Longitudinal Genetic Analysis of Childhood IQ in 6- and 7-year-old Russian Twins

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Using a longitudinal twin study of Moscow children, we have studied the development of psychometric intelligence during the transition from preschool (age 6) to school (age 7). Children were tested using the Wechsler Intelligence Scale for Children (WISC). Simplex models were applied to explore the relationship of different sources of phenotypic variance. The following sources of variation were considered: genetic effects, common or shared family environment and unique environment. At age 6, genetic influences were much greater than those of shared environment but the magnitude of genetic influences decreased and the magnitude of shared environment influences increased substantially by age 7.

Having provided strong evidence for a substantial genetic contribution to individual differences in general cognitive ability (Bouchard & McGue, 1981; Plomin et al., 2000), in the last two decades behavior genetics research has shifted its focus towards the developmental course of genetic-environmental influences on intelligence and cognitive abilities. Most studies investigating age differences in genetic and environmental contributions to variance in cognitive performance are cross-sectional. Summaries of cross-sectional studies of intellectual performance (McCartney et al., 1990; McGue et al., 1993) indicated that the heritability of general cognitive ability may increase from 40% in childhood to as high as 80% in adulthood, and the effects of shared family environment may decrease to negligible levels by adolescence. This finding was supposed to be a result of amplification of genetic effects existing early in development (Plomin, 1986). However, the most appropriate way to explore the developmental changes and the nature of these changes is a longitudinal study. Only longitudinal studies can disentangle age from cohort effects and, what is even more important, only longitudinal studies can reveal how genes and environment operate throughout development. Unfortunately, this type of research is sparse, thus allowing us to draw only preliminary conclusions concerning the age dynamics of genotype-environmental influences underlying individual differences in cognitive abilities.

With the exception of the early longitudinal adoption study by Skodak and Skeels (1949), the Louisville Twin Study (LTS; e.g., Matheny, 1990; Wilson, 1974; Wilson, 1983; Wilson & Harpring, 1972) and the Colorado Adoption Project (CAP; e.g., Baker et al., 1983; DeFries et al., 1987; LaBuda et al., 1986; Plomin et al., 1997) have been the primary long-term behavioral genetics studies using a longitudinal design. Evidence from these studies has suggested that the effects of genetic and non-shared environment on IQ measures increase during childhood through to adolescence to adulthood, while the importance of shared environment decreases.

The LTS followed twins from birth to age 15 years with a follow-up visit between ages 21 to 25 and found generally increasing heritability for cognitive performance, usually resulting from increasing MZ twin similarity as subjects aged (Matheny, 1990; Wilson, 1983). A comparison of children's intellectual development profiles showed a similar picture. The application of a general developmental model to the longitudinal IQ data from the LTS demonstrated the persisting and accumulating effects of a single set of genes (Eaves et al., 1986). The shared environmental effects were persistent, but showed age-specific input as well, while non-shared environmental effects were more occasion-specific and less persistent. Heritability has also been found to increase from 7 to 9 months in the Twin Infant Project (Bishop et al., 1991; DiLalla & Fulker, 1989), although reports from the MacArthur Longitudinal Twin Study suggested no increasing heritability across ages studied (14, 20 and 24 months) and substantial continuity of genetic influences on general cognitive ability from 14 to 24 months of age; in addition, significant new genetic variance in cognitive ability appeared at 24 months (Cherny et al., 1994; Plomin et al., 1993).

The most recent report from the CAP (Plomin et al., 1997) provided parent-offspring correlations from age 1 to 12 and at age 16, and found that, over time, the adopted children showed less and less resemblance to their adoptive parents in terms of IQ and increasing resemblance to their biological parents. The near-zero parent–sibling correlations for adoptive parent and adopted children suggested that shared environment did not contributed importantly to parent-offspring resemblance. Similar trends were identified in the Texas Adoption Study that found a significant contribution of shared environment to IQ prediction at the first test (between ages 3 and 14), but not 10 years later (Loehlin et al., 1989). Heritability slightly increased over the course of 10 years.

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No increasing genetic influences across ages were found in two analyses of a large data set combined from several major twin and adoption projects (Cardon et al., 1992; Fulker et al., 1993). The analysis of heritability showed that it was fairly constant across the ages included in the studies (i.e.,, from age 1 to age 9). Genetic forces appeared to be the major contributor to change as well as continuity in general cognitive ability across development. Early genetic effects persisted through all ages tested as well as the new genetic variation, which appeared at around age 7 and persisted through the last age tested, age 9. In contrast, the effect of shared environment was significant but relatively constant and unchanging across development, while nonshared environment was found to be totally time specific, having no continuous effect across development.

Consistent results have been obtained in the twin studies conducted on European samples. In the Czech Longitudinal Study twins were tested yearly and the MZ twin siblings' resemblance slightly exceeded the DZ twin resemblance before the age of 4. After the age of 4 the DZ twin siblings' resemblance decreased, thus leading to an increase in heritability, which constituted more than 0.50 at ages 6 and 7 (Drabkova, 1992). The longitudinal study of 192 pairs of Dutch twins found increased importance of genetic factors from age 5 to age 7 (Boomsma & Van Baal, 1998). The covariance in IQ between ages 5 and 7 was mainly explained by stable genetic factors and was to a lesser extent explained by the stability of the shared environment.

Overall, despite some contradictory results, evidence from the vast majority of twin (e.g., Wilson, 1983) and adoption studies (e.g., Loehlin et al., 1989; Plomin et al., 1997) has suggested that the effects of genetic and nonshared environment on IQ measures increase during childhood and adolescence, while the importance of shared environment decreases. One should keep in mind that this conclusion was derived from genetic research of cognitive abilities that was conducted on American and European samples. Meanwhile, the balance of genetic and environmental influences found in any one population is specific to that population. The genetic control of a particular behavior depends on the population genetic structure (Krushinsky, 1977), thus IQ heritability can be different in different human populations. Therefore, the findings obtained on a particular population cannot be generalized to other ones. To check the universality (or uniqueness) of the identified developmental trends, similar studies employing samples from different populations need to be carried out.

To date, no longitudinal study examining cognitive abilities in the Russian population of twins has been conducted. The only study to investigate the developmental changes in heritability of general cognitive ability used a cross-sectional design (Kantonistowa, 1978). The author examined a sample of 61 MZ and 57 DZ twin pairs aged 7–16 years. The results suggested an increase in shared environmental influences underlying individual differences in IQ across age cohorts studied (Kantonistowa, 1978). The genetic influences increased from 58% to 67% between ages 7–10 and 11–13 and decreased to 25% at ages 14–16 (Kantonistowa, 1978). However, the cross-sectional design of these studies does not allow concluding whether the suggested dynamic in heritability estimates was due to an age-related change, or was associated with cohort effects. Therefore, the results obtained in these studies are rather ambiguous, and probably reflect the small sample size used.

Obviously, Russian culture differs much from American and European cultures, although the educational level of people from Russia, America and Europe is rather similar. This fact allows us to compare the genetic and environmental sources of cognitive abilities between two populations.

We administered the same IQ test, at ages 6 and 7, to a sample of Russian twins. Our study had two main objectives. First, we aimed to analyze the origins of individual differences in IQ as well as the origins of change and continuity in the course of development in Russian children. Second, we aimed to study the genetic control of child psychometric intelligence during the transition from preschool to school years, between ages 6 and 7, in a period of significant environmental changes, and to examine the impact of the strong shared environmental factor related to the start of school education.

Method

The sample included 32 MZ and 29 DZ twin pairs that were tested at the age of 6 years (M = 98.2, SD = 10.3) and again at the age of 7 years (M = 103.2, SD = 12.7). All the children entered school *after* the first testing and were in first grade at the time of the second testing. The sample was ascertained through voluntary participation of individuals from a city-wide, population-based twin registry maintained by the Developmental Behavior Genetic Laboratory of the Institute of Psychology of the Russian Academy of Education. All twin pairs included in this analysis were raised in Russian-speaking, predominantly middle-class homes. Zygosity determination was based upon a combination of the information obtained from a Russian translation of a twin diagnostic questionnaire (Cohen et al., 1973) based upon reported physical similarity and degree of mistaken identity at the time of assessment and a direct examination of physical similarity. Only twin pairs in which zygosity was assigned with confidence were included in the analysis. All pairs were same sex twins.

Children were tested with a Russian adaptation of the Wechsler Intelligence Scale for Children (WISC; Wechsler, 1949)[1]. This instrument is used to assess the cognitive abilities of children aged 5–16 years and consists of 12 subtests. Six subtests (information, comprehension, arithmetic, similarities, vocabulary, digit span) provide scores for the verbal scale and six subtests (picture completion, picture arrangement, block design, object assembly, digit symbol, labyrinths) provide scores for the performance scale. Three total indices of verbal (VIQ), performance (PIQ) and general (GIQ) intelligence are calculated.

Genetic Analysis

Models were fitted to the IQ data obtained at ages 6 and 7. The following sources of variation were considered: genetic effects (G), common or shared family environment (C) and unique environment (E). Two sets of latent factors consisting of these three influences were specified for the discussed ages. For each age, a measured phenotype (IQ) was considered as a

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linear function of three aforementioned latent factors (univariate factor model).

Following Boomsma et al. (1989), the relationships of latent factors were expressed by the first-order auto-regression equations:

$$G_2 = \beta_g G_1 + Z_g,$$
$$C_2 = \beta_c C_1 + Z_c,$$
$$E_2 = \beta_e E_1 + Z_e,$$

where β_g, β_c, β_e were the regressions of latent variables at age 7 on the corresponding variables at age 6, Z_g, Z_c, Z_e represented random input terms (innovations) that were uncorrelated with the corresponding factors G_1, C_1, E_1 (see also Figure 1). The employed patterns are kinds of structures that are called simplex models after Guttman (1954).

The correlation between additive genetic factors for MZ twins is 1 and for DZ twins is 0.5. For both types of twins the correlation between their common environmental factors is 1 (i.e., it was supposed that they were reared in the same family). Unique environment for different twins does not correlate by definition. The covariances between IQ scores for ages 6 and 7 were modeled with the aid of latent factors.

Model parameters of interest were determined using the maximum likelihood method (see Appendix). To obtain the necessary number of statistics, characteristics of interest were estimated for both monozygotic (MZ) and dizygotic (DZ) twin pairs (each twin type had its own expected and observed covariance matrices of dimension 4 by 4). The sum of corresponding fitting functions for MZ and DZ pairs was used as the criterion to be minimized. To estimate parameters of best-fitted models, the authors used a macro written for Microsoft Excel, which implemented the procedure of numerical multivariate non-linear optimization called Generalized Reduced Gradient.

Results

Descriptive statistics for the GIQ data are presented in Table 1. There were no significant differences in GIQ mean and variance between MZ and DZ twins. However, the effect of age on average GIQ and variance in GIQ scores was revealed. There was a significant difference in GIQ mean (t = 3.324, p = .01) and variance (F = 0.656, p = .01) between 6- to and 7-year-old twins.

Results of model fitting for general intelligence scores are presented in Table 2. The first (saturated) model includes the following parameters: genetic effects (G_1 and G_2), common or shared environment (C_1 and C_2), and unique environment (E_1 and E_2). The covariance between GIQ scores at ages 6 and 7 was modeled by latent G, C and E factors that influenced GIQ at both age 6 and age 7. A second set of latent factors was considered for age-specific genetic and environmental effects at age 7. The saturated model fits the observed data well enough (χ^2_{11} = 10.36, p = 0.498).

After analyzing the saturated model, components C and G were omitted sequentially. Leaving out C (model 2) implies that resemblance in pairs is entirely caused by shared genes. Leaving out G takes place in model 3 in which all resemblance between twins can be attributed to the common family environment shared by them. Models 2 and 3 yield essentially worse goodness-of-fit measures than the saturated one ($\Delta\chi^2_3$ = 10.625, p = 0.014 and $\Delta\chi^2_3$ = 11.329, p = 0.010, correspondingly).

These data indicate that both genetic and common environment factors are important in GIQ scores. At the following

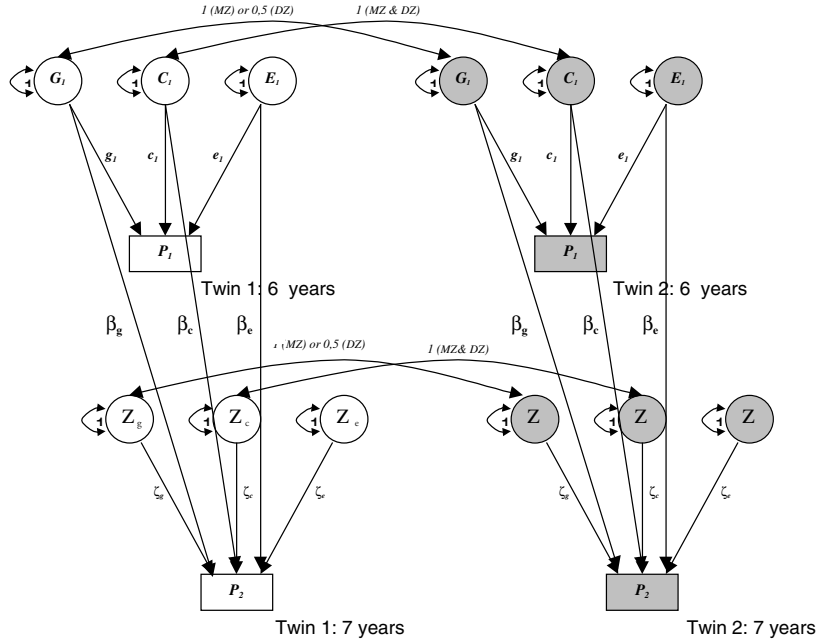

Figure 1

Path diagram representing the simplex model for IQ longitudinal study (6 and 7 years).

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Table 1

Descriptive Statistics for GIQ

	Mean		Variance	
Age	MZ	DZ	MZ	DZ
6 years	100.02	96.24	105.75	100.22
7 years	104.42	101.79	170.67	150.24

Table 2

Model Fitting of Twin GIQ Data Measured at Ages 6 and 7

No	Model	χ^2	*df*	*p*-value	AIC	$\Delta\chi^2$ (comp. with model 1)	Δdf	*p*-value
1	Saturated model	10.363	11	0.498	–11.637	—	—	—
2	GE-model (only heritability and unique environment)	20.988	14	0.102	–7.012	10.625	3	0.014*
3	CE-model (only common and unique environment)	21.693	14	0.085	–6.307	11.329	3	0.010*
4	Reduced model (no heritability innovation)	10.382	12	0.583	–13.618	0.019	1	1.000
5	Reduced model (no common environment innovation)	11.898	12	0.454	–12.102	1.534	1	0.215
6	Reduced model (no G covariance of ages 6, 7)	17.757	12	0.123	–6.243	7.393	1	0.007*
7	Reduced model (no C covariance of ages 6, 7)	13.382	12	0.342	–10.618	3.019	1	0.082
8	Reduced model (no E covariance of ages 6, 7)	10.363	12	0.584	–13.637	0.000	1	1.000
9	Reduced model (no E covariance of ages 6, 7; no heritability and common environment innovations)	12.157	14	0.594	–15.843	1.794	3	0.616

Note: * significant increase in χ^2, implying a significantly worse model fitting.

stage, it was tested if the latent genetic and environment factors that are expressed only at age 7 could be omitted and whether the genetic or environmental covariances could be constrained at zero (i.e., genetic and environmental influences are age specific).

Insignificant increases of χ^2 measures were the cases for the models that have no new genetic (model 4) or new common environment influences (model 5). Thus, new genetic or common environment influences (innovations) were not detected at age 7.

To test whether genetic and environment variables at ages 6 and 7 were independent, models 6 and 7 were analyzed. The genetic and common environment covariances were constrained at zero there. Model fitting showed that corresponding influences, which formed GIQ at age 6, were kept in the main at age 7 (p equals to .007 and .082, correspondingly). The fit of model 8 indicated the unique environment influence might be considered as specific for age ($\Delta\chi^2$ is not significant).

The best fitting model (model 9) suggests that GIQ stability between ages 6 and 7 may be explained by the stability of genetic and common environment factors.

Parameter estimates for model 9 are given in Table 3.[2] It is clear that the heritability contribution in GIQ variance decreases with age (from 78% at age 6 to 19% at age 7), but the common environment contribution increases (7% and 69%, correspondingly). Drop in proportionate heritability estimates results from both reducing genetic variance part and raising the part of common environment variance.

In our sample, the correlation between intelligence scores at ages 6 and 7 was 0.60. In the bivariate model the GIQ covariance was partitioned into a genetic and an environmental part. It turned out that approximately two thirds of the covariance (63%) might be explained by stable genetic influences and another part (37%) by the stability of shared environmental background.

Discussion

The questions addressed in this developmental-genetic analysis concern the origins of individual differences in IQ and the origins of change and continuity between ages 6 and 7 in a Russian twin sample. Regarding the first question, we found essential differences in genetic and shared environmental influences on individual variability in IQ measured at ages 6 and 7 years. At age 6, genetic and shared environment influences determined essentially different amounts of variance in IQ: 78% and 7%, correspondingly. At the age of 7, genetic influences decreased sharply to 19% and shared environmental influences, in contrast, increased to 69%.

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Table 3

Genetic, Common Environmental and Unique Environmental Variances for Ages 6 and 7; Genetic and Common Environmental Covariances for Ages 6 and 7; Standardized Parameter Estimates from Best Fitting Model (GIQ)

	Components			
	G	C	E	Total
Variances and covariances				
Variance — 6 years	78.3	7.3	15.3	100.8
Variance — 7 years	30.3	112.4	20.4	163.2
Covariances for ages 6 and 7	48.7	28.6	—	77.3
Innovations	0.0	0.0	20.4	—
Standardized estimates				
Variance — 6 years	0.78	0.07	0.15	1
Variance — 7 years	0.19	0.69	0.12	1
Covariances for ages 6 and 7	0.63	0.37	—	1
Innovations	0%	0%	100%	—

The data on the origins of change and continuity between ages 6 and 7 revealed that genetic influences persisted between age 6 and age 7, without evidence of new genetic influences being expressed. Their relative and total importance became much smaller. Shared environment factors, which persisted between age 6 and age 7, greatly gained both in relative and total importance during this period.

Speaking of these facts, it is necessary to comment on well-known influence of assortative mating on cognitive variables. Since additive genetic variance arising from this source increases both MZ and DZ covariances to the same extent, this contribution is completely confounded with the effect of shared environment in twin studies. Therefore, great increase of the shared environment part may be partly explained by this factor.

Unique environmental influences did not contribute to the continuity in IQ; they were age specific and accounted for 15% and 12% of total variance in IQ at ages 6 and 7.

Most longitudinal twin studies conducted on American and Western European samples (e.g., Drabkova, 1992; Wilson, 1983) showed greater genetic influence (more than 50%) and less shared environmental influence (10–30%) at age 7 than was found in the present study of Russian twins, and similar influences at age 6. Also, 58.3% of IQ heritability estimate was reported for a group of 7- to 10-year-old Russian twins in Kantonistowa's (1978) study. Moreover, the data that were obtained in our study indicated a developmental trend that differs from that reported in the majority of studies (e.g., DeFries et al., 1987; Loehlin et al., 1989; Plomin et al., 1997; Wilson R., 1983), where an increasing heritability and decreasing shared environmental influences on IQ variability from infancy to adolescence were found. However, in a number of studies, the similar tendency for genetic influences to decrease in the magnitude around age 7 was identified. For example, the analysis of data from the LTS showed that heritability increased monotonously from age 1 through to age 6 (at age 1 — 10%, 2 — 16%, 3 — 18%, 4 — 24%, 5 — 38%, 6 — 54%). At age 7, the genetic influences decreased to 50%, at age 8 and 9 — to 34% and 36% respectively, and at age 15 increased again to 68% (Wilson, 1983). In the analysis of combined data from the CAP, TIP, and MALTS conducted by Cardon and colleagues (1992) the heritability estimates constituted .55 at age 1, .68 at age 2, .59 at age 3, .53 at age 4, and .52 at age 7. The genetic influences' tendency to decrease around age 7 was more pronounced in the other analysis of combined data from the CAP, TIP, and MALTS (Fulker et al., 1993). In this study heritability estimates for the best-fitting model increased from .53 at age 1 to .57 at age 4, then dropped to .43 at age 7 and then again increased to .74 at age 9 (Patrick, 2000).

We found that the magnitude of genetic influences decreased and the magnitude of shared environment influences increased as our subjects aged from age 6 to age 7. From our point of view, these results could be explained by the fact that children in our sample started school at 7 years and this led to an increased influence of shared environment, related to the educational process. The results obtained on American samples also support this hypothesis, since the children who took part in these investigations also started school at age 7. The proposed explanation is also consistent with the results of the study conducted on a Dutch sample (Boomsma & Van Baal, 1998). Heritability estimates obtained in Dutch twins at age 5 were low (27%) comparing to those reported in the majority of studies (Cardon et al., 1992; Fulker et al., 1993; Wilson, 1983) and approached the heritability estimates found in our sample at age 7 (19%). Lower values of heritability estimates (27%), obtained in the Dutch study for 5-year-olds could also be related to the time of starting school education, because in the Netherlands children start to school at the age of 5. At age 7, the data indicated an increase in genetic influences and they reached the magnitude reported in the American samples (50%).

It is possible that the abrupt increase in the diapason of environmental factors directly affecting childrens' cognitive development leads to a decrease in heritability estimates and an increase in shared environment effects. This phenomenon is observed in our sample only in the first schooling year. The more recent data from the Russian longitudinal study suggest that heritability estimates increase to 42% at age 14, and to 57% at age 16 (Malykh et al.,

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2003), thus confirming the general increase in heritability of general cognitive ability across the life-span. Our finding may reflect the transient environmental change and is not inconsistent with the general trend identified in the majority of studies.

The longitudinal behavior genetic design allowed us to assess not only the extent of continuity and developmental change present in the IQ from age 6 to age 7, but also the extent to which the observed continuity and change are because of genetic differences among individuals or differences in the environments in which the children are raised. The analysis revealed that genetic and shared environmental factors significantly contributed to continuity across ages, each explaining about essential parts of the covariance in cognitive performance between ages 6 and 7. Evidence for persistence in genetic influences for general cognitive ability has been provided in the majority of analyses (e.g., Boomsma & Van Baal, 1998; Cherny et al., 1998; Fulker et al., 1993). Although the absence of new genetic variance at age 7 is in parallel with findings from the LTS (Eaves et al., 1986) and the study of Boomsma and Van Baal (1998), these data are not consistent with the results from the CAP indicating genetic change and appearance of new genetic influences around age 7 (Cardon et al., 1992; Fulker et al., 1993). When shared environmental effects have been found in other studies, they also have been shown to be persistent (e.g., Cardon et al., 1992; Cherny et al., 1998; Fulker et al., 1993). It has been suggested that stable features of the home, such as socioeconomic status, may contribute to this effect (Cardon et al., 1992). In our analysis the non-shared environment was found to contribute only to change in IQ across development, the finding consistent with the literature.

Considering some important limitations of the present study, including the small sample size and twins' age range analyzed, our conclusions are inevitably tentative. The future analyses of data from this longitudinal study of Russian twins will help to clarify the roles of genetic and environmental influences on general cognitive ability across development.

Endnotes

1 Adapted and standardized in Russia by A.U. Panasyuk (1973).

2 Estimation of confidence intervals for the parameters presented in Table 3 was carried out using the procedure described by Neale and Miller (1997). The central idea of these calculations was to move a parameter as far away as possible from its estimate at the optimal solution for a given amount of increase in the maximum likelihood fit function (e.g., 3.84 Chi-squared units for 95% confidence intervals), with all the other parameters in a model being still free to vary. As expected, obtained confidence bounds were wide (up to ± 75% of the parameter estimates). As our maximum likelihood fit function is asymptotically distributed as a Chi-square, reliability of the mentioned results is questionable because of small sample sizes. Therefore, confidence bounds are not shown in Table 3.

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Appendix

On the assumption of multivariate normality of observed data, formulation of the maximum likelihood method is equivalent to the optimization problem with the following fitting function to be minimized:

$$F = [\ln |\Sigma| - \ln |S| + tr(S\Sigma^{-1}) - p]\,(N-1)\,,$$

where S — observed covariance matrix,

Σ — expected covariance matrix expressed via free parameters to be estimated,

$|\Sigma|$ and $|S|$ — determinants of matrices Σ and S,

$tr(S\Sigma^{-1})$ — trace of matrix $(S\Sigma^{-1})$,

N — sample size used to calculate S,

p — order of matrices Σ and S.

Minimum of this function, which resulted from optimization problem solution, shows whether the model fits the observed data. Large values correspond to bad fit and small values — to good fit. For sufficiently large samples this goodness-of-fit measure is distributed as a Chi-square (χ^2). The degrees of freedom are calculated as the number of independent observed statistics in all observed matrices minus the number of independent free parameters.

In case of several observed groups (for example, MZ and DZ twins) the global fitting function to be minimized is formed as a sum of partial fitting functions for each group. As these partial functions are proportionate to corresponding sample sizes minus unity, possible inequality of sample sizes for different groups is taken into account in the global sum.

χ^2-statistic is used not only to clarify the model fit but to compare alternative models (viz, a goodness-of-fit measure for the full model may be compared with the corresponding characteristics of reduced models in which some of the parameters to be found are supposed to be equal to zero). Since difference in χ^2-statistics for the full and reduced models is itself distributed as a Chi-square, it may be used to ascertain if the parameters dropped in a model are significant or not.

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