctly convey the genetic and environmental effects on the items, we do not detail the possible confirmatory common and independent pathway models one may fit to the data given the EFA results. Instead, we present the solution obtained by the EFA of the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ variance components (see Figure 6 and Table 5). As evident from both Table 5 and Figure 6, a two-A, three-C, two-E structure is clearly supported by the EFA results, and both the factor loading structure and the values of the factor loading parameters are recovered correctly (lower panel of Figure 6).

Finally, we note that the chosen dimensionalities of the $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$ matrices represent only one instance of a violation of the common pathway model. In the present simulation, the values within the $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices are still consistent with a common pathway model (i.e., the nonzero elements of $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices satisfy the proportionality constraint $a_i/a_{i+1} = c_i/c_{i+1} = e_i/e_{i+1} = k$, where $i = 1, \ldots 11$ and k is a constant). In other words, the correlation between the nonzero values in the $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices is 1; that is, the factor loadings are collinear. It is possible to further violate the common pathway structure by manipulating the correlations between the values in the $\mathbf{\Lambda}_A$, $\mathbf{\Lambda}_C$, and $\mathbf{\Lambda}_E$ matrices. However, this violation is less detrimental to model fit than are the differences in dimensionalities of $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, $\mathbf{\Psi}_E$ matrices.

The present simulation shows that the clustering of the items with respect to genetic and environmental effects is required to

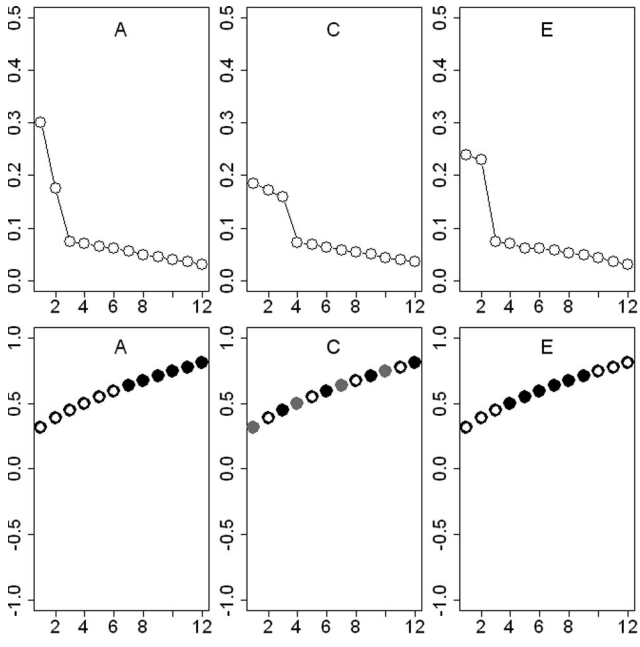


Figure 6. Data Set 3: Normalized eigenvalues for the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ matrices (upper panel) and factor loadings obtained in exploratory factor analysis solutions with two A, three C, and two E factors (lower panel). Shading codes for different latent factors. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor.

be identical for a unidimensional latent variable model to hold. This is in line with the theoretical derivation presented earlier in this article. In addition, it shows that if genetic and environmental effects do not cluster identically, psychometric analyses may fail to correctly indicate the dimensionality of the latent space. In these cases, the data either will show a significant degree of indeterminacy with respect to alternative dimensional hypotheses or will support an incorrect latent structure. However, attending to the genetic and environmental antecedents of the items successfully resolved the dimensionality issue. We now apply this methodology to an empirical data set.

Illustration: Childhood Internalizing Problems

Internalizing problems concern conditions such as anxiety, depression, and somatization. Dimensionality assessment has traditionally been difficult for such problems. For instance, current diagnostic systems like the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) distinguish anxiety and mood disorders as separate categories, but there is a significant amount of evidence to suggest that the overlap between such disorders is larger than can be reasonably expected were such a categorical distinction between types of disorders correct (e.g., Brady & Kendall, 1992). This is supported by genetic analyses, which univocally suggest that the genetic effects that impact anxiety and depression are shared, whereas the unique environmental effects are not (see, e.g., Kendler et al., 1987; Kendler, Neale, Kessler, Heath, & Eaves, 1992; Middeldorp, Cath, Van Dyck, & Boomsma, 2005). This presents an extraordinarily difficult task for the test constructor. For how should items that probe different anxiety and mood related problems be allocated to subscales? Can we reasonably expect a clear outcome of dimensionality assessment in this case? In the present example, we show that such an outcome is unrealistic given the genetic and environmental background of internalizing problems. In addition, we show how the use of genetic information uncovers a complex dimensional pattern that can be used to further the psychometric understanding of test scores.

Data

The data were obtained from the Netherlands Twin Register at VU University Amsterdam (Bartels, van Beijsterveldt, et al., 2007; Boomsma et al., 2006) and consist of maternal ratings of 11,565 twins (including 2,085 MZ and 3,599 DZ complete twin pairs) of mean age 10.1 years ($SD = 0.4$) on the Internalizing grouping of the Dutch version of the Child Behavior Checklist for Ages 4–18 (CBCL/4–18; Achenbach, 1991; Verhulst, Van der Ende, & Koot, 1996). The Internalizing grouping of the CBCL is a scale designed to measure disturbances in intropunitive emotions and moods in children, and consists of three subscales: Anxious/Depressed, Withdrawn, and Somatic Complaints, comprising 31 discrete items (listed in Appendix C) in total. Responses are given on a 3-point scale.[9]

Descriptive Statistics

The item distributions were positively skewed, with response rates ranging from 54.8% to 96.8% ($M = 84.8$, $SD = 10.3$) for the response 0 (*symptom not present*), 2.9% to 41.2% ($M = 13.2$, $SD = 9.4$) for the response 1 (*symptom somewhat/sometimes present*), and 0.2% to 6.4% ($M = 1.5$, $SD = 1.3$) for the response 2 (*symptom very/often present*). MZ and DZ twin item correlations and the distribution of inter-item correlations are depicted in Appendix C.

Analyses

As in the preceding example, the analyses consisted of two parts. In the first set of analyses, the phenotypic dimensionality of the data set was assessed using EFA, and the solutions obtained in EFA were tested in a confirmatory manner, by (a) specifying and fitting simple structure phenotypic models based on the EFA results[10] and (b) subsequently using these simple structure models as a basis for specifying genetic common and independent pathway models. In common pathway models, the variance in the phenotypic factors obtained in EFA was decomposed into A, C, and E components. The independent pathway models were based on the common pathway models, in the sense that they retain the structure employed in the common pathway models (i.e., they contain the same number of A, C, and E factors, affecting the same clusters of items) but dispose of the psychometric factors (i.e., allow for the items to load directly on the A, C, and E factors). Thus, the common pathway models represent a special case of (i.e., are nested under) the independent pathway models. By comparing the fit of these common and independent pathway models, we address the focal question of whether one can interpret the phenotypic common factors substantively and causally.

In the second set of analyses, independent pathway modeling was applied in an exploratory manner. In particular, the analyses consisted of estimating the unconstrained genetic and environmental covariance matrices (i.e., the 31×31 additive genetic, shared and unshared environmental covariance matrices $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$) and subjecting each of these covariance matrices to EFA to obtain an indication of their dimensionality (i.e., the order of the covariance matrices $\mathbf{\Psi}_A$, $\mathbf{\Psi}_C$, and $\mathbf{\Psi}_E$; Equation 3).

As in the simulation example, the analyses were performed using Mplus, Mx, and R.[11] Given the discrete nature of the items, we fitted discrete factor models (i.e., we assumed the discrete indicator variables to be a realization of a continuous normal latent process and fit models to polychoric correlations; Flora & Curran,

[9] Returning to the aforementioned assumptions of the twin design, in the present study we tested a number of these assumptions, including absence of rater bias and absence of recruitment bias. The issue of rater bias was addressed by comparing the standard deviations observed in our sample to those of normative samples (Verhulst et al., 1996). These were found to differ only slightly: The ratios of our standard deviations to those of normative samples are .91, .83, and .95, for the Anxious/Depressed, Withdrawn, and Somatic Complaints scales, respectively. The issue of rater bias in the present data has been addressed in the past. Bartels, Boomsma, Hudziak, van Beijsterveldt, and van den Oord (2007) reported that in a subset ($N = 7{,}718$) of the present sample, the estimate of the upper bound of the phenotypic variance that may contain rater bias is ~.14.

[10] EFA and CFA were performed using split-half validation. Cases were randomly assigned to either half of the sample; one half was subsequently used for EFA ($N = 5{,}782$), and the other for CFA ($N = 5{,}783$).

[11] The scripts used to perform the analyses may be obtained from the first author.

2004; Wirth & Edwards, 2007) using the robust weighted least squares estimator (Muthén & Muthén, 2007b). The polychoric correlations between the 31 items and between the 62 (31 per twin) items served as input in the phenotypic and the genetic factor analyses, respectively. In evaluating model fit, we used CFI, TLI, and RMSEA. As both our sample size and the models employed were large, the chi-square statistic was of limited use as an overall fit measure (Jöreskog, 1993), and was employed only to test local hypotheses concerning comparisons of nested models, as these comparisons are associated with a smaller approximation error.

Results

The initial analysis involved phenotypic EFA of the 31 items. The term *phenotypic* here indicates that only the observed (phenotypic) covariation is analyzed; that is, the analysis does not exploit the fact that the sample consists of familially related individuals.[12] EFA indicated two well-fitting models, a three- and a four-factor model (depicted in Figures 7A and 7B). Interestingly, in both of these models, the items originally belonging to the Anxious/Depressed scale cluster into those appearing to be more relevant to anxiety (3. Fears doing something bad, 4. Must be perfect, 8. Nervous, tense, 9. Fearful, anxious, 10. Feels too guilty, 11. Self-conscious, 14. Worries) and those more related to depression (1. Lonely, 2. Cries a lot, 5. Feels unloved, 6. Others out to get him, 7. Feels worthless, 12. Suspicious, 13. Sad). Note that the depiction in the figure is simplified insofar as only the path with the highest factor loading is shown for each item. The factor loadings associated with the paths omitted from the figure equal .05 on average; for comparison, the mean of the factor loadings for the depicted paths equals .58. Detailed results, including item content, factor loadings, factor correlations, and proportion of variance explained (R^2), are given in Appendix C.

Subsequently, based on the EFA results and the standard CBCL/4–18 model, a three- and a four-factor phenotypic model (Figures 7C and 7B, respectively) were specified and fitted to the data. As is evident from the figure, a firm distinction was hard to make between the fit of a model in which the anxiety and depression items represent a single dimension (Figure 7C) and a model in which they represent two distinct dimensions (Figure 7B). The additional solution provided by EFA (Figure 7A), in which items associated with anxiety load on the Withdrawn factor, obtained a similar fit. Whereas items pertaining to somatic complaints consistently form one dimension, the dimensionality of items pertaining to depression, anxiety, and withdrawn behavior therefore remains less clear. This is perhaps not surprising in the light of the well-established difficulty of distinguishing phenotypically the dimensions of anxiety and depression (see, e.g., Clark & Watson, 1991).

In the next step, the results of phenotypic analyses were used as a basis for specifying genetic factor models. In common pathway models, the factor structure of the models tested in the phenotypic CFA (Figures 7B and 7C) was retained, and the contributions of the A, C, and E factors to the phenotypic latent factors were investigated. The three- and four-factor common pathway models specified in this way differ only minimally in terms of model fit: The respective fit measures were $\chi^2(583) = 2030$, $p < .001$, CFI = .952, TLI = .966, RMSEA = .030, and $\chi^2(584) = 1811$, $p < .001$, CFI = .959, TLI = .971, RMSEA = .027. In independent pathway modeling, the structure employed in the three- and four-factor common pathway models was retained, but the psychometric factors are disposed of; that is, the items were allowed to load directly on the A, C, and E factors. Again, the three- and four-factor models differed only minimally in terms of model fit; the fit measures associated with the two models were $\chi^2(534) = 1142$, $p < .001$, CFI = .980, TLI = .984, RMSEA = .020, and $\chi^2(542) = 1161$, $p < .001$, CFI = .979, TLI = .984, RMSEA = .020, respectively.[13]

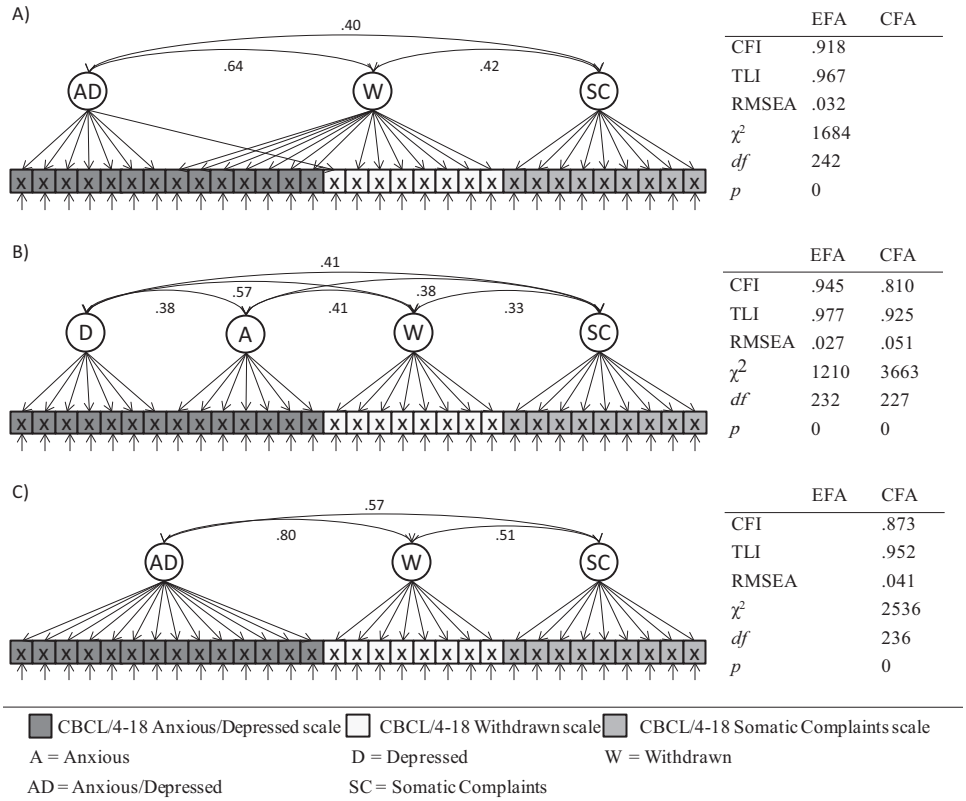
Returning to the focal question of whether independent pathway models fit the data appreciably better than the corresponding common pathway models, we compared the general fit of the models and carried out likelihood ratio tests of the proportionality constraints mentioned above. These tests revealed both the three- and four-factor-based independent pathway models to fit better than their common pathway counterparts (chi-square difference tests:[14] $\chi^2(25) = 1066$, $p < .001$, for the three-factor-based models and $\chi^2(23) = 864$, $p < .001$, for the four-factor-based models). This implies that the common pathway models, in which the latent variables mediate all the A, C and E effects on individual phenotypic differences, fail to convey accurately the genetic and environmental effects on the items. Again, we note that the misspecification of the common pathway models was not evident in the fit measures associated with the models. Both common pathway models obtained a good fit, and the same is true of the phenotypic models.

In the second set of analyses, we employed EFA to evaluate the dimensionalities of the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ covariance matrices. The results are shown in Figure 8. An inspection of scree plots indicates a one-dimensional C structure. The structures of A and E matrices remain, however, somewhat less clear. To explore the A and E structures further, we use the EFA results as a basis for specifying a number of competing independent pathway models with varying A, C, and E dimensionalities and fit these models to the phenotypic covariance matrix. An example of these confirmatory independent pathway models is depicted in Figure 9. A detailed overview of the fit measures and interfactor correlations associated with each of the models is given in Table C6. Overall, a comparison of these models indicated a model with two A, one C, and four E factors as the best fitting model, $\chi^2(531) = 1082$, $p < .001$, CFI = .982, TLI = .986, RMSEA = .019. This model is depicted in Figure 9. It should, however, be noted that most of the models tested did not differ considerably in terms of model fit; therefore the structure in Figure 9 need not necessarily be conclu-

[12] As treating observations from the same family as independent may result in biased estimates, we performed a correction for clustering available in Mplus, which has been shown to work well in this context (Rebollo, de Moor, Dolan, & Boomsma, 2006).

[13] Given that the fit of the three- and four-factor models is virtually indistinguishable, in practice one might simply accept the three-factor model on the basis of parsimony. However, given our interest in the specific reasons for the nearly identical fit, at this point we make no decisions on which model to accept and proceed with the analyses.

[14] For robust weighted least squares estimators the standard approach of taking the difference between chi-square values and the corresponding degrees of freedom is not appropriate because the chi-square difference is not chi-square distributed (Muthén & Muthén, 2007b). We therefore performed chi-square difference testing using scaling correction factors (Satorra & Bentler, 2001).



	EFA	CFA
CFI	.918	
TLI	.967	
RMSEA	.032	
χ^2	1684	
df	242	
p	0	

	EFA	CFA
CFI	.945	.810
TLI	.977	.925
RMSEA	.027	.051
χ^2	1210	3663
df	232	227
p	0	0

	EFA	CFA
CFI		.873
TLI		.952
RMSEA		.041
χ^2		2536
df		236
p		0

Figure 7. The three-factor model based on exploratory factor analysis (EFA; A), the four-factor model based on EFA (B), and the standard Child Behavior Checklist for Ages 4–18 (CBCL/4–18) three-factor model (C). Fit indices (on the right) obtained in EFA (geomin rotation) and confirmatory factor analysis (CFA). N = 5,782 for EFA, N = 5,783 for CFA. For EFA solutions, only the path with the highest factor loading is shown for each item. CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root-mean-square error of approximation.

sive. In addition, rejecting a common pathway model in favor of the corresponding independent model does not establish the structure employed in the independent model as in any way definitive, and there is, naturally, a possibility of other types of models (e.g., the mutualism model of van der Maas, et al., 2006, or the network model of Cramer et al., 2010) providing a better account of the data. The use of the independent pathway model, as presented in this article, is merely instrumental to testing the mediation of external effects on item covariation by a latent variable. Furthermore, the present results do strongly suggest a unidimensional C structure and multidimensional (but mutually differing) A and E structures.

In light of the present results, the results of the phenotypic analyses start to make more sense; the inability of phenotypic modeling to distinguish between several models appears to be due to the phenotypic structure being generated by three sources: a one-dimensional C, a two-dimensional A, and a four-dimensional E source.

Discussion

Even though the analysis and determination of dimensionality is of central importance in psychological science, currently available strategies for dimensionality assessment often leave the issue undecided. Building on ideas concerning genetic item analysis, as developed in quantitative genetics (Eaves, 1983; Heath, Jardine, Eaves, & Martin, 1989; Kendler et al., 1987; Neale, Lubke, Aggen, & Dolan, 2005; van den Berg, Glas, & Boomsma, 2007; Waller & Reise, 1992), the present article has outlined how genetic information may be brought to bear on the dimensionality assessment problem. In particular, the methodology outlined in this article may be used with genetically informative data to (a) put latent variable hypotheses to a stronger test than is possible in purely phenotypic analyses and (b) gain insight into why dimensionality issues may be difficult to settle.

The methodology proposed in this article may therefore not only improve dimensionality assessment, but may also suggest explanations of why specific dimensional hypotheses are violated. Although dimensionality assessment remains a difficult and to some extent subjective task, these methods therefore offer enhanced resolution relative to that possible in purely phenotypic analyses. Importantly, we do not claim that assessment of phenotypic dimensionality without incorporating genetic information cannot produce correct results, or that genetic analyses render standard methods obsolete. Rather, we think that genetic designs offer an

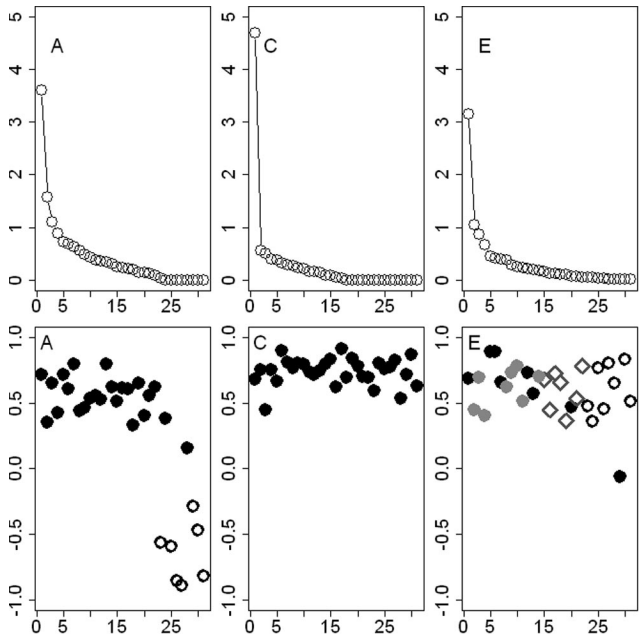


Figure 8. Child Behavior Checklist for Ages 4–18 data: Eigenvalues of the $\mathbf{\Sigma}_A$, $\mathbf{\Sigma}_C$, and $\mathbf{\Sigma}_E$ matrices (upper panel) and factor loadings obtained in exploratory factor analysis solutions with two A, one C, and four E factors (lower panel). Shading and shapes code for different latent factors. Only the highest factor loading for each item is shown.

underutilized and informative source of data that may help researchers to better understand the dimensionality of their constructs. Practically we envisage a situation in which phenotypic dimensionality research produces varied results, which will in practice simply result in disagreement concerning dimensionality. For instance, this is the case for cognitive abilities, with respect to which there are competing models that differ in dimensionality (notwithstanding many decades of research). One solution to this is to collect larger data sets. However, the present article suggests to researchers that the greater resolution provided by larger data sets may not provide the answer. We propose that it might be useful to seek out twin data in order to investigate possible differences in dimensionality of genetic and environment influences on major constructs.

As mentioned previously, the logic underlying our approach is essentially the same as that involved in measurement invariance research and MIMIC modeling. Moreover, common and independent pathway model comparisons have been considered outside the context of genetics (see, e.g., Carlson & Mulaik, 1993). However, what makes the analyses presented here different is that unlike in standard MIMIC modeling, the A, C, and E factors determine the variance of the latent variable completely. Furthermore, the situation in which the common pathway model is rejected is at least as informative as that in which it is retained, as the information contained in genetically informative data sets allows one to examine the exact nature of violations of dimensional assumptions; something that is typically not the case in standard MIMIC modeling. In the twin model, one can establish whether or not the common pathway model fits and, in case of misfit, can arrive at a detailed account of the cause of misfit, thereby moving the question of dimensionality from the phenotypic level to the genetic level and the environmental level. This increased resolution (i.e., the possibility to view the lack of unidimensionality of the observed covariation structure as a function of the dimensionalities of its underlying genetic and environmental structures) is unique to the twin design and is, in our opinion, a particularly powerful aspect of the present method.

In our illustrative analyses, the incorporation of genetic information turned out to be highly informative. In standard phenotypic analyses, it proved difficult to decide whether a three- or four-dimensional latent structure underlies the data—a situation that is not uncommon in psychometric investigations into dimensionality, where one often has to decide between solutions that differ substantively but appear to be nearly equivalent statistically. Incorporating genetic information, however, suggested that the reason for the ambiguity in the data with respect to these structures is that *several* models are correct, but apply to different sources of item covariation: A two-factor model seems to better reflect additive genetic influences, whereas a four-factor model better reflects unique environmental influences. Interestingly, common environmental influences appear to influence item scores across the board, suggesting that the common part of environmental variation varies along a single dimension.[15]

The question of how many dimensions are measured by the Internalizing scales of the CBCL can now be viewed from a new perspective, which may be surprising to the psychometrician: In terms of genetic variance, the items appear to measure two dimensions, corresponding to genetic influences distinctly affecting symptoms of depression, anxiety, and withdrawal, on the one hand, and somatic complaints, on the other. This implies, for instance, that genes act in a nonspecific way to influence the chance of developing depression-, anxiety-, and withdrawal-related symptomatology. Individual-specific environmental influences distinctly affect symptoms of depression, anxiety, withdrawal, and somatic complaints (thus, individual-specific environmental events may be, for example, specifically depressogenic or specifically anxiogenic), whereas environmental events shared by family members appear to have either a positive or a negative effect on the entire range of symptoms. In terms of, for instance, the anxiety–depression distinction, the present results suggest that these two syndromes share the same genetic basis but are distinctly affected by individual-specific environmental events—a finding that is in line with prior genetic investigations into the dimensionality of anxiety and depression (e.g., Kendler et al., 1987; 1992; Middeldorp et al., 2005).

It should be noted that the current results do not necessarily reflect upon the utility of the CBCL in the clinical context; we do not doubt its usefulness for diagnostic purposes, especially given that the broad structure found in our analyses is in line with the current item allocation of the CBCL. However, in the context of research one should bear in mind that the current scales may not measure three distinct sets of genetic, common environmental, and individual-specific environmental influences but possibly reflect a

[15] It is possible that a unidimensional C component partly stems from method variance. For instance, variance due to rater bias, if not explicitly modeled, is absorbed by C (Neale & Cardon, 1992). Given data by multiple raters, it is possible to test for presence of rater bias.

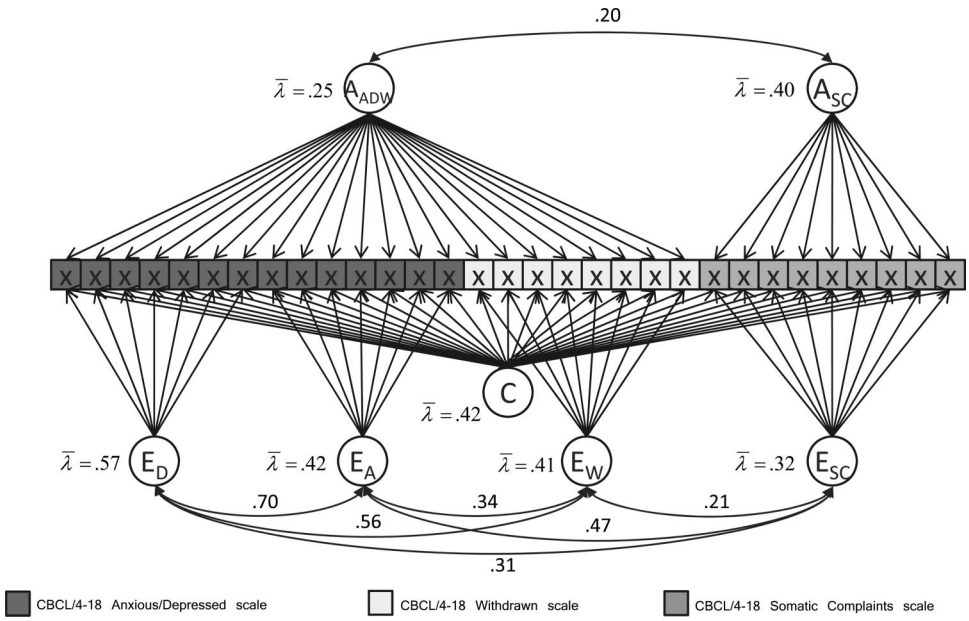


Figure 9. The 2A 1C 4E independent pathway model for the Child Behavior Checklist for Ages 4–18 (CBCL/4–18) Internalizing scale. Item residuals are not depicted but are estimated in the model. $\bar{\lambda}$ denotes the average factor loading associated with a factor. A = Anxious; D = Depressed; W = Withdrawn; SC = Somatic Complaints.

more complex underlying structure. Depending on the specific research goals, the results of this type of analysis may provide a basis for redefining the current scales to arrive at distinct measures of each of these sources of influences (e.g., if one's aim is to measure common environmental influences, one may view the item set as unidimensional and accordingly derive a single sum score from the data).

Naturally, the results of this type of analyses are relevant not only to theories of psychopathology; we consider their implications to be much wider. For instance, theories in developmental psychology may benefit from investigating the individual differences in the development of behavior as a function of genetic and environmental influences, or examining how the various dimensions of environmental and genetic influences change and develop over time. Also, the results might have implications for genetics itself. Specifically, the search for genes affecting specific behaviors is often based on a composite measure of a phenotype, such as a sum score. However, if these phenotypes are heterogeneous, analogous to the way the CBCL appears heterogeneous, using a total score as a basis for gene search would appear suboptimal, as the total score itself might not accurately reflect the genetic structure underlying the data (van der Sluis, Verhage, Posthuma, & Dolan, 2010). We consider this issue to be important, because power to detect the effects of measured genes is likely to suffer if the phenotypic measure is not correctly defined (van der Sluis, et al., 2010). Independent pathway item-level analysis, as described in this article, offers possibilities for redefining the phenotypic scores in terms of genetic and environmental effects. This may in turn allow for using latent trait estimates derived from a model such as that in Figure 7 as a basis for gene search.

In addition to these practical benefits of the present methodology, there are important conceptual considerations that follow from the ideas presented in this article. For instance, latent variable models like the factor model can be viewed as incorporating hypotheses concerning a common-cause structure that underlies item covariation (Borsboom, 2008; Borsboom et al., 2003; Haig, 2005a, 2005b). However, the question of whether latent variables hypothesized in a given context may be said to exist and have causal relevance is a point of dispute in many fields; one need only consider the fields of intelligence and personality research, where considerable controversy exists regarding the theoretical status of variables such as the *g* factor and the five factors of personality. To the extent that such models survive the confrontation with genetic information, as described here, they may be considered more strongly corroborated than they could be in analyses of purely phenotypic data. However, if models for genetic and environmental effects have different structures, as was the case for the illustration data in this article, the common factors found in our phenotypic analysis may in fact be an amalgam of several different genetic and environmental models. Clearly, in this case, the ascription of causal force to such amalgams is problematic.

In conclusion, we expect that the methodology proposed in this article may bear considerable fruit in disentangling dimensionality issues in the research areas where they have generated controversy, and shed light on the theoretical status of important hypothesized latent variables in intelligence, psychopathology, and personality research. The time is ripe for investigations along these lines. In the past decades, behavior genetics researchers have constructed large and well-archived twin and family registries that are perfectly suited for analyses such as

those reported here (e.g., a 1998 review lists 16 twin registries in Europe alone; Boomsma, 1998; Busjahn, 2002). The data sets contained in those registries are typically obtainable via protocols for collaborative projects, and in some cases even publically available (e.g., Add Health; see Harris, Halpern, Smolen, & Haberstick, 2006). In addition, the development of psychometric software as well as the current speed of computers has led to a situation where the required statistical analyses have become feasible. In our view, this opens up a wealth of possibilities for refining and extending psychometric investigations beyond the analysis of purely phenotypic covariation.

References

Achenbach, T. M. (1966). Classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs: General and Applied, 80*(7, Whole No. 615). doi:10.1037/h0093906

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 profile*. Burlington: University of Vermont, Department of Psychiatry.

Ackerman, P. L., Beier, M. E., & Boyle, M. O. (2005). Working memory and intelligence: The same or different constructs? *Psychological Bulletin, 131*(1), 30–60. doi:10.1037/0033-2909.131.1.30

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40*(1), 57–87. doi:10.1111/1469-7610.00424

Bartels, M., Boomsma, D. I., Hudziak, J. J., van Beijsterveldt, T. C. E. M., & van den Oord, E. J. C. G. (2007). Twins and the study of rater (dis)agreement. *Psychological Methods, 12*(4), 451466. doi:10.1037/1082-989X.12.4.451

Bartels, M., van Beijsterveldt, C. E. M., Derks, E. M., Stroet, T. M., Polderman, T. J. C., Hudziak, J. J.,& Boomsma, D. I. (2007). Young Netherlands Twin Register (Y-NTR): A longitudinal multiple informant study of problem behavior. *Twin Research and Human Genetics, 10*(1), 3–11. doi:10.1375/twin.10.1.3

Bollen, K. A. (1989). *Structural equations with latent variables*. New York, NY: Wiley.

Boomsma, D. I. (1998). Twin registers in Europe: An overview. *Twin Research, 1*(1), 34–51.

Boomsma, D., Busjahn, A., & Peltonen, L. (2002). Classical twin studies and beyond. *Nature Reviews Genetics, 3*(11), 872–882. doi:10.1038/nrg932

Boomsma, D. I., de Geus, E. J. C., Vink, J. M., Stubbe, J. H., Distel, M. A., Hottenga, J.-J., . . . Willemsen, G. (2006). Netherlands Twin Register: From twins to twin families. *Twin Research and Human Genetics, 9*(6), 849–857. doi:10.1375/twin.9.6.849

Boomsma, D. I., & Molenaar, P. C. M. (1986). Using LISREL to analyze genetic and environmental covariance structure. *Behavior Genetics, 16*(2), 237–250. doi:10.1007/BF01070799

Boomsma, D. I., & Molenaar, P. C. M. (1987). The genetic-analysis of repeated measures. 1. Simplex models. *Behavior Genetics, 17*(2), 111–123. doi:10.1007/BF01065991

Borsboom, D. (2008). Psychometric perspectives on diagnostic systems. *Journal of Clinical Psychology, 64*(9), 1089–1108. doi:10.1002/jclp.20503

Borsboom, D., Mellenbergh, G. J., & van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review, 110*(2), 203–219. doi:10.1037/0033-295X.110.2.203

Brady, E. U., & Kendall, P. C. (1992). Comorbidity of anxiety and depression in children and adolescents. *Psychological Bulletin, 111*(2), 244–255. doi:10.1037/0033-2909.111.2.244

Busjahn, A. (Ed.). (2002). Twin registers as a global resource for genetic research [Special Issue]. *Twin Research, 5*(5).

Carlson, M., & Mulaik, S. A. (1993). Trait ratings from descriptions of behavior as mediated by components of meaning. *Multivariate Behavioral Research, 28*(1), 111–159. doi:10.1207/s15327906mbr2801_7

Carroll, J. B. (2003). The higher-stratum structure of cognitive abilities: Current evidence supports *g* and about ten broad factors. In H. Nyborg (Ed.), *The scientific study of general intelligence: Tribute to Arthur R. Jensen* (pp. 5–21). New York, NY: Pergamon. doi:10.1016/B978-008043793-4/50036-2

Cattell, R. B. (1941). *Some theoretical issues in adult intelligence testing* [Abstract of poster presented at the annual meeting of the American Psychological Association]. *Psychological Bulletin, 38*(7), 592.

Cherny, S. S. (2008). Variance components and related methods for mapping quantitative trait loci. *Sociological Methods & Research, 37*(2), 227–250. doi:10.1177/0049124108324524

Clark, L. A. (1989). The anxiety and depressive disorders: Descriptive psychopathology and differential diagnosis. In P. C. Kendall & D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features* (pp. 83–129). New York, NY: Academic Press.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316–336. doi:10.1037/0021-843X.100.3.316

Costa, P. T., Jr., & McCrae, R. R. (1985). *The NEO Personality Inventory manual*. Odessa, FL: Psychological Assessment Resources.

Cramer, A. O. J., Waldorp, L. J., van der Maas, H. L. J., & Borsboom, D. (2010). Comorbidity: A network perspective. *Behavioral and Brain Sciences, 33*(2–3), 137–150. doi:10.1017/S0140525X09991567

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin, 52*(4), 281–302. doi:10.1037/h0040957

Derks, E. M., Dolan, C. V., & Boomsma, D. I. (2006). A test of the equal environment assumption (EEA) in multivariate twin studies. *Twin Research and Human Genetics, 9*(3), 403–411. doi:10.1375/twin.9.3.403

Dolan, C. V. (1992). *Biometric decomposition of phenotypic means in human samples* (Unpublished doctoral dissertation). University of Amsterdam, Amsterdam, the Netherlands.

Eaves, L. J. (1983). Errors of inference in the detection of major gene effects on psychological test scores. *American Journal of Human Genetics, 35*(6), 1179–1189.

Eaves, L. J., Long, J., & Heath, A. C. (1986). A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behavior Genetics, 16*(1), 143–162. doi:10.1007/BF01065484

Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods, 4*(3), 272–299. doi:10.1037/1082-989X.4.3.272

Falconer, D. S., & Mackay, T. F. C. (1996). *Introduction to quantitative genetics* (4th ed.). Harlow, England: Longmans Green.

Flora, D. B., & Curran, P. J. (2004). An empirical evaluation of alternative methods of estimation for confirmatory factor analysis with ordinal data. *Psychological Methods, 9*(4), 466–491. doi:10.1037/1082-989X.9.4.466

Franić, S., Dolan, C. V., Borsboom, D., & Boomsma, D. I. (2012). Structural equation modeling in genetics. In R. H. Hoyle (Ed.), *Handbook of structural equation modeling* (pp. 617–635). New York, NY: Guilford Press.

Goldberg, L. R. (1990). An alternative "description of personality": The Big-Five factor structure. *Journal of Personality and Social Psychology, 59*(6), 1216–1229. doi:10.1037/0022-3514.59.6.1216

Gorsuch, R. L. (1983). *Factor analysis* (2nd ed.). Hillsdale, NJ: Erlbaum.

Haig, B. D. (2005a). An abductive theory of scientific method. *Psychological Methods, 10*(4), 371–388. doi:10.1037/1082-989X.10.4.371

Haig, B. D. (2005b). Exploratory factor analysis, theory generation, and scientific method. *Multivariate Behavioral Research, 40*(3), 303–329. doi:10.1207/s15327906mbr4003_2

Harris, K. M., Halpern, C. T., Smolen, A., & Haberstick, B. C. (2006). The National Longitudinal Study of Adolescent Health (Add Health) twin data. *Twin Research and Human Genetics, 9*(6), 988–997. doi:10.1375/twin.9.6.988

Heath, A. C., Jardine, R., Eaves, L. J., & Martin, N. G. (1989). The genetic structure of personality—II. Genetic item analysis of the EPQ. *Personality and Individual Differences, 10*(6), 615–624. doi:10.1016/0191-8869(89)90221-3

Henson, R. K., & Roberts, J. K. (2006). Use of exploratory factor analysis in published research: Common errors and some comment on improved practice. *Educational and Psychological Measurement, 66*(3), 393–416. doi:10.1177/0013164405282485

Horn, J. L. (1965). *Fluid and crystallized intelligence: A factor analytic and developmental study of the structure among primary mental abilities* (Unpublished doctoral dissertation). University of Illinois, Champaign.

Jöreskog, K. G. (1993). Testing structural equation models. In K. A. Bollen & S. J. Long (Eds.), *Testing structural equation models* (pp. 294–316). Newbury Park, CA: Sage.

Jöreskog, K., & Goldberger, S. (1975). Estimation of a model with multiple indicators and multiple causes of a single latent variable. *Journal of American Statistical Association, 70,* 631–639.

Kendler, K. S., Heath, A. C., Martin, N. G., & Eaves, L. J. (1987). Symptoms of anxiety and symptoms of depression: Same genes, different environments. *Archives of General Psychiatry, 44*(5), 451–457. doi:10.1001/archpsyc.1987.01800170073010

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Major depression and generalized anxiety disorder: Same genes, (partly) different environments. *Archives of General Psychiatry, 49*(9), 716–722. doi:10.1001/archpsyc.1992.01820090044008

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York, NY: Guilford Press.

Lawley, D. N., & Maxwell, A. E. (1971). *Factor analysis as a statistical method* (2nd ed.). London, England: Butterworths.

Lilienfeld, S. O., Waldman, I. D., & Israel, A. C. (1994). A critical examination of the use of the term and concept of *comorbidity* in psychopathology research. *Clinical Psychology: Science and Practice, 1*(1), 71–83. doi:10.1111/j.1468-2850.1994.tb00007.x

Lykken, D. T., McGue, M., Bouchard, T. J., & Tellegen, A. (1990). Does contact lead to similarity or similarity to contact. *Behavior Genetics, 20*(5), 547–561. doi:10.1007/BF01065871

Lykken, D. T., McGue, M., & Tellegen, A. (1987). Recruitment bias in twin research: The rule of two-thirds reconsidered. *Behavior Genetics, 17*(4), 343–362. doi:10.1007/BF01068136

Martin, N., Boomsma, D., & Machin, G. (1997). A twin-pronged attack on complex traits. *Nature Genetics, 17*(4), 387–392. doi:10.1038/ng1297-387

Martin, N. G., & Eaves, L. J. (1977). Genetic analysis of covariance structure. *Heredity, 38*(1), 79–95. doi:10.1038/hdy.1977.9

Martin, N. G., & Wilson, S. R. (1982). Bias in the estimation of heritability from truncated samples of twins. *Behavior Genetics, 12*(4), 467–472. doi:10.1007/BF01065638

Matzke, D., Dolan, C. V., & Molenaar, D. (2010). The issue of power in the identification of "g" with lower-order factors. *Intelligence, 38*(3), 336–344. doi:10.1016/j.intell.2010.02.001

McArdle, J. J., & Goldsmith, H. H. (1990). Alternative common factor models for multivariate biometric analyses. *Behavior Genetics, 20*(5), 569–608. doi:10.1007/BF01065873

McCrae, R. R., & Costa, P. T., Jr. (1999). A five-factor theory of personality. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 139–153). New York, NY: Guilford Press.

McCrae, R. R., & John, O. P. (1992). An introduction to the five-factor model and its applications. *Journal of Personality, 60*(2), 175–215. doi:10.1111/j.1467-6494.1992.tb00970.x

Medland, S. E., & Neale, M. C. (2010). An integrated phenomic approach to multivariate allelic association. *European Journal of Human Genetics, 18*(2), 233–239. doi:10.1038/ejhg.2009.133

Mellenbergh, G. J. (1989). Item bias and item response theory. *International Journal of Educational Research, 13*(2), 127–143. doi:10.1016/0883-0355(89)90002-5

Meredith, W. (1993). Measurement invariance, factor analysis and factorial invariance. *Psychometrika, 58*(4), 525–543. doi:10.1007/BF02294825

Middeldorp, C. M., Cath, D. C., Van Dyck, R., & Boomsma, D. I. (2005). The co-morbidity of anxiety and depression in the perspective of genetic epidemiology. A review of twin and family studies. *Psychological Medicine, 35*(5), 611–624. doi:10.1017/S003329170400412X

Mulaik, S. A. (1987). A brief history of the philosophical foundations of exploratory factor analysis. *Multivariate Behavioral Research, 22*(3), 267–305. doi:10.1207/s15327906mbr2203_3

Muthén, L. K., & Muthén, B. O. (2007a). *Mplus* (Version 5.1) [Computer software]. Los Angeles, CA: Muthén & Muthén.

Muthén, L. K., & Muthén, B. O. (2007b). *Mplus user's guide* (5th ed.). Los Angeles, CA Muthén & Muthén.

Neale, M. C. (2000). *MxGui* (Version 1.7.03) [Computer software]. Richmond: Virginia Commonwealth University. Retrieved from http://www.vcu.edu/mx/

Neale, M. C., & Cardon, L. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, the Netherlands: Kluwer Academic. doi:10.1007/978-94-015-8018-2

Neale, M. C., Eaves, L. J., Kendler, K. S., & Hewitt, J. K. (1989). Bias in correlations from selected samples of relatives: The effects of soft selection. *Behavior Genetics, 19*(2), 163–169. doi:10.1007/BF01065901

Neale, M. C., Lubke, G., Aggen, S. H., & Dolan, C. V. (2005). Problems with using sum scores for estimating variance components: Contamination and measurement noninvariance. *Twin Research and Human Genetics, 8*(6), 553–568. doi:10.1375/twin.8.6.553

Pearl, J. (2000). *Causality: Models, reasoning, and inference*. New York, NY: Oxford University Press.

Plomin, R., Defries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (5th ed.). New York, NY: Freeman.

Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research, 5*(6), 554–571.

R Development Core Team. (2009). R: A language and environment for statistical computing (Version 2.15.2) [Computer software]. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://cran.r-project.org/bin/windows/base/

Rebollo, I., de Moor, M. H. M., Dolan, C. V., & Boomsma, D. I. (2006). Phenotypic factor analysis of family data: Correction of the bias due to dependency. *Twin Research and Human Genetics, 9*(3), 367–376. doi:10.1375/twin.9.3.367

Rummel, R. J. (1970). *Applied factor analysis*. Evanston, IL: Northwestern University Press.

Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika, 66*(4), 507–514. doi:10.1007/BF02296192

Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychological Bulletin, 125*(4), 392–409. doi:10.1037/0033-2909.125.4.392

van den Berg, S. M., Glas, C. A. W., & Boomsma, D. I. (2007). Variance decomposition using an IRT measurement model. *Behavior Genetics, 37*(4), 604–616. doi:10.1007/s10519-007-9156-1

van der Maas, H. L. J., Dolan, C. V., Grasman, R. P. P. P., Wicherts, J. M., Huizenga, H. M., & Raijmakers, M. E. J. (2006). A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychological Review, 113*(4), 842–861. doi:10.1037/0033-295X.113.4.842

van der Sluis, S., Dolan, C. V., Neale, M. C., & Posthuma, D. (2008). Power calculations using exact data simulation: A useful tool for genetic study designs. *Behavior Genetics, 38*(2), 202–211. doi:10.1007/s10519-007-9184-x

van der Sluis, S., Verhage, M., Posthuma, D., & Dolan, C. V. (2010). Phenotypic complexity, measurement bias, and poor phenotypic resolution contribute to the missing heritability problem in genetic association studies. *PLoS ONE, 5*(11), e13929. doi:10.1371/journal.pone.0013929

van Dongen, J., Draisma, H. H. M., Martin, N. G., & Boomsma, D. I. (2012). The continuing value of twin studies in the omics era. *Nature Reviews Genetics, 13*(9), 640–653. doi:10.1038/nrg3243

Verhulst, F. C., Van der Ende, J., & Koot, H. M. (1996). *Handleiding voor de CBCL/4–18* (CBCL/4–18 manual]. Rotterdam, the Netherlands: Erasmus University, Academic Medical Centre Rotterdam/ Sophia Children's Hospital, Department of Child Psychiatry.

Waller, N. G., & Reise, S. P. (1992). Genetic and environmental influences on item response pattern scalability. *Behavior Genetics, 22*(2), 135–152. doi:10.1007/BF01066993

Wirth, R. J., & Edwards, M. C. (2007). Item factor analysis: Current approaches and future directions. *Psychological Methods, 12*(1), 58–79. doi:10.1037/1082-989X.12.1.58

Yung, Y. F., Thissen, D., & McLeod, L. D. (1999). On the relationship between the higher-order factor model and the hierarchical factor model. *Psychometrika, 64*(2), 113–128. doi:10.1007/BF02294531

Zwick, W. R., & Velicer, W. F. (1982). Factors influencing four rules for determining the number of components to retain. *Multivariate Behavioral Research, 17*(2), 253–269. doi:10.1207/s15327906mbr1702_5

Zwick, W. R., & Velicer, W. F. (1986). Comparison of five rules for determining the number of components to retain. *Psychological Bulletin, 99*(3), 432–442. doi:10.1037/0033-2909.99.3.432

Appendix A

Model for the Residuals in the Simulation Study

The residuals of items in the model may also be subjected to a decomposition into genetic and environmental components. The model for the residuals was not detailed in the main text, where it was merely stated that the parameter values for the residual matrices were obtained by subtraction, for example, $\boldsymbol{\Theta}_{\mathbf{cp}} = \boldsymbol{\Theta}_{\mathrm{ip}} = \mathbf{I} - \mathrm{diag}[\boldsymbol{\Lambda}(\boldsymbol{\Phi}_{\mathrm{A}} + \boldsymbol{\Phi}_{\mathrm{C}} + \boldsymbol{\Phi}_{\mathrm{E}})\boldsymbol{\Lambda}^{t}]$. However, the residuals in the current simulations conform to an ACE model, and could alternatively be simulated by specifying the expected residual covariance matrix as a function of residual ACE parameters:

$$\begin{pmatrix} \boldsymbol{\Theta}_{11} & \boldsymbol{\Theta}_{12} \\ \boldsymbol{\Theta}_{21} & \boldsymbol{\Theta}_{22} \end{pmatrix} = \begin{pmatrix} \boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}^{t} + \boldsymbol{\Delta}_{\mathrm{C}}\boldsymbol{\Pi}_{\mathrm{C}}\boldsymbol{\Delta}_{\mathrm{C}}^{t} + \boldsymbol{\Delta}_{\mathrm{E}}\boldsymbol{\Pi}_{\mathrm{E}}\boldsymbol{\Delta}_{\mathrm{E}}^{t} & r_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}^{t} + \boldsymbol{\Delta}_{\mathrm{C}}\boldsymbol{\Pi}_{\mathrm{C}}\boldsymbol{\Delta}_{\mathrm{C}}^{t} \\ r_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}^{t} + \boldsymbol{\Delta}_{\mathrm{C}}\boldsymbol{\Pi}_{\mathrm{C}}\boldsymbol{\Delta}_{\mathrm{C}}^{t} & \boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}^{t} + \boldsymbol{\Delta}_{\mathrm{C}}\boldsymbol{\Pi}_{\mathrm{C}}\boldsymbol{\Delta}_{\mathrm{C}}^{t} + \boldsymbol{\Delta}_{\mathrm{E}}\boldsymbol{\Pi}_{\mathrm{E}}\boldsymbol{\Delta}_{\mathrm{E}}^{t} \end{pmatrix},$$

where $\boldsymbol{\Theta}_{11}(\boldsymbol{\Theta}_{22})$ is the 12 × 12 matrix of residual item variances of Twin 1 (Twin 2); $\boldsymbol{\Theta}_{12}(\boldsymbol{\Theta}_{21})$ is the 12 × 12 Twin 1 – Twin 2 residual covariance matrix; $\boldsymbol{\Delta}_{\mathrm{A}}$, $\boldsymbol{\Delta}_{\mathrm{C}}$, and $\boldsymbol{\Delta}_{\mathrm{E}}$ are the diagonal matrices containing the factor loadings of the items on the residual (item-specific) A, C, and E factors; $\boldsymbol{\Pi}_{\mathrm{A}}$, $\boldsymbol{\Pi}_{\mathrm{C}}$, and $\boldsymbol{\Pi}_{\mathrm{E}}$ are the covariance matrices of the residual A, C, and E factors; and r_{A} is the additive genetic twin correlation (1 for monozygotic [MZ] twins, .5 for dizygotic [DZ] twins). Note that because the residual covariance matrices were simulated to be equal across all the models above (e.g., $\boldsymbol{\Theta}_{\mathrm{ip}} = \boldsymbol{\Theta}_{\mathrm{cp}}$, $\boldsymbol{\Theta}_{\mathrm{ip21mz}} = \boldsymbol{\Theta}_{\mathrm{cp21mz}}$, and $\boldsymbol{\Theta}_{\mathrm{ip21dz}} = \boldsymbol{\Theta}_{\mathrm{cp21dz}}$), in the expression above we dispose of the "cp" and "ip" notation. Figure A1 depicts the ACE model for the residuals.

The residual parameter values were equal across the four models:

$$\Pi_{\mathrm{A}} = \boldsymbol{\Pi}_{\mathrm{C}} = \boldsymbol{\Pi}_{\mathrm{E}} = \mathrm{diag}(1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1),$$

$$\boldsymbol{\Delta}_{\mathrm{A}} = \mathrm{diag}(\sqrt{.450}, \sqrt{.425}, \sqrt{.400}, \sqrt{.375}, \sqrt{.350}, \sqrt{.325}, \sqrt{.300}, \sqrt{.275}, \sqrt{.250}, \sqrt{.225}, \sqrt{.200}, \sqrt{.175}),$$

$$\boldsymbol{\Delta}_{\mathrm{C}} = \mathrm{diag}(\sqrt{.270}, \sqrt{.255}, \sqrt{.240}, \sqrt{.225}, \sqrt{.210}, \sqrt{.195}, \sqrt{.180}, \sqrt{.165}, \sqrt{.150}, \sqrt{.135}, \sqrt{.120}, \sqrt{.105}),$$

$$\boldsymbol{\Delta}_{\mathrm{E}} = \mathrm{diag}(\sqrt{.18}, \sqrt{.17}, \sqrt{.16}, \sqrt{.15}, \sqrt{.14}, \sqrt{.13}, \sqrt{.12}, \sqrt{.11}, \sqrt{.10}, \sqrt{.09}, \sqrt{.08}, \sqrt{.07}).$$

Coming back to Expression 1 in the main text:

$$\begin{pmatrix} \boldsymbol{\Sigma}_{11} & \boldsymbol{\Sigma}_{12} \\ \boldsymbol{\Sigma}_{21} & \boldsymbol{\Sigma}_{22} \end{pmatrix} = \begin{pmatrix} \boldsymbol{\Sigma}_{\mathrm{A}} + \boldsymbol{\Sigma}_{\mathrm{C}} + \boldsymbol{\Sigma}_{\mathrm{E}} & r_{\mathrm{A}}\boldsymbol{\Sigma}_{\mathrm{A}} + \boldsymbol{\Sigma}_{\mathrm{C}} \\ r_{\mathrm{A}}\boldsymbol{\Sigma}_{\mathrm{A}} + \boldsymbol{\Sigma}_{\mathrm{C}} & \boldsymbol{\Sigma}_{\mathrm{A}} + \boldsymbol{\Sigma}_{\mathrm{C}} + \boldsymbol{\Sigma}_{\mathrm{E}} \end{pmatrix},$$

we may now note that $\boldsymbol{\Sigma}_{\mathrm{A}}$ is a function of both common and residual additive genetic influences, that is, $\boldsymbol{\Sigma}_{\mathrm{A}} = \boldsymbol{\Lambda}\boldsymbol{\Phi}_{\mathrm{A}}\boldsymbol{\Lambda}^{t} + \boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}{}^{t}$ (or $\boldsymbol{\Sigma}_{\mathrm{A}} = \boldsymbol{\Lambda}_{\mathrm{A}}\boldsymbol{\Psi}_{\mathrm{A}}\boldsymbol{\Lambda}_{\mathrm{A}}{}^{t} + \boldsymbol{\Delta}_{\mathrm{A}}\boldsymbol{\Pi}_{\mathrm{A}}\boldsymbol{\Delta}_{\mathrm{A}}{}^{t}$ in an independent pathway model). Similarly, $\boldsymbol{\Sigma}_{\mathrm{C}} = \boldsymbol{\Lambda}\boldsymbol{\Phi}_{\mathrm{C}}\boldsymbol{\Lambda}^{t} + \boldsymbol{\Delta}_{\mathrm{C}}\boldsymbol{\Pi}_{\mathrm{C}}\boldsymbol{\Delta}_{\mathrm{C}}{}^{t}$, etc.

(*Appendices continue*)

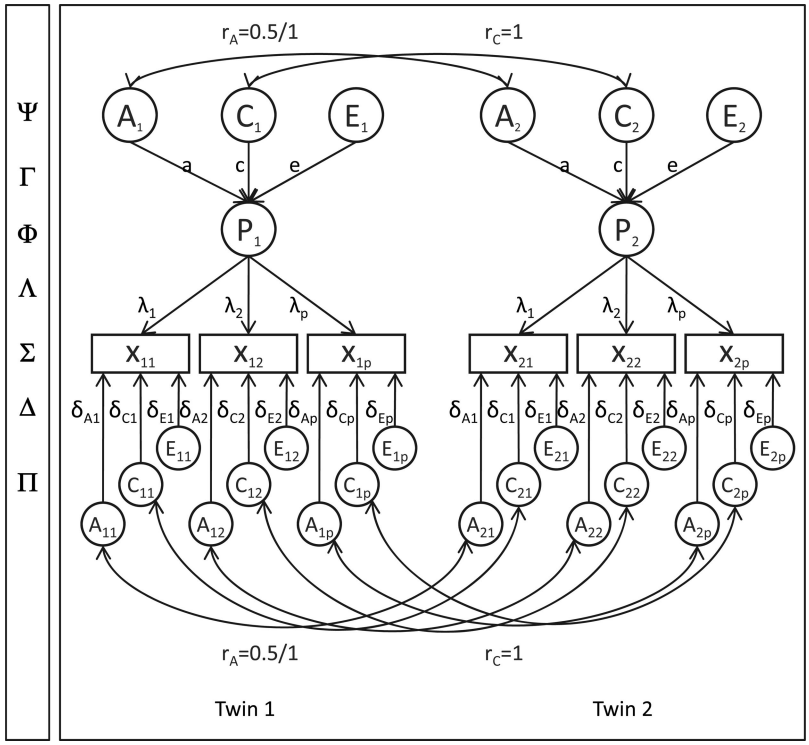


Figure A1. A common pathway genetic factor model with common and residual additive genetic (A), shared environmental (C), and individual-specific environmental (E) factors.

Appendix B

Simulation Script

The R code used to simulate data and perform the phenotypic exploratory factor analysis (EFA) is given below. The notation in the code corresponds to that in the text (e.g., Λ is *lambda* in the code). Note: In writing the code we tried to be explicit and have therefore sacrificed concision for clarity.

```
#Model 0:
library(MASS)
n=1000 #sample size
nx=12 #number of indicators
np=1 # number of psychometric factors
na=nc=ne=1 # number of A, C, and E factors
l=sqrt(seq(.1,,by=.05,length.out=nx)) #
loadings of the indicators on the
psychometric factor
a=sqrt(.5) # a factor loadings
c=sqrt(.3) # c factor loadings
e=sqrt(.2) # e factor loadings
psy.a=psy.c=psy.e=1 # variances of the A,
C, and E factors
##########
lambda=matrix(l,nx,np)
gamma.a=matrix(c(a),np,na) # matrix of a
factor loadings
gamma.c=matrix(c(c),np,nc) # matrix of c
factor loadings
gamma.e=matrix(c(e),np,ne) # matrix of e
factor loadings
phi.a=gamma.a%*%psy.a%*%t(gamma.a) # A
variance component of the psychometric
factor
```

(*Appendices continue*)

```
phi.c=gamma.c%*%psy.c%*%t(gamma.c) # C
variance component of the psychometric
factor
phi.e=gamma.e%*%psy.e%*%t(gamma.e) # E
variance component of the psychometric
factor
phi=phi.a+phi.c+phi.e # variance of the
psychometric factor
theta=diag(diag(diag(nx)-
lambda%*%phi%*%t(lambda))) # diagonal matrix
of item residuals for twin1 (twin2)
sigma11=lambda%*%phi%*%t(lambda)+theta #
expected covariance matrix of twin1 (twin2)
lp1.mz=lambda%*%(phi.a+phi.c)%*%t(lambda)
lp1.dz=lambda%*%(.5*phi.a+phi.c)%*%t(lambda)
theta12.mz=diag(diag(diag(.8,nx)-lp1.mz)) #
diagonal matrix of MZ twin1-twin2 covariance
among the residuals
theta12.dz=diag(diag(diag(.55,nx)-lp1.dz)) #
diagonal matrix of DZ twin1-twin2 covariance
among the residuals
sigma12.mz=lp1.mz+theta12.mz # expected MZ
twin1-twin2 covariance matrix
sigma12.dz=lp1.dz+theta12.dz # expected DZ
twin1-twin2 covariance matrix
sigma.mz=cbind(rbind(sigma11,sigma12.mz),
rbind(sigma12.mz,sigma11)) # expected MZ
covariance matrix
sigma.dz=cbind(rbind(sigma11,sigma12.dz),
rbind(sigma12.dz,sigma11)) # expected DZ
covariance matrix
data.ph=mvrnorm(n,rep(0,nx),sigma11,emp=T)
# simulate data for phenotypic analyses
data.mz=mvrnorm(n,rep(0,nx*2),sigma.mz,
emp=T) # simulate MZ twin data
data.dz=mvrnorm(n,rep(0,nx*2),sigma.dz,
emp=T) # simulate DZ twin data
data=cbind(rbind(data.mz,data.dz),rep(0:1,
each=n)) # 0 = MZ, 1 = DZ
write(t(data),"data 1a1c1e.dat",
ncol=2*nx+1)
##########
# generate the same covariance structure
from an IP model:
lambda.a=a*lambda
lambda.c=c*lambda
lambda.e=e*lambda
sigma11=lambda.a%*%psy.a%*%t(lambda
.a)+lambda.c%*%psy.c%*%t(lambda.c)+lambda
.e%*%psy.e%*%t(lambda.e)+theta
sigma12.mz=lambda.a%*%psy.a%*%t(lambda
.a)+lambda.c%*%psy.c%*%t(lambda.c)+theta12
.mz
sigma12.dz=.5*lambda.a%*%psy.a%*%t(lambda
.a)+lambda.c%*%psy.c%*%t(lambda.c)+theta12
.dz
#lambda.a/lambda.c # proportionality of a,
c, and e effects
#lambda.a/lambda.e
#lambda.c/lambda.e
##########
# or (to specify the ACE model for the
residuals explicitly):
delta.a=diag(rep(sqrt(seq(.45,,by=-.025,
length.out=nx)))) # matrix of A residual
factor loadings
delta.c=diag(rep(sqrt(seq(.27,,by=-.015,
length.out=nx)))) # matrix of C residual
factor loadings
delta.e=diag(rep(sqrt(seq(.18,,by=-.01,
length.out=nx)))) # matrix of E residual
factor loadings
pi.a=pi.c=pi.e=diag(nx)
theta.a=delta.a%*%pi.a%*%t(delta.a) # A
residual variance component in twin1 (twin2)
theta.c=delta.c%*%pi.c%*%t(delta.c) # C
residual variance component in twin1 (twin2)
theta.e=delta.e%*%pi.e%*%t(delta.e) # E
residual variance component in twin1 (twin2)
theta=theta.a+theta.c+theta.e # diagonal
matrix of item residuals for twin1 (twin2)
theta12.mz=theta.a+theta.c # residual MZ
twin1 - twin2 covariance
theta12.dz=.5*theta.a+theta.c # residual DZ
twin1 - twin2 covariance
#lambda.a%*%psy.a%*%t(lambda.a)+theta.a # A
variance component
#lambda.c%*%psy.c%*%t(lambda.c)+theta.c # C
variance component
#lambda.e%*%psy.e%*%t(lambda.e)+theta.e # E
variance component
#lambda.a%*%psy.a%*%t(lambda.a)+theta
.a+lambda.c%*%psy.c%*%t(lambda.c)+theta
.c+lambda.e%*%psy.e%*%t(lambda.e)+theta.e
#sigma11
##########
# phenotypic EFA:
cov.ph=cov(data.ph)
plot(1:nx,eigen(cov.ph)$values,type="b",
xlab="Item",ylab="Eigenvalue")
text(10.5,5,"Model 0')
efa=chi=df=p=rmsea=f.cor=as.list(rep(0,4))
for (i in 1:4){
efa[[i]]=factanal(covmat=cov.ph,n.obs=n,
factors=i,rotation="promax")
chi[[i]]=round(efa[[i]]$STATISTIC,2)
df[[i]]=efa[[i]]$dof
```

(*Appendices continue*)

```
p[[i]]=round(efa[[i]]$PVAL,2)
if(chi[[i]]-df[[i]]>0) {rmsea
[[i]]=(sqrt(chi[[i]]-df[[i]]))/n/df[[i]]}
if(i > 1) {rm=promax(unclass(efa
[[i]]$loadings))$rotmat
f.cor[[i]]=solve(rm%*%t(rm))
}}
res=cbind(chi,df,p,rmsea)
dimnames(res)[[1]]=paste(1:4,"factors',
sep=")
res
##########
#Model 1:
na_m2=2
lambda.a_m2=matrix(c(a*lambda[1:6,],rep(0,
nx),a*lambda[7:12,]),nx,na_m2)
psy.a_m2=diag(na_m2)
sigma11_m2=lambda.a_m2%*%psy.a_m2%*%t(lambda
.a_m2)+lambda.c%*%psy.c%*%t(lambda
.c)+lambda.e%*%psy.e%*%t(lambda.e)+theta
sigma12.mz_m2=lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c%*%psy
.c%*%t(lambda.c)+theta12.mz
sigma12.dz_m2=.5*lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c%*%psy
.c%*%t(lambda.c)+theta12.dz
sigma.mz_m2=cbind(rbind(sigma11_m2,sigma12
.mz_m2),rbind(sigma12.mz_m2,sigma11_m2)) #
expected MZ covariance matrix
sigma.dz_m2=cbind(rbind(sigma11_m2,sigma12
.dz_m2),rbind(sigma12.dz_m2,sigma11_m2)) #
expected DZ covariance matrix
data.ph=mvrnorm(n,rep(0,nx),sigma11_m2,
emp=T) # simulate data for phenotypic
analyses
data.mz=mvrnorm(n,rep(0,nx*2),sigma.mz_m2,
emp=T) # simulate MZ twin data
data.dz=mvrnorm(n,rep(0,nx*2),sigma.dz_m2,
emp=T) # simulate DZ twin data
data=cbind(rbind(data.mz,data.dz),rep(0:1,
each=n)) # 0 = MZ, 1 = DZ
write(t(data),"data 2a1c1e.dat",
ncol=2*nx+1)
##########
#Model 2:
ne_m3=2
lambda.e_m3=matrix(c(e*lambda[1:3],rep(0,6),
e*lambda[10:12],rep(0,3),e*lambda[4:9],
rep(0,3)),nx,ne_m3)
psy.e_m3=diag(ne_m3)
sigma11_m3=lambda.a_m2%*%psy.a_m2%*%t(lambda
.a_m2)+lambda.c%*%psy.c%*%t(lambda
.c)+lambda.e_m3%*%psy.e_m3%*%t(lambda
.e_m3)+theta
sigma12.mz_m3=lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c%*%psy
.c%*%t(lambda.c)+theta12.mz
sigma12.dz_m3=.5*lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c%*%psy
.c%*%t(lambda.c)+theta12.dz
sigma.mz_m3=cbind(rbind(sigma11_m3,sigma12
.mz_m3),rbind(sigma12.mz_m3,sigma11_m3)) #
expected MZ covariance matrix
sigma.dz_m3=cbind(rbind(sigma11_m3,sigma12
.dz_m3),rbind(sigma12.dz_m3,sigma11_m3)) #
expected DZ covariance matrix
data.ph=mvrnorm(n,rep(0,nx),sigma11_m3,
emp=T) # simulate data for phenotypic
analyses
data.mz=mvrnorm(n,rep(0,nx*2),sigma.mz_m3,
emp=T) # simulate MZ twin data
data.dz=mvrnorm(n,rep(0,nx*2),sigma.dz_m3,
emp=T) # simulate DZ twin data
data=cbind(rbind(data.mz,data.dz),rep(0:1,
each=n)) # 0 = MZ, 1 = DZ
#write(t(data),"data 2a1c2e.dat",
ncol=2*nx+1)
##########
#Model 3:
nc_m4=3
lambda.c_m4a=t(matrix(c(
1,0,0,
0,1,0,
0,0,1,
1,0,0,
0,1,0,
0,0,1,
1,0,0,
0,1,0,
0,0,1,
1,0,0,
0,1,0,
0,0,1)
,nx,nc_m4,byrow=T))
lambda.c_m4a[lambda.c_m4a==1]=c*lambda
lambda.c_m4=t(lambda.c_m4a)
psy.c_m4=diag(nc_m4)
sigma11_m4=lambda.a_m2%*%psy.a_m2%*%t(lambda
.a_m2)+lambda.c_m4%*%psy.c_m4%*%t(lambda
.c_m4)+lambda.e_m3%*%psy.e_m3%*%t(lambda
.e_m3)+theta
sigma12.mz_m4=lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c_m4%*%psy
.c_m4%*%t(lambda.c_m4)+theta12.mz
sigma12.dz_m4=.5*lambda.a_m2%*%psy
.a_m2%*%t(lambda.a_m2)+lambda.c_m4%*%psy
.c_m4%*%t(lambda.c_m4)+theta12.dz
```

(*Appendices continue*)

```
sigma.mz_m4=cbind(rbind(sigma11_m4,sigma12
.mz_m4),rbind(sigma12.mz_m4,sigma11_m4)) #
expected MZ covariance matrix
sigma.dz_m4=cbind(rbind(sigma11_m4,sigma12
.dz_m4),rbind(sigma12.dz_m4,sigma11_m4)) #
expected DZ covariance matrix
data.ph=mvrnorm(n,rep(0,nx),sigma11_m4,
emp=T) # simulate data for phenotypic
analyses
data.mz=mvrnorm(n,rep(0,nx*2),sigma.mz_m4,
emp=T) # simulate MZ twin data
data.dz=mvrnorm(n,rep(0,nx*2),sigma.dz_m4,
emp=T) # simulate DZ twin data
data=cbind(rbind(data.mz,data.dz),rep(0:1,
each=n)) # 0 = MZ, 1 = DZ
write(t(data),"data 2a3c2e.dat",
ncol=2*nx+1)
```

Appendix C

Child Behavior Checklist for Ages 4–18 (CBCL/4–18) Internalizing Scale: Item Correlations, Item Content, and Detailed Results of Phenotypic and Genetic Independent Pathway Analyses

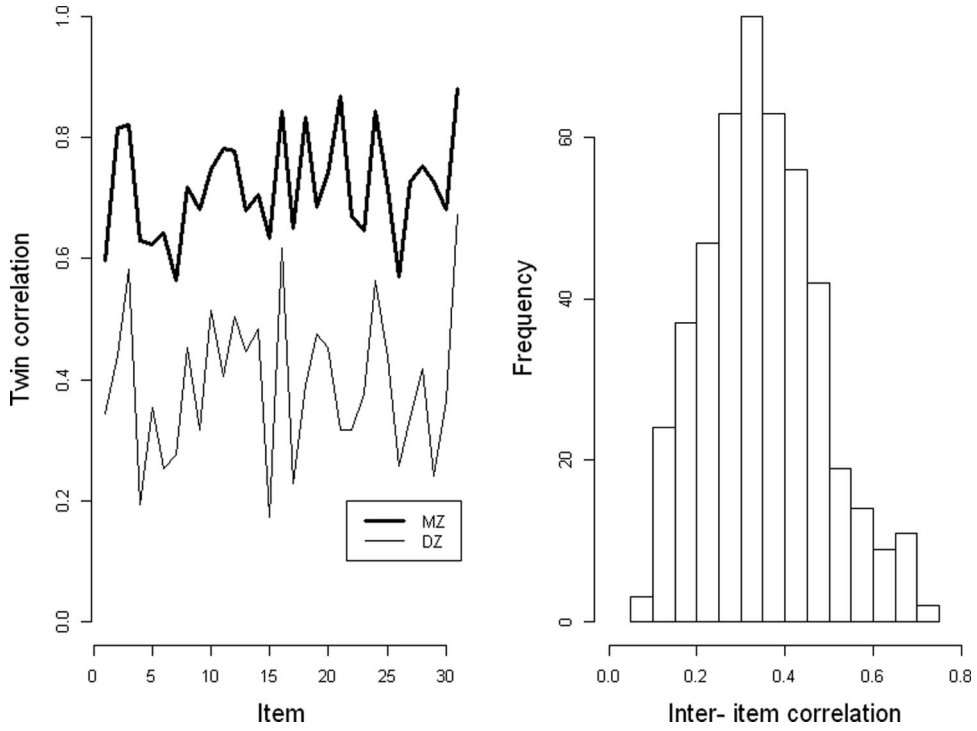


Figure C1. Monozygotic (MZ) and dizygotic (DZ) polychoric twin correlations (left) and distribution of inter-item polychoric correlations (right) for the 31 items of the Child Behavior Checklist for Ages 4–18 Internalizing scale.

(*Appendices continue*)

Table C1
Item Content of the CBCL/4–18 Internalizing Scale

Anxious/depressed	Withdrawn	Somatic complaints
1. Lonely	15. Rather be alone	23. Feels dizzy
2. Cries a lot	16. Would not talk	24. Overtired
3. Fears doing something bad	17. Secretive	25. Aches, pains
4. Must be perfect	18. Shy, timid	26. Headaches
5. Feels unloved	19. Stares blankly	27. Nausea
6. Others out to get him	20. Sulks	28. Eye problems
7. Feels worthless	21. Lacks energy	29. Skin problems
8. Nervous, tense	22. Withdrawn	30. Stomachaches
9. Fearful, anxious		31. Vomiting
10. Feels too guilty		
11. Self-conscious		
12. Suspicious		
13. Sad		
14. Worries		

Note. CBCL/4–18 = Child Behavior Checklist for Ages 4–18.

Table C2
Three-Factor Exploratory Factor Analysis Solution for the CBCL/4–18 Internalizing Scale

	Factor				
Item	AD	W	SC	Item content	R^2
AD1	**0.67**			Lonely	0.47
AD2	**0.41**			Cries a lot	0.20
AD3	0.25	**0.39**		Fears doing something bad	0.22
AD4	0.21	**0.27**		Must be perfect	0.12
AD5	**0.90**			Feels unloved	0.81
AD6	**0.78**			Others out to get him	0.61
AD7	**0.68**	0.21		Feels worthless	0.51
AD8		**0.42**		Nervous, tense	0.23
AD9		**0.56**		Fearful, anxious	0.35
AD10	0.29	**0.41**		Feels too guilty	0.26
AD11		**0.82**		Self-conscious	0.71
AD12	**0.54**	0.20		Suspicious	0.33
AD13	**0.6**	0.26		Sad	0.44
AD14	0.35	**0.41**		Worries	0.31
W1		**0.55**		Rather be alone	0.31
W2		**0.64**		Would not talk	0.43
W3		**0.78**		Secretive	0.62
W4		**0.95**		Shy, timid	1.09
W5		**0.51**		Stares blankly	0.30
W6	**0.40**			Sulks	0.20
W7		**0.51**		Lacks energy	0.27
W8		**0.82**		Withdrawn	0.69
SC1			**0.56**	Feels dizzy	0.33
SC2		0.26	**0.35**	Overtired	0.22
SC3			**0.76**	Aches, pains	0.58
SC4			**0.71**	Headaches	0.50
SC5			**0.84**	Nausea	0.70
SC6			**0.32**	Eye problems	0.11
SC7			**0.23**	Skin problems	0.06
SC8			**0.73**	Stomachaches	0.54
SC9			**0.67**	Vomiting	0.46
		Correlations			
Factor					
AD	—				
W	.64	—			
SC	.40	.42	—		
		Determinacies			
Factor	.96	.96	.94		

Note. N = 5,782. Only factor loadings larger than .2 are depicted. Highest factor loadings for each item appear in bold. CBCL/4–18 = Child Behavior Checklist for Ages 4–18; AD = Anxious/Depressed; W = Withdrawn; SC = Somatic Complaints.

(*Appendices continue*)

Table C3
Four-Factor Exploratory Factor Analysis Solution for the CBCL/4–18 Internalizing Scale

Item	Factor				Item content	R^2
	D	A	W	SC		
AD1	**0.70**				Lonely	0.50
AD2	**0.43**				Cries a lot	0.21
AD3	0.33	**0.52**			Fears doing bad	0.40
AD4	0.27	**0.40**			Must be perfect	0.23
AD5	**0.91**				Feels unloved	0.84
AD6	**0.80**				Others out to get him	0.64
AD7	**0.74**	0.2			Feels worthless	0.58
AD8	0.23	**0.38**			Nervous, tense	0.23
AD9		**0.50**			Fearful, anxious	0.31
AD10	0.38	**0.59**			Feels too guilty	0.50
AD11		0.47	**0.48**		Self-conscious	0.46
AD12	**0.57**				Suspicious	0.35
AD13	**0.65**				Sad	0.46
AD14	0.43	**0.45**			Worries	0.39
W1			**0.52**		Rather be alone	0.28
W2			**0.64**		Would not talk	0.44
W3			**0.78**		Secretive	0.61
W4		0.36	**0.68**		Shy, timid	0.71
W5	0.26		**0.44**		Stares blankly	0.27
W6	**0.42**				Sulks	0.23
W7			**0.50**		Lacks energy	0.27
W8			**0.71**		Withdrawn	0.52
SC1				**0.55**	Feels dizzy	0.32
SC2	0.20			**0.36**	Overtired	0.21
SC3				**0.78**	Aches, pains	0.63
SC4				**0.72**	Headaches	0.52
SC5				**0.84**	Nausea	0.71
SC6				**0.34**	Eye problems	0.13
SC7				**0.24**	Skin problems	0.07
SC8				**0.74**	Stomachaches	0.55
SC9				**0.67**	Vomiting	0.46
			Correlations			
Factor						
D	—					
A	.38	—				
W	.57	.41	—			
SC	.41	.38	.33	—		
			Determinacies			
Factor	.96	.89	.94	.96		

Note. $N = 5{,}782$. Only factor loadings larger than .2 are depicted. Highest factor loadings for each item appear in bold. CBCL/4–18 = Child Behavior Checklist for Ages 4–18; D = Depressed; A = Anxious; W = Withdrawn; SC = Somatic Complaints.

(*Appendices continue*)

Table C4
Three-Factor Confirmatory Factor Analysis Model for the CBCL/4–18 Internalizing Scale: Factor Loadings, R^2, and Factor Correlations

Item	Factor			Item content	R^2
	AD	W	SC		
AD1	0.75			Lonely	0.56
AD2	0.56			Cries a lot	0.31
AD3	0.54			Fears doing bad	0.29
AD4	0.50			Must be perfect	0.25
AD5	0.77			Feels unloved	0.59
AD6	0.74			Others out to get him	0.55
AD7	0.80			Feels worthless	0.64
AD8	0.62			Nervous, tense	0.39
AD9	0.66			Fearful, anxious	0.44
AD10	0.73			Feels too guilty	0.54
AD11	0.62			Self-conscious	0.39
AD12	0.69			Suspicious	0.48
AD13	0.84			Sad	0.71
AD14	0.76			Worries	0.57
W1		0.59		Rather be alone	0.35
W2		0.66		Would not talk	0.44
W3		0.72		Secretive	0.52
W4		0.56		Shy, timid	0.31
W5		0.67		Stares blankly	0.45
W6		0.63		Sulks	0.40
W7		0.62		Lacks energy	0.38
W8		0.83		Withdrawn	0.68
SC1			0.64	Feels dizzy	0.41
SC2			0.79	Overtired	0.62
SC3			0.79	Aches, pains	0.62
SC4			0.64	Headaches	0.40
SC5			0.81	Nausea	0.65
SC6			0.37	Eye problems	0.14
SC7			0.33	Skin problems	0.11
SC8			0.69	Stomachaches	0.48
SC9			0.60	Vomiting	0.36
		Correlations			
Factor					
AD	—				
W	.80	—			
SC	.57	.51	—		

Note. N = 5,783. CBCL/4–18 = Child Behavior Checklist for Ages 4–18; AD = Anxious/Depressed; W = Withdrawn; SC = Somatic Complaints.

(*Appendices continue*)

Table C5
Four-Factor Confirmatory Factor Analysis Model for the CBCL/4–18 Internalizing Scale: Factor Loadings, R^2, and Factor Correlations

Item	Factor				Item content	R^2
	D	A	W	SC		
AD1	1.00				Lonely	1.00
AD2	0.58				Cries a lot	0.34
AD3		1.00			Fears doing bad	1.00
AD4		0.53			Must be perfect	0.28
AD5	0.79				Feels unloved	0.62
AD6	0.77				Others out to get him	0.59
AD7	0.83				Feels worthless	0.68
AD8		0.66			Nervous, tense	0.44
AD9		0.70			Fearful, anxious	0.49
AD10		0.77			Feels too guilty	0.59
AD11		0.66			Self-conscious	0.44
AD12	0.72				Suspicious	0.51
AD13	0.87				Sad	0.75
AD14		0.81			Worries	0.65
W1			0.60		Rather be alone	0.35
W2			0.66		Would not talk	0.44
W3			0.72		Secretive	0.52
W4			0.56		Shy, timid	0.31
W5			0.67		Stares blankly	0.45
W6			0.63		Sulks	0.39
W7			0.62		Lacks energy	0.38
W8			0.83		Withdrawn	0.68
SC1				0.64	Feels dizzy	0.41
SC2				0.79	Overtired	0.62
SC3				0.79	Aches, pains	0.62
SC4				0.64	Headaches	0.40
SC5				0.81	Nausea	0.65
SC6				0.37	Eye problems	0.13
SC7				0.33	Skin problems	0.11
SC8				0.69	Stomachaches	0.48
SC9				0.60	Vomiting	0.36
		Factor correlations				
Factor						
D	—					
A	.73	—				
W	.73	.73	—			
SC	.49	.54	.51	—		

Note. N = 5,783. CBCL/4–18 = Child Behavior Checklist for Ages 4–18; D = Depressed; A = Anxious; W = Withdrawn; SC = Somatic Complaints.

(*Appendices continue*)

Table C6
Fit Measures and Interfactor Correlations for Independent Pathway Models With Different Dimensionalities of A, C, and E Factors

Model	χ2	*df*	CFI	TLI	RMSEA	Interfactor correlations											
						Factor	A1	A2	A3	Factor	C1	C2	C3	Factor	E1	E2	E3
4A 4C 4E	1161	542	.979	.984	.020	A2	.64			C2	***.99***			E2	.79		
						A3	***.99***	.52		C3	***.99***	***1.00***		E3	.47	.54	
						A4	.31	.35	.32	C4	***.99***	***.97***	***.96***	E4	.39	.34	.17
3A 3C 3E	1142	534	.980	.984	.020	A2	.85			C2	***1.00***			E2	.55		
						A3	.32	.14		C3	***1.00***	***1.00***		E3	.36	.26	
3A 1C 3E	1140	532	.980	.984	.020	A2	***.98***							E2	.52		
						A3	.34	.13						E3	.36	.26	
2A 1C 3E	1148	532	.979	.984	.020	A2	.26							E2	.52		
														E3	.39	.17	
2A 1C 3E[a]	1245	533	.976	.982	.022	A2	.16							E2	.78		
														E3	.30	.47	
2A 1C 4E	1082	531	.982	.986	.019	A2	.20							E2	.70		
														E3	.56	.34	
														E4	.31	.47	.21

Note. The three- and the four-factor-based independent pathway models based on the phenotypic results (see Figures 7B and 7C) are shown in bold. Interfactor correlations greater than .9 are shown in italic bold. A = additive genetic factor; C = shared environmental factor; E = individual-specific environmental factor; CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root-mean-square error of approximation.
[a] The clustering for the E structure is Depressed, Anxious/Withdrawn, Somatic Complaints, instead of Anxious/Depressed, Withdrawn, Somatic Complaints.

Received December 18, 2009
Revision received February 5, 2013
Accepted February 20, 2013 ■

Correction to Franić, Dolan, Borsboom, Hudziak, van Beijsterveldt, and Boomsma (2013)

In the article "Can Genetics Help Psychometrics? Improving Dimensionality Assessment Through Genetic Factor Modeling" by Sanja Franić, Conor V. Dolan, Denny Borsboom, James J. Hudziak, Catherina E. M. van Beijsterveldt, and Dorret I. Boomsma (*Psychological Methods*, Vol. 18, No. 3, pp. 406–433. doi: 10.1037/a0032755), funding information was omitted from the author note. The author note should have stated that this research was funded by the European Research Council Grant 230374 to D.I. Boomsma.

DOI: 10.1037/a0036139