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Decomposition of Multivariate Phenotypic Means in Multigroup Genetic Covariance Structure Analysis

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Observed differences in phenotypic means between groups such as parents and their offspring or male and female twins can be decomposed into genetic and environmental components. The decomposition is based on the assumption that the difference in phenotypic means is due to a difference in the location of the normal genetic and environmental distributions underlying the phenotypic individual differences. Differences between the groups in variance can be accommodated insofar as they are due to differences in unique variance or can be modeled using a scale parameter. The decomposition may be carried out in the standard analysis of genetic covariance structure using, for instance, LISREL. Illustrations are given using simulated data and twin data relating to blood pressure. Other possible applications are mentioned.

KEY WORDS: group differences in phenotypic means; genetic means; environmental means; genetic and environmental covariance structure; twin data; parent–offspring data.

INTRODUCTION

Behavior genetics is concerned with the relationship between genotypic and environmental differences and phenotypic differences in behavior. Where control can be exercised over the genetic and environmental influences on the subjects, differences in means and variances between groups (e.g., true breeding lines and their crosses) may be studied. Where

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this is not the case, as in human populations, behavioral genetic analyses are usually limited to individual differences (Mather and Jinks, 1977).

Nonetheless, attempts to obtain a biometric decomposition of phenotypic means in human samples have been undertaken. Notably, McArdle (1986) has fit individual growth curves to repeatedly measured twins and simultaneously obtained estimates of genetic and environmental contributions to individual differences in the shape and level parameters. A similar approach was taken by Vandenberg and Falkner (1965), using less sophisticated methods, and is well-known in livestock research (e.g., Grossman and Bohren, 1985). In two papers Dolan *et al.* (1989, 1991) have considered the analysis of means and individual differences by expressing the phenotypic means as linear combinations of genetic and environmental (i.e., latent) means. Their approach can be conveyed simply as $\mathbf{m} = \mathbf{An}$, where $\mathbf{m}$ is a vector of phenotypic means, $\mathbf{A}$ can be seen as a weight matrix, and $\mathbf{n}$ is the vector of latent means. The matrix $\mathbf{A}$ and the vector $\mathbf{m}$ are known, so that $\mathbf{n}$ can be estimated given that $\mathbf{A}$ is full column rank. The information in $\mathbf{A}$ is obtained from the standard genetic covariance structure analysis, be it based on the common factor model (Martin and Eaves, 1977; Heath *et al.*, 1989) or some variation of the simplex model (Eaves *et al.*, 1986; Boomsma and Molenaar, 1987; Hewitt, 1990).

There are two points to be made about this approach. First, one requires multiple indicators of the common latent variables for purposes of identification. Second, one has to take into account the arbitrariness of measurement scale and the possibility that the phenotypic means are only in part due to the latent variables in the analysis. The latter point implies that $\mathbf{n}$ should contain parameters to take into account the measurement origin. In repeated-measures designs, one naturally has multiple indicators measured on a commensurate scale so that, given certain testable assumption, these requirements can be met (see Dolan *et al.*, 1991). In the cross-sectional designs, on the other hand, one may have multiple indicators, but these will generally not have commensurate scales. This makes it difficult to take into account the possible difference in measurement origin. In Dolan *et al.* (1989) this point is ignored, prompting Hewitt *et al.* (1989) to remark that this approach "implies a strong hypothesis about the action of the genes and/or the environment and about the scale of measurement. This strong hypothesis is unlikely to be confirmed in practice for psychological data" (p. 763).

In the present paper we apply a method due to Sörbom (1974) to multivariate data collected in a cross-sectional twin study. Sörbom's approach takes into account the arbitrariness of the measurement scales by

estimating *differences* in means between groups instead of absolute latent means within a group (as by Dolan *et al.*, 1989).

The Sörbom method allows a decomposition of multivariate phenotypic differences in means into genetic and environmental components based on the following model concerning the constitution of the phenotypic means. A phenotypic mean is modeled as a linear combination of (1) a contribution due to those genetic and environmental factors that also contribute to the individual differences and (2) a contribution of unknown origins which may include the contribution of those genetic and environmental factors which contribute to the overall level but not to the individual differences. The former are referred to as species-variable influences; the latter, as species-constant influences. The differences in phenotypic means are assumed in the present approach to be due to differences between the groups in the contributions to the phenotypic means of the species variable genetic and environmental factors.

The decomposition of the phenotypic means observed in human samples is carried out under the following conditions: (1) the species-constant influences are assumed to be identical in both groups, and (2) the latent environmental and genetic variables have either identical variances across the groups or differences in variances which can be modeled by means of a scalar parameter (see below) or as differences in unique variance. The latter condition is necessary because it is not possible to identify both differences between groups in the common-factor variances (other than those attributable to a scalar effect; see below) and differences in location parameters of the common latent distribution; the former, because it is not possible to identify mean differences originating from differences in species-constant influences. The assumptions mentioned here are shown to be testable in the analysis of covariance structure (Martin and Eaves, 1977).

The Sörbom method is explained for twin data, where group membership is determined by gender, and applied to simulated and real twins. Other possibilities, including parent–offspring comparison and repeatedly measured twins, are mentioned.

SÖRBOM'S METHOD APPLIED TO MULTIVARIATE TWIN DATA

Consider the multigroup biometrical common-factor model (cf. Martin and Eaves, 1977):

$$\mathbf{Y}_{zk} = \boldsymbol{\nu} + \boldsymbol{\Lambda}\,\boldsymbol{\eta}_{zk} + \boldsymbol{\epsilon}_{zk} \qquad (1)$$

where the subscript z denotes the zygosity (dizygotic or monozygotic) and k denotes the group membership (subject subscripts are dropped to ease presentation). Let p indicate the number of observed variables measured obtained from each individual and q the number of common factors underlying the covariance structure of the p variables. Then $\mathbf{Y}_{zk}$ ($2p \times 1$) is a vector of observed variables of the first, $\mathbf{Y}_{zk1}$ ($p \times 1$), and second $\mathbf{Y}_{zk2}$ ($p \times 1$), member of a twin pair: $\mathbf{Y}_{zk}^t = [\mathbf{Y}_{zk1}^t, \mathbf{Y}_{zk2}^t]$. The $2p$-dimensional vector $\boldsymbol{\nu}^t = [\boldsymbol{\nu}^{*t}, \boldsymbol{\nu}^{*t}]$, where $\boldsymbol{\nu}^*$ ($p \times 1$) contains the measurement origins of the observed variables. The measurement origins consist of an arbitrary scale constant and the contribution of species constant genetic and environmental factors. These parameters are pooled because they are indistinguishable in the present model. The matrix $\boldsymbol{\Lambda}$ ($2p \times 2q$) contains the factor loadings of the observed variables on the common latent genetic and environmental variables. The parameters in $\boldsymbol{\Lambda}$ are invariant across groups in accordance with the assumption that the latent variances are identical across the groups. The $2q$-dimensional vector $\boldsymbol{\eta}_{zk}$ contains the common latent genetic and environmental variables of each member of a twin pair of zygosity z in group k and the $2p$-dimensional vector $\boldsymbol{\epsilon}_{zk}$ contains unique factors in group k and zygosity z.

The covariance matrix of group $_{zk}$ can be written as

$$\begin{aligned}\boldsymbol{\Sigma}_{zk} &= \mathbf{E}[(\mathbf{Y}_{zk} - \boldsymbol{\nu} - \boldsymbol{\Lambda}\mathbf{E}[\boldsymbol{\eta}_{zk}])(\mathbf{Y}_{zk} - \boldsymbol{\nu} - \boldsymbol{\Lambda}\mathbf{E}[\boldsymbol{\eta}_{zk}]^t] \\ &= \boldsymbol{\Lambda}\boldsymbol{\Psi}_{zk}\boldsymbol{\Lambda}^t + \boldsymbol{\Theta}_{zk} \qquad (2)\end{aligned}$$

where $\boldsymbol{\Psi}_{zk}$ is the ($2q \times 2q$) covariance matrix of the common factors in group $_{zk}$, and $\boldsymbol{\Theta}_{zk}$ ($2p \times 2p$) the covariance matrix of the unique factors in group $_{zk}$. The matrix $\boldsymbol{\Theta}_{zk}$ is taken to be diagonal, although this is not necessary.

The phenotypic averages are a linear combination of the measurement origins and the common-factor means. The unique factors are assumed not to contribute to the observed means.

$$\mathbf{E}[\mathbf{Y}_k] = \boldsymbol{\nu} + \boldsymbol{\Lambda}\mathbf{E}[\boldsymbol{\eta}_k] \qquad (3)$$

We have now dropped the subscript denoting the zygosity, as the phenotypic means are taken to be equal across the zygosities but not across the groups. Equation (3) clarifies the necessity of invariant (across groups) factor loadings. If the factor loadings were not invariant, a difference in phenotypic mean vectors could not be interpreted unambigously as being due to differences in the means of factors. It could be due to either a difference in factor loading or a difference in mean or both. This requirement is reminiscent of the requirement of homogeneity of (co-)variances in the multivariate analysis of variance (MANOVA) model. In

the present case, however, unequal variances can be accommodated to a degree. First, differences across the groups in unique variances ($\mathbf{\Theta}_{zk}$) can be modeled. Second, differences in the dispersion in the common factor space can be accommodated insofar as they are attributable to a simple scalar effect. In this case Eq. (2) is replaced by

$$\mathbf{\Sigma}_{zk} = \mathbf{\Lambda}_k \mathbf{\Gamma}_k \mathbf{\Psi}_{zk} \mathbf{\Gamma}_k \mathbf{\Lambda}^t + \mathbf{\Theta}_{zk} \tag{4}$$

where the diagonal matrix $\mathbf{\Gamma}_k$ ($2q \times 2q$) equals $\gamma_k I$ and γ_k represents the scale parameter in group k. This device, which is applied below, allows one to retain an equal ratio of the common additive genetic variance to the common (in the sense of the common factor) environmental variance across the groups, while the absolute values of these variance terms may differ. The introduction of unequal unique variances and the introduction of a scalar effect leave the equation for the means [Eq. (3)] unaffected.

Let us assume that the group membership is determined by gender (m for male, f for female). Given three ($p=3$) observed variables for each subject and common additive genetic and unshared environmental factors ($q=2$), Eqs. (2) and (3) imply the following equations for the phenotypic means:

$$\begin{aligned}
\mu_{\text{mzf}i} &= \nu_i + \lambda_{gi} E[G_{\text{f}}] + \lambda_{ei} E[E_{\text{f}}] \\
\mu_{\text{mzm}i} &= \nu_i + \lambda_{gi} E[G_{\text{m}}] + \lambda_{ei} E[E_{\text{m}}] \\
\mu_{\text{dzf}i} &= \nu_i + \lambda_{gi} E[G_{\text{f}}] + \lambda_{ei} E[E_{\text{f}}] \\
\mu_{\text{dzm}i} &= \nu_i + \lambda_{gi} E[G_{\text{m}}] + \lambda_{ei} E[E_{\text{m}}]
\end{aligned} \tag{5}$$

Here $\mu_{\text{mzf}i}$ is the observed phenotypic mean of the monozygotic female (mzf) twins on variable i ($i=1,3$). Each factor loading, λ, has two subscripts which denote, respectively, whether it is a genetic or an environmental factor loading (g or e) and the variable to which the loading belongs ($i=$ 1 to 3). Considering only the nonredundant equations of the phenotypic means (redundancy arising from the assumption that means differ across the sexes but not across the zygosities), we have the following linear equations:

$$\begin{aligned}
\mu_{if} &= \nu_i + \lambda_{ei} E[E_{\text{f}}] + \lambda_{gi} E[G_{\text{f}}], \qquad i = 1, 3 \\
\mu_{im} &= \nu_i + \lambda_{ei} E[E_{\text{m}}] + \lambda_{gi} E[G_{\text{m}}], \qquad i = 1, 3
\end{aligned} \tag{6}$$

Assuming that the factor loadings are known, there are six equations and seven unknown parameters ($E[E_{\text{f}}]$, $E[G_{\text{f}}]$, $E[E_{\text{m}}]$, $E[G_{\text{m}}]$, ν_1, ν_2, ν_3) so that, given three phenotypic variables, the parameters are not identified. The addition of phenotypic indicators may render the number of equations greater than the number of unknowns, but even then the equations can be shown to be inconsistent due to the equality of the factor loadings.

Following Sörbom (1974), this problem is solved by fixing either the female or the male latent means to equal zero, so that the parameters are reduced to $\Delta E[G] = E[G_m] - E[G_f]$, $\Delta E[E] = E[E_m] - E[E_f]$, ;$\nu_1$, ν_2, and ν_3. In this way the differences in latent means are modeled as contrasts, yielding the following equations:

$$\begin{aligned} \mu_{if} &= \nu_i + \lambda_{ei}\Delta E[E] + \lambda_{gi}E[G], \qquad i = 1, 3 \\ \mu_{im} &= \nu_i \qquad\qquad\qquad\qquad\qquad\quad i = 1, 3 \end{aligned} \tag{7}$$

By equating the parameters ν_i with the phenotypic means in group m, these parameters now consist of three components. Two components are indistinguishable: the contributions of species constant factors and the contribution of an arbitrary measurement origin. The third component consists of the contribution of the species variable factor in group m. The parameters $\Delta E[E]$ and $\Delta E[G]$, as mentioned above, now represent the differences in the contributions of the species variable factors between the groups.

The hypothesis is tested that the common factors (E and G) account for the differences in phenotypic means between the sexes. It is possible to specify biometric models for those components of variance which are not explained by the common factors (Martin and Eaves, 1977). Such unique submodels may contain group-related differences in parameters because we are concerned only with the contribution of the communal part of the model to the difference in the mean vectors. However, the introduction of additional parameters to model the contribution of unique terms to the phenotypic means would quickly render the model for the means void in the sense that the parameters would outnumber the equations.

The model has been discussed for the situation in which the communal part of the model consists of an additive genetic factor and an unshared environmental factor. The inclusion of additional common latent variables (e.g., a shared environmental factor) would require additional observed variables. However, if one is satisfied with the decomposition into G and E components without estimating the respective contributions of unshared and common environmental influences, the introduction of a shared environmental factor can be avoided by estimating the correlation between the environmental factors of the twins. This is demonstrated below.

Generally one will minimally require one phenotypic variable in addition to the number of distinct latent common variables which contribute to the means. In the case of a bivariate phenotypic vector, one can, of course, still test the hypothesis that a group-related difference in

the phenotypic means vectors is due to any one of the communal latent factors.

Having set out the principle of the Sörbom method of the decomposing phenotypic means, we now give a number of applications. First, the model is illustrated using simulated twin data.

ILLUSTRATION USING SIMULATED TWIN DATA

In the present illustration using simulated data, we again assume that gender determines group membership. Four samples were generated consisting of a trivariate phenotype in 50 monozygotic (MZ) female twin pairs, 50 MZ male twin pairs, 50 dizygotic (DZ) female twin pairs, and 50 DZ male twin pairs. The common part of the biometrical factor model contains an additive genetic factor and a specific environmental factor which are uncorrelated. The additive genetic correlations between the genetic factors are .5 for the DZ twins and 1.0 for the MZ twins. Unique variances, which are uncorrelated between the phenotypes of the twin pairs, were chosen to equal 6 for each variable.

True parameter values are given in Table 1. The following latent mean values were chosen: $E[E_m] = 1$, $E[G_m] = 3$, $E[E_f] = 2$, $E[G_f] = 2$. The contrasts then equal $\Delta E[E] = -1$ and $\Delta E[G] = 1$. These

Table I. True and Recovered Parameters (ML Estimates) for the Analysis of Simulated Twin Data[a]

		Covariance	
Parameter	True	No means	With means
λg1	3	3.262 (.27)	3.248 (.27)
λg2	4	4.602 (.48)	4.495 (.48)
λg3	2	2.407 (.34)	2.494 (.30)
λe1	2	1.985 (.24)	1.975 (.24)
λe2	5	5.002 (.41)	5.155 (.37)
λe3	3	3.240 (.32)	3.019 (.23)
ν_1	10		9.584 (.37)
ν_2	18		18.229 (.57)
ν_3	10		10.059 (.36)
$\Delta E[G]$	1		1.063 (.32)
$\Delta E[E]$	−1		−1.219 (.32)
χ^2(dF)		86.25 (75)	109.86 (94)
p		.17	.13

[a] Standard errors are given in parentheses. Error variances are not shown. λg1 denotes the factor loading of the first phenotypic variable of the genetic factor. ν_1 denotes the measurement origin of the first phenotypic variable. $\Delta E[G]$ denotes the contrast $E[G_f] - E[G_m]$.

parameters give rise to male phenotypic means equaling 11, 17, and 9. The female phenotypic means are 10, 18, and 10.

First, the covariance structure is analyzed using the program LISREL VII (Jöreskog and Sörbom, 1988). The estimation in this and all subsequent analyses is by normal-theory maximum likelihood (ML). When the assumption of multivariate normality proves untenable, Browne's (1984) asymptotically distribution free (ADF) estimator can be used to obtain asymptotically correct results (Browne, 1984) (in LISREL VII this loss function is referred to as weighted least squares). Muthén (1989, Eqs. 6 and Eq. 16) has extended this estimator for multigroup analyses including structured means, so that it can be applied readily in the present context. The ADF estimator does, however, require large sample sizes.

The results are shown in Table I. The overall goodness of fit is acceptable [$\chi^2(75) = 86.25, p = .17$]. The parameter estimates of the factor loadings and the unique variances do not, judging by their standard errors, differ significantly from their true values. Having established the simple genetic model as providing a good explanation of the covariance structure, the means are introduced. The results are again shown in Table 1. First, the overall goodness of fit is acceptable [$\chi^2(94) = 109.86, p = .13$]. The factor loadings are again close to the true values and similar to those obtained in the previous analysis. The true differences in latent means equal $\Delta E[G] = 1$ and $\Delta E[E] = -1$ and are estimated as 1.063(SE = .322) and −1.219(SE = .321).

It was mentioned in the Introduction that the decomposition of the phenotypic means into measurement origins and contrasts is based on the assumption that those (species constant) factors that contribute to the means, but not to the variances, are identical in the groups. It is important that the model be rejected when these factors differ between the groups in their contributions to the means. If the model cannot be rejected in these circumstances, any estimated difference in the latent means cannot unequivocally be attributed to those genetic and environmental factors which also contribute to the individual differences. To test this aspect of the model, the female data were transformed adding a constant to the first and third phenotypic means and subtracting it from the second ($\mu_{1f} + c$, $\mu_{2f} + c$, $\mu_{3f} + c$). Three values for c were chosen to equal 1, 2, and 3 so that we may see the effect of an inceasingly greater departure of the assumption of identical species constant influences. In the male samples, the original phenotypic means were retained. The analysis of the augmented moment matrices were repeated after each transformation. The following $\chi^2(94)$ were obtained: 109.86 ($c = 0, p = .13$), 114.48 ($c = 1, p = .07$), and 123.72 ($c = 2, p < .01$). When c was set to equal 3, it was not possible to obtain estimates because the estimation

procedure consistently failed to converge. As such the model is rejected only when $c = 2$. The failure to reject the model when $c = 1$ is attributable partly to the fact that minor changes in the factor loadings may partly compensate for the difference in means. The analyses were therefore repeated with the factor loadings fixed to the values obtained from the analysis of covariance structure without the means. We do subtract degrees of freedom for these fixed factor loadings so that the number of degrees of freedom remains the same. The $\chi^2(94)$ values thus obtained equaled 113.67 ($c = 0, p = .08$), 127.84 ($c = 1, p = .01$), and 215.74 ($c = 2, p < 0.001$).

APPLICATION TO REAL TWIN DATA: BLOOD PRESSURE

To illustrate the Sörbom method using real data, data relating to blood pressure are analyzed. As a part of a larger experiment three variables relating to blood pressure were recorded in a sample of twins under a number of experimental conditions [see Boomsma *et al.* (1990) for details of this experiment]. These variables are systolic (SYST), diastolic (DIAST), and mean arterial pressure (MAP) recorded in a rest condition. This condition followed a choice reaction time task and preceded a mental arithmetic task. The sample sizes were 35 MZM pairs, 31 DZM pairs, 35 MZF pairs, 30 DZF pairs and 29 DZOS pairs. Summary statistics are given in Table II.

Again, LISREL is used to obtain normal-theory ML estimates. In view of the requirement of multivariate normality, marginal and multivariate skewness and kurtosis were calculated for each group. These statistics are presented in Table II. Multivariate skewness and kurtosis (Mardia, 1980) are tested using asymptotic sample distributions. Let $r_i^2 = (x_i - E[x_i])'\mathbf{S}^{-1}(x_i - E[x_i])$ and $r_{ij} = (x_i - E[x_i]'\mathbf{S}^{-1}(x_j - E[x_j])$. Here x_i is the q-variate phenotype of case i and $\mathbf{S}$ is the sample covariance matrix. Multivariate kurtosis is calculated as $1/n\ \Sigma r_i^4$, and multivariate skewness as $1/n^2\ \Sigma\Sigma r_{ij}^3$ (n is sample size). The asymptotic sample distributions are given by Mardia (1980, pp. 310–311). Mardia's measure of relative multivariate kurtosis, η, which is derived from the multivariate kurtosis statistic, is also given in Table II. This measure often appears in articles using ML estimation, as an informal criterion of peakedness: η is the ratio of the observed multivariate kurtosis to the expected multivariate kurtosis. Under multivariate normality η thus equals one. The PRELIS program (Jöreskog and Sörbom, 1986) includes an option to calculate this statistic.

There is no evidence of a departure of multivariate normality judging by the univariate skewnesses and kurtoses except perhaps in the MZF

Table II. Blood Pressure Data Summary Statistics[a]

Monozygotic males (n pair = 35)					
Covariance matrix					
t1syst	105.19				
t1diast	43.73	55.85			
t1map	62.85	45.56	58.95		
t2syst	73.18	27.85	42.45	150.00	
t2diast	32.70	23.69	28.88	55.99	43.80
t2map	42.88	20.53	31.15	74.00	43.52
Mean	129.69	72.94	90.90	131.63	73.60
Skew	.35	−.08	.22	.13	.07
Kurtosis	.37	−.57	.19	−.34	.81
Multivariate skewness	11.8				
Multivariate kurtosis	48.7			η = 48.7/48.0 = 1.01	
Dizygotic males (n pair = 31)					
Covariance matrix					
t1syst	139.01				
t1diast	86.36	84.76			
t1map	98.53	75.26	85.23		
t2syst	47.87	24.52	27.56	171.89	
t2diast	46.39	33.27	28.67	87.83	81.17
t2map	42.90	27.21	27.64	105.66	75.24
Mean	129.61	73.84	90.80	129.65	74.17
Skew	.01	−.30	.30	.65	−.44
Kurtosis	−.11	.72	.33	1.36*	−.07
Multivariate skewness	8.8				
Multivarate kurtosis	44.2			η = 44.2/48.0 = 0.92	
Monozygotic females (n pair = 35)					
Covariance matrix					
t1syst	93.27				
t1diast	23.28	21.07			
t1map	49.02	23.85	42.73		
t2syst	65.37	12.89	27.46	104.13	
t2diast	20.29	11.37	14.97	38.32	36.09
t2map	35.41	16.24	26.17	54.12	35.33
Mean	124.27	75.13	90.24	125.46	73.81
Skew	.94*	.51	1.03*	.21	.73*
Kurtosis	.40	.74	1.40*	−.50	.59
Multivariate skewness	10.1				
Multivarate kurtosis	47.0			η = 47.0/48.0 = 0.98	

Table II. Blood Pressure Data Summary Statistics[a] (Continued)

Dizygotic females (n pair = 30)						
Covariance matrix						
t1syst	196.51					
t1diast	69.78	43.89				
t1map	93.46	45.76	66.32			
t2syst	73.26	23.81	30.42	183.33		
t2diast	31.87	9.65	9.68	76.10	47.45	
t2map	43.96	12.61	18.22	109.73	53.66	80.32
Mean	126.29	77.18	91.86	125.69	75.85	90.51
Skew	.88*	−.20	.28	.63	.31	.52
Kurtosis	.44	−.29	.79	−.43	−.85	−.29
Multivariate skewness	13.4					
Multivarate kurtosis	48.0			η = 48.0/48.00 = 1.00		
Dizygotic Opposite Sex (n pair = 29)						
Covariance matrix						
tmsyst	177.16					
tmdiast	106.86	87.47				
tmmap	104.35	79.10	82.48			
tfsyst	−.74	−2.56	7.00	122.89		
tfdiast	4.40	1.56	3.70	54.51	44.97	
tfmap	−5.38	−5.47	−2.53	68.55	44.86	60.67
Mean	130.07	73.65	91.54	123.50	74.03	89.33
Skew	.52	.09	.40	.55	.57	.38
Kurtosis	−.57	−.46	−.43	−.61	−.50	−.64
Multivariate skewness	10.5					
Multivarate kurtosis	44.8			η = 44.8/48.0 = 0.93		

[a] t1syst denotes systolic blood pressure in twin 1. Diast stands for diastolic blood pressure, and map for mean arterial pressure. Significance of univariate skewnesses and kurtoses established using Royston (1985); significance of multivariate skewness and kurtoses tested against theoretical sample distribution (see Mardia, 1980).

* $p < .05$.

sample. Caution should be taken in drawing hard and fast conclusions, however, in view of the fact that the present sample sizes do not justify the use of asymptotic sample distributions of either the multivariate or the univariate statistics. The ADF estimator (Browne, 1984; Muthén, 1989) mentioned above is not considered here in view of the small sample sizes.

We observe the following means in the total female sample (N =

159, i.e., 35 * 2 MZF twins + 30 * 2 DZF twins + 29 DZOS female twins): 124.9 (SYST). 75.1 (DIAST), and 90.2 (MAP). In the total male sample (N = 161, derived analogously), we find 130.1 (SYST), 73.6 (DIAST), and 91.0 (MAP).

Table III contains the χ^2 statistics for the models that were fit to the 5 (6 × 6) covariance matrices. We started with the following simple model: common $E1$ and G factors and unique variances (as in the analysis of simulated twin data above). This model is denoted model 1 in Table III. All estimated parameters are constrained to be equal across the samples. This model gave a $\chi^2(96)$ equal to 122.5 (p = .03). Introducing a scale parameter [see Eq. (4)] resulted in a significant (Table III) improvement in fit: $\chi^2(95)$ equal to 108.95 (p = .15). Subsequently the unique variances were free to vary across the sexes. The improvement in fit was again found to be significant: $\chi^2(92)$ equal to 98.5 (p = .30). Finally, the correlation between the environmental factors was estimating allowing for shared environmental influences. The improvement in fit was significant at the .05 level: $\chi^2(91)$ equal to 94.05 (p = .40). The correlation between the twin environments was estimated as, .274 (SE = .11), indicating that about 8% of the environmental variance is shared.

Table III Model Selection Results[a]

	df	χ^2	p
Model (means unconstrained)			
1: $E1$, G, and constrained Θ	96	122.5	.035
2: $E1$, G, constrained Θ, and scale parameter	95	108.9	.155
3: $E1$, G, unconstrained Θ, and scale parameter	92	98.52	.302
4: $E1$, G, unconstrained Θ, scale parameter, and $r(E1,E2)$	91	94.05	.400
1 vs 2: sex difference in scale	1	13.6	<.001
2 vs 3: sex difference in Θ	3	10	<.02
4 vs 3: shared environment	1	4.5	.032
Model (structured means in model 4)			
5 $\Delta E[E]$	117	165.5	.002
6 $\Delta E[G]$	117	120.9	.38
7 $\Delta E[E]$ and $\Delta E[G]$	116	116.3	.47
5 vs 7: $\Delta E[G] = 0$	1	49.2	<.001
6 vs 7: $\Delta E[E] = 0$	1	4.6	.030

[a] Model 1 has a common unshared environmental ($E1$) and additive genetic factor (G) and a diagonal matrix containing unique variances (Θ). All estimated parameters are constrained to be equal across the sexes in model 1.

The parameter estimates for this model (model 4 in Table III) are given in Table IV. Figure 1a depicts the path diagram for the DZOS sample. Using model 4 as a point of departure, the structured means were included. The results are given in Table III. First a model was fit in which both E and G contributed to the difference in phenotypic means between the males and females (model 7). The path diagram for the DZOS sample is given in Fig. 1b. The χ^2 goodness of fit equaled 116.3 (df = 116, p = .47). The parameter estimates are given in Table IV. $\Delta E[G]$ equals 1.147(SE, .23), and $\Delta E[E]$ equals −.652 (SE, .23). The correlation between the estimates of the parameters $\Delta E[G]$ and $\Delta E[E]$ was obtained by requesting the standardized inverse of the information matrix in the LISREL output. The correlation equals $r(\Delta E[G], \Delta E[E])$ = .240, indicating that given the avilable information, the two parameter estimates are fairly independent.

Fixing the factor loadings to those obtained from the analysis without means did not seriously alter the results: $\chi^2(116)$ equal to 120.37 (p = .37), $\Delta E[G]$ = 1.09 (SE, .164), and $\Delta E[E]$ = −.63 (SE, .14).

Table IV. ML Estimates for the Analysis of Blood Pressure with (Model 7) and without (Model 4) Structured Means[a]

	Covariance	
Parameter	No means	With means
λg syst	9.523 (1.13)	8.974 (1.14)
λg diast	2.940 (.75)	2.609 (.90)
λg map	4.822 (.96)	4.829 (.94)
λe syst	6.939 (1.16)	7.184 (1.31)
λe diast	6.323 (.57)	6.592 (.54)
λe map	6.920 (.68)	6.781 (.68)
θf1 unique	23.52 (5.21)	26.98 (5.22)
θf2	5.97 (1.85)	4.81 (1.87)
θf3	7.10 (1.39)	7.49 (1.76)
θm1 unique	26.38 (5.71)	26.65 (5.46)
θm2	13.25 (2.67)	11.12 (2.52)
θm3	2.01 (1.97)	3.55 (1.77)
r(Et1,Et2)	.274 (.111)	.286 (.106)
γ scale	.891 (.072)	.885 (.071)
ν_1		124.63 (1.05)
ν_2		75.02 (.59)
ν_3		89.88 (.71)
$\Delta E[G]$		1.147 (.23)
$\Delta E[E]$		−.652 (.23)
χ^2 (dF)	94.05 (91)	116.28 (116)
p	.40	.47

[a] Standard errors are given in parentheses.

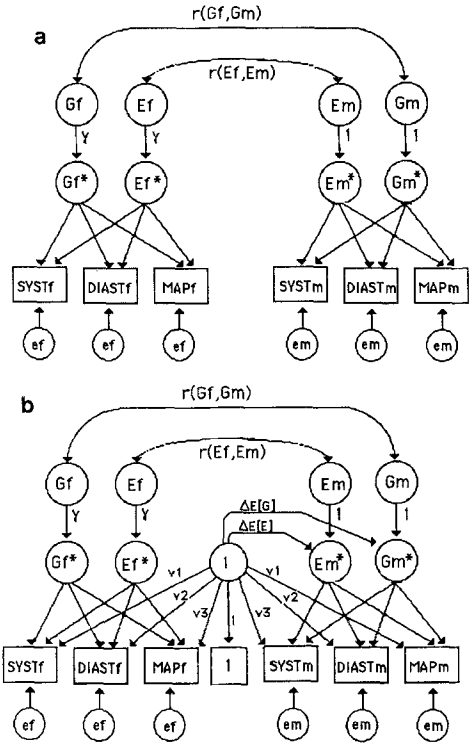

Fig. 1. (a) Twin model for DZOS twin covariance structure including a scale parameter (γ). Factor loadings are not shown. The latent variables G_f^*, E_f^*, etc., are dummy variables with zero variance. (b) Twin model for DZOS twin covariance structure and structured means including a scale parameter (γ), scale constants (ν_1, ν_2, ν_3), and differences in genetic and environmental mean ($\Delta E[G]$ and $\Delta E[E]$). Factor loadings are not shown. The latent variables G_f^*, E_f^*, etc., are dummy variables with zero variance.

Note that, again, we do subtract degrees of freedom for these fixed parameters.

Fitting the model with $\Delta E[E]$ fixed to equal zero (model 5), yielded a $\chi^2(117)$ equal to 120.9 ($p = .38$). The difference is $\chi^2(1)$ equals 4.6 ($p = .04$), but the genetic factor loadings were found to deviate wildly from the values obtained in the analysis without structured means: 4.9, $-.6$, and 1.4, compared to 9.523, 2.940, and 4.822 (as shown in Table IV). Regardless of the significance of the $\Delta E[E]$, instability of this magnitude should rouse suspicion regarding the adequacy of the model.

Fixing $\Delta E[G]$ to equal zero (model 6) resulted in $\chi^2(117) = 165.5$ ($p = .002$). Again, the environmental factor loadings deviated consid-

erably from the previously reported values: 10.4, 4.2, and 6.5, compared to 6.939, 6.323, and 6.920 (as shown in Table IV).

These illustrative results suggest that compared to the female sample the genetic distribution underlying the three measures of blood pressure in the male sample is characterized by a larger mean and the environmental distribution by a lower mean.

DISCUSSION

In the present paper we have used Sörbom's method (1974) to study differences in latent means in multivariate twin data. By restricting the analysis to a comparison between groups, the results pertain only to the additive contributions of common genetic and environmental factors to the deviation of the group means from what can be considered a vector-valued grand mean.

Assumptions made in the decomposition of the phenotypic means relate to the constitution of the phenotypic means and to the equality of the variances in each group. The latter assumption is testable in the standard analysis of covariance structure by means of equality constraints on the relevant parameters across the groups; the former has been shown to be testable in the illustration involving simulated data.

It may be noted that these assumptions mentioned are in fact commonly made in the standard MANOVA models for the analyses of differences in means. However, no distinction is made in the MANOVA approach between within-group true score and error variance so that both contribute to the within sums of squares (error) matrix. Generally, an important aspect of Sörbom's (1974) models incorporating structured means is the increase in statistical power afforded by the ability to distinguish between true score variance and (possibly heterogeneous) error variance. A failure to reject the null hypothesis of equal means by means of MANOVA cannot therefore be taken to be proof positive for the absence of differences in means as modelled by means of Sörbom's approach.

Although we have considered gender effects in twin data, other group comparisons are feasible. For instance, a parent–offspring comparison may be carried out. It is then necessary to estimate the latent contrasts in the offspring group, because of the regressions of the parent additive genetic factors on the offspring additive genetic factors. An estimated additive genetic contrast in the parent groups would contribute to the offspring means via the genetic path, thus spoiling the contrast between the latent parent and the latent offspring means. Another pos-

sibility is the comparison of a genetically informative sample (e.g., twins) over time by defining group membership by measurement occasion.

The requirement of suitable multivariate data may hamper the application of this approach to the decomposition of phenotypic means. However, in any situation where there are multiple indicators of a latent (phenotypic) construct (e.g., questionnaire items), multivariate data can be created by judicious combination of the indicators. Simulations in which three subscores were created from a pool of six items has shown that this approach is feasible. On the other hand, one might consider modeling the indicators themselves instead of their aggregates. In this case the psychometric model proposed by McArdle and Goldsmith (1984) could be used (for an application see Heath *et al.*, 1989)

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REFERENCES

Boomsma, D. I., and Molenaar, P. C. M. (1987). The genetic analysis of repeated measures. I. Simplex models. *Behav. Genet.* **17**:111–123.

Boomsma, D. I., Molenaar, P. C. M., and Orlebeke, J. F. (1990). Estimation of individual genetic and environmental factor scores. *Genet. Epidemiol.* **7**:83–91.

Browne, M. W. (1984). Asymptotically distribution-free methods for the analysis of covariance structures. *Br. J. Math. Stat. Psychol.* **37**:62–83.

Dolan, C. V., Molenaar, P. C. M., and Boomsma, D. I. (1989). LISREL analysis of twin data with structured means. *Behav. Genet.* **17**:51–62.

Dolan, C. V., Molenaar, P. C. M., and Boomsma, D. I. (1991). Simultaneous genetic analysis of longitudinal means and covariance structure in the simplex model using twin data. *Behav. Genet.* **21**:49–65.

Eaves, L. J. Long, J., and Heath, A. C. (1986). A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behav. Genet.* **16**:143–162.

Grossman, M., and Bohren, B. B. (1985). Logistic growth curve of chickens: Heritability of parameters. *J. of Hered.* **76**:459–462.

Heath, A. C., Eaves, L. J., and Martin, N. G. (1989). The genetic structure of personality. III. Multivariate genetic item analysis of the EPQ scales. *Person. Individ. Diff.* **10**(8):877–888.

Heath, A. C., Neale, M. C., Hewitt, J. K., Eaves, L. J., and Fulker, D. W., (1989). Testing structural equation models for twin data using LISREL. *Behav. Gen.* **19**:9–38.

Hewitt, J. K. (1990). Changes in genetic control learning, development, and aging. In Hahn, M. E., Hewitt, J. K., Henderson, N. D., and Benno, R. H. (eds.), *Developmental Behavior Genetics: Neural, Biometrical and Evolutionary Approaches*, Oxford University Press, Oxford.

Hewitt, J. K., Eaves, L. J., Mosteller, M., and Schieken, R. (1989). Can increases in mean heart rate during aerobic exercise be accounted for by scalar increments in the effects of genes that control individual differences. *Behav. Genet.* **19**:763.

Jöreskog, K. G. and Sörbom, D. (1986). *PreLis: A Preprocessor for LISREL*, Scientific Software, Mooresville, USA.
Jöreskog, K. G., and Sörbom, D. (1988). *LISREL VII: A Guide to the Program and Applications*, SPSS, Chicago.
Mardia, K. V. (1980). Tests of univarite and multivariate normality. In Krishnaiah, P. R. (ed.), *Handbook of Statistics, Vol. 1*, North-Holland Publishing Company, Amsterdam, pp. 279–320.
Martin, N. G., and Eaves, L. J. (1977). The genetical analysis of covariance structures. *Heredity* **38**:79–95.
Mather, K., and Jinks, J. L. (1977). *Introduction to Biometrical Genetics*, Chapman and Hall, London.
McArdle, J. J. (1986). Latent variable growth within behavior genetic models. *Behav. Genet.* **16**:163–200.
McArdle, J. J., and Goldsmith, H. H. (1984). Structural equation modeling applied to the twin design: Comparative multivariate models of the WAIS. *Behav. Genet.* **14**:609.
Muthén, B. (1989). Multi-group structural modelling with non-normal continuous variables. *Br. J. Math. Stat. Psychol.* **42**:55–62.
Royston, J. P. (1985). The distribution of skewness and kurtosis. *Appl. Stat.* **34**(1):87–94.
Sörbom, D. (1974). A general method for studying differences in factor means and factor structure between groups. *Br. J. Math. Stat. Psychol.* **27**:229–239.
Sörbom, D. (1976). A statistical model for the measurement of change in true scores. In de Gruyter, D. N. M., and van der Kamp, L. J. T. (eds.), *Advances in Psychological and Educational Measurement*, J. Wiley and Sons, New York.
Sörbom, D. (1978). An alternative to the methodology for analysis of covariance. *Psychometrica* **43**:381–396.
Vandenberg, S. G., and Falkner, F. (1965). Hereditary factors in human growth. *Hum. Biol.* **37**:357–365.

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