GENETIC AND ENVIRONMENTAL SOURCES OF FAMILIAL TRANSMISSION IN BISCAY FAMILIES. IV. BODY FATNESS INDICATORS

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Abstract: The present research analyses a cross-sectional sample of 307 individuals (91 fathers, 91 mothers, 60 sons and 65 daughters) in 91 nuclear families from the province of Biscay (Basque Country, Spain). The aim was to establish the transmissible and non-transmissible components of six body fatness (quantity and distribution) indicators, which act on familial resemblance. The standardised data of each generation and sex were adjusted to a BETA model of path analysis, independent of sex effects. This methodology permitted a differentiation of the transmissible (genetic and cultural) and non-transmissible (environmental) components acting on the observed phenotypic variance in the Biscay offspring. The findings support the full model of familial transmission for the six studied traits. None of the tested reduced models were considered adequate for any of these traits.

Keywords: Familial transmission; Body fatness; Path analysis; Basque Country.

Introduction

Epidemiological research has demonstrated a close association between fat distribution and the risk of developing several diseases, such as atherosclerosis or non-insulin dependent *Diabetes mellitus*, which are both associated with central fat distribution (Bouchard 1992). Research conducted on familial resemblance for fat distribution, body mass indices, such as BMI and others, together with body circumferences related to body fatness (waist, hip, thigh, calf, arm, etc.), supports a genetic influence on the quantity and especially the distribution of body fat. The striking resemblance of these traits among family members, especially during growth, is very important from an epidemiological point of view, due to the predictable value of these indices for adult pathologies (Tiret et al. 1991).

Changes in the distribution of fat tissue seem to be independent of changes in adiposity levels, suggesting that the pattern of fat distribution develops physiologically. During infancy, and especially during adolescence, genetic factors exert an important influence on the establishing of body fatness patterns (Mueller 1983). However, several reports have been published regarding the relative contribution of environmental factors, particularly those related to socio-economic status and nutritional adequacy (e.g. Bogin and Sullivan 1986). Mueller (1982) has suggested, for instance, that the differences between black and white populations regarding fat distribution patterns could be partly determined by environment. The results of several studies (e.g. Bogin and Sullivan 1986) leave no doubt that environmental factors can alter the fat distribution pattern, even

though the degree to which genetic and environmental factors modify its distribution is still unknown.

Both genetic and environmental factors are transmitted across generations and together with non-transmissible environmental factors, they act on the phenotype of the individual and on the familial resemblance of relatives. For almost three decades, some researchers have been using path analysis techniques, not only to analyse the familial resemblance of individuals, but also to analyse more deeply the genetic and environmental sources of resemblance between relatives for a large number of anthropometric traits. These models of human genetics allow (always within the limits intrinsic to the design of the sample, the chosen model, etc.) an estimation of some parameters of bioanthropological interest, which include the common environment of the individuals, paternal and maternal effects, phenotypic and/or social assortative mating, in addition to the cultural transmission which can influence the phenotypic resemblance of individuals. It should of course be remembered that the results obtained are always specific for the studied population.

The aim of this study was to estimate the transmissible (genetic and cultural) and non-transmissible (non-transmitted environment) components which act on familial resemblance for several body fatness indicators in a sample of nuclear families from the province of Biscay (Basque Country, Spain). We have investigated which model best fitted the transmission of the studied traits from parents to offspring, as well as the relative importance of genetics, together with transmissible and non-transmissible environmental factors, on the inheritance of the studied traits in this population sample.

Material and Methods

The studied population consisted of a cross-sectional sample of 307 individuals (91 fathers, 91 mothers, 60 sons and 65 daughters) in 91 nuclear families from the province of Biscay (Basque Country, Spain). Ages of individuals ranged from 22 to 66 years for fathers, 22 to 58 for mothers, 4 to 22 for sons and 4 to 21 for daughters. The decimal age of each individual was computed as the difference existing between the day of sampling and the day of birth. The triceps, subscapular, suprailiac and calf skinfolds (mm) of each individual were measured according to the International Biological Program criteria (Weiner and Lourie 1981) using a Lange calliper. The sum of these four skinfolds and the Centripetal Fat Ratio (CFR) index (lg subscapular/lg subscapular + lg triceps) were also calculated, and the Kolmogorov-Smirnov test was applied to the variables, though separately for each kind of relative, in order to establish the normality of the sample distribution.

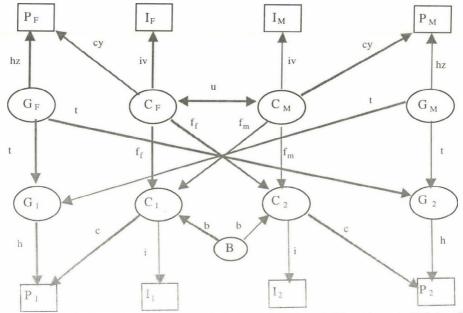
Subjects were asked about the food (solid and/or liquid) which constituted their daily breakfast. Breakfast was defined as any food (solid or liquid) which was consumed between 05.00 h and 09.00 h for adults and between 05.00 h and 10.00 h for children (Aranceta et al. 2000a). Traditionally, in southern European countries such as Spain, the breakfast is not considered an important meal. Moreover, this attitude has become more widespread over recent years, due to the increasing tendency of women to work outside the home. Nevertheless, current nutritional recommendations of the sanitary and educational authorities in Spain continue to emphasize more and more the importance of this first meal of the day (Aranceta et al. 2000a).

In general, it has been shown that breakfast quantity and quality are conditioned by the presence of parents during this meal, a higher socio-economic status and cultural level of the family, greater awareness concerning health matters, and eating breakfast in a relaxed atmosphere (Aranceta et al. 2000b). These factors used to be associated in the past. This observation cannot perhaps be generalised to all populations and has to be analysed as a function of local factors, but in the particular case of the Spanish population, and surely of the present Basque situation, breakfast can be considered as a good environmental index. People who typically had breakfast, but for different reasons had not eaten anything on the morning of the interview, were registered as having their usual breakfast. The "no breakfast" answer was only considered for those people who usually did not intake any food during the first hours of the morning or even until lunchtime.

In order to compare different kinds of relatives and to avoid the eventual effects of secular trends on the studied traits, the standardisation of data was performed separately for each generation and sex. For each individual, normalised values (Standard Deviation Scores or SDS) were computed by using the LMS method (Cole 1988). The individual SDSs were used for the calculation of all correlation coefficients, as well as for path analysis. Despite the broad range of ages between and within generations, data standardisation gave the same biological meaning to adiposity either between or within generations. A multiple correspondence analysis, which is able to analyse categorical or ordinal variables, was used in order to build up the environmental index, which was subsequently introduced in the path analysis. The data to which this analysis was applied were the answers obtained from the questionnaire concerning food ingested during daily breakfast. The obtained scores of the objects represented the individual data of the consumption of food during breakfast and constituted the environmental index for path analysis.

In order to ascertain the genetic and environmental sources of familial resemblance, the path analysis method (type BETA) was employed. The statistical method of analysis used in this study, named PATHMIX, allows the calculation of the correlations existing within nuclear families and/or the adjusting of path analysis models to familial data. (Cloninger et al. 1979a,b). The PATHMIX method offers the advantage of preventing families with a higher number of children from introducing proportionally larger information. More details on this method can be found elsewhere (Rao et al. 1984, Salces et al. 2003).

Based on previous experience with the PATHMIX program of both our team (Salces et al. 2003) and the developers of the program (Rao et al. 1984), we chose a special case of the full lineal model in this analysis. This reduced model consists of 8 parameters (Table 1), fixing at value 0 the rest of the parameters in Figure 1. Familial environmental features are assumed to be the only source of correlation between phenotype and index. Besides the full model, several additional reduced models were also tested, as shown in Table 2.



Abbreviations: B, non-transmissible socio-cultural factors; C, transmissible environment; F, father; G, genotype; I, index; M, mother; P, phenotype; I and 2, offspring. For parameters see Table 1.

Figure 1: The BETA model of path analysis (modified from Rao et al. 1984) used for the analysis of the Biscay familial sample.

Table 1. Parameters estimated by the PATHMIX method of path analysis.

| Parameter | Definition | | | | | | | | |
|-----------|---|--|--|--|--|--|--|--|--|
| h | Effect of childhood genotype on childhood phenotype | | | | | | | | |
| Z | (h , z)=effect of adulthood genotype on adulthood phenotype | | | | | | | | |
| u | Correlation between parental adulthood transmissible environment through social homogamy | | | | | | | | |
| ff | Effect of paternal transmissible environment on child environment, both measured simultaneously | | | | | | | | |
| fm | Effect of maternal transmissible environment on child environment, both measured simultaneously | | | | | | | | |
| b | Effect of non-transmitted common sibship environment on the child environment | | | | | | | | |
| i | Effect of childhood transmissible environment on the child index | | | | | | | | |
| V | (i . v)=effect of adulthood transmissible environment on the adult index | | | | | | | | |

Table 2. Summary of the tested models of familial transmission, the number of fixed parameters in each model and the corresponding number of degrees of freedom in the likelihood ratio test.

| Model of transmission | Hypothesis | d.f. |
|--------------------------|------------|------|
| Full model | | 6 |
| No social homogamy | u=0 | 1 |
| No cultural transmission | b=0 | 1 |
| No genetic effects | h=z=0 | 2 |
| No environmental effects | i=v=1 | 2 |

For parameters, see Table 1.

Results

In Table 3, familial correlations together with the corresponding sample sizes and their standard errors are displayed.

Table 3. Estimation by maximum likelihood of the familial correlations (r) for the three considered traits, the corresponding sample sizes (n) and the standard errors of the correlations (se).

| Service . | $P_F - P_M (n=91)$ | | P_F-P_C | (n=91) | P_M-P_C (1 | n=91) | $P_{C1}-P_{C2}(n=31)$ | |
|------------------------|--------------------|-------|----------------------|--------|------------------------|-------|------------------------|-------|
| Variables | r | se | r | se | r | se | Г | se |
| lg triceps skinfold | 0.037 | 0.105 | 0.105 | 0.096 | 0.208* | 0.092 | 0.421** | 0.121 |
| lg subcapular skinfold | 0.071 | 0.103 | 0.280** | 0.087 | 0.334*** | 0.085 | 0.528*** | 0.107 |
| lg suprailiac skinfold | 0.094 | 0.103 | 0.155 | 0.090 | 0.311*** | 0.085 | 0.298* | 0.135 |
| lg calf skinfold | 0.046 | 0.104 | 0.098 | 0.098 | 0.178 | 0.097 | 0.560*** | 0.110 |
| lg sum of 4 skinfolds | 0.078 | 0.105 | 0.124 | 0.096 | 0.345*** | 0.086 | 0.497*** | 0.117 |
| CFR | 0.160 | 0.101 | 0.281** | 0.086 | 0.030 | 0.094 | 0.176 | 0.156 |
| | $I_F - I_M (n=91)$ | | $I_F - I_C (n=91)$ | | $I_{M}-I_{C} (n=91)$ | | $I_{C1}-I_{C2} (n=91)$ | |
| | r | se | г | se | r | se | r | se |
| lg triceps skinfold | 0.312** | 0.090 | 0.149 | 0.088 | 0.119 | 0.093 | 0.082 | 0.194 |
| lg subcapular skinfold | 0.315** | 0.090 | 0.168 | 0.087 | 0.119 | 0.093 | 0.098 | 0.193 |
| lg suprailiac skinfold | 0.322*** | 0.089 | 0.161 | 0.087 | 0.125 | 0.093 | 0.087 | 0.190 |
| lg calf skinfold | 0.317*** | 0.090 | 0.168 | 0.085 | 0.116 | 0.092 | 0.053 | 0.204 |
| lg sum of 4 skinfolds | 0.322*** | 0.090 | 0.151 | 0.088 | 0.120 | 0.094 | 0.077 | 0.194 |
| CFR | 0.312** | 0.090 | 0.141 | 0.092 | 0.124 | 0.094 | 0.097 | 0.194 |
| | $P_F - I_M (n=91)$ | | $P_{F}-I_{C}$ (n=91) | | $I_F - P_C (n=91)$ | | $P_{M}-I_{C}(n=91)$ | |
| | Г | se | Г | se | r | se | r | se |
| lg triceps skinfold | -0.075 | 0.074 | -0.087 | 0.089 | -0.133 | 0.094 | -0.065 | 0.091 |
| lg subcapular skinfold | -0.029 | 0.075 | -0.062 | 0.088 | -0.155 | 0.092 | -0.047 | 0.09 |
| lg suprailiac skinfold | -0.038 | 0.075 | -0.048 | 0.088 | -0.077 | 0.091 | -0.057 | 0.092 |
| lg calf skinfold | -0.045 | 0.075 | -0.101 | 0.089 | -0.098 | 0.097 | 0.068 | 0.093 |
| lg sum of 4 skinfolds | -0.059 | 0.076 | -0.100 | 0.087 | -0.106 | 0.095 | -0.040 | 0.094 |
| CFR | 0.068 | 0.075 | 0.107 | 0.095 | -0.108 | 0.092 | 0.025 | 0.092 |
| | I_M-P_C (1 | 1=91 | $P_{F}-I_{F}(n=91)$ | | $P_{C1}-I_{C2} (n=31)$ | | $P_C - I_C (n=91)$ | |
| | r | se | r | se | r | se | Γ | se |
| lg triceps skinfold | -0.036 | 0.099 | -0.141 | 0.072 | 0.004 | 0.112 | -0.070 | 0.089 |
| lg subcapular skinfold | -0.075 | 0.096 | -0.117 | 0.073 | 0.021 | 0.113 | -0.077 | 0.09 |
| lg suprailiac skinfold | -0.037 | 0.095 | -0.110 | 0.074 | 0.076 | 0.114 | -0.028 | 0.09 |
| lg calf skinfold | -0.195 | 0.096 | -0.092 | 0.074 | -0.012 | 0.119 | -0.038 | 0.09 |
| lg sum of 4 skinfolds | -0.092 | 0.097 | -0.128 | 0.074 | 0.050 | 0.114 | -0.041 | 0.09 |
| CFR | -0.084 | 0.094 | 0.068 | 0.075 | 0.005 | 0.125 | -0.014 | 0.09 |

Statistical significance: *: $p \le 0.05$; **: $p \le 0.01$; ***: $p \le 0.001$. For abbreviations, see Figure 1.

It can be observed that the correlations between mates for the 6 studied traits were relatively low and non-significant. In contrast, phenotypic resemblance was higher between children and their mothers than with their fathers, except in the case of the CFR index. Resemblance between siblings was in general higher than parent-offspring resemblance and with a higher statistical significance, once again, excepting the CFR index.

The results obtained according to the goodness of fit test of the transmission model applied to the studied traits are shown in Table 4.

Table 4. Tested models, tests of hypotheses and estimated parameters under each model of familial transmission for the three considered traits

| | Tests of hypothesis | | | | Parameters | | | | | | | |
|----------------|---------------------|------|----------------|-------|------------|-------|-------|-------|-------|-------|-------|--|
| Variables | Model | d.f. | p ² | h | Z | u | ff | fm | b | i | V | |
| lg triceps | Full | 8 | 7.017 | 0.923 | 0.372 | 0.742 | 0.503 | 0.000 | 0.292 | 0.495 | 1.311 | |
| skinfold | u=0 | 1 | 23.805*** | 0.554 | 0.997 | | 0.187 | 0.217 | 0.525 | 0.000 | 0.836 | |
| | b=0 | 1 | 26.625*** | 0.562 | 0.994 | 0.503 | 0.189 | 0.215 | | 0.000 | 0.847 | |
| | h=z=0 | 2 | 22.496*** | | | 0.397 | 0.162 | 0.079 | 0.226 | 0.939 | 0.947 | |
| | i=v=1 | 2 | 7.017* | 0.923 | 0.372 | 0.312 | 0.137 | 0.077 | 0.227 | | | |
| lg subscapular | Full | 8 | 6.457 | 1.000 | 0.565 | 0.707 | 0.462 | 0.027 | 0.279 | 0.519 | 1.286 | |
| skinfold | u=0 | 1 | 25.794*** | 0.753 | 1.047 | | 0.213 | 0.235 | 0.523 | 0.000 | 0.840 | |
| | b=0 | 1 | 32.904*** | 0.799 | 1.055 | 0.524 | 0.224 | 0.244 | | 0.000 | 0.853 | |
| | h=z=0 | 2 | 38.821** | | | 0.401 | 0.169 | 0.080 | 0.223 | 0.933 | 0.950 | |
| | i=v=1 | 2 | 6.525* | 1.000 | 0.566 | 0.315 | 0.141 | 0.080 | 0.000 | | | |
| lg suprailiac | Full | 8 | 6.107 | 0.777 | 0.688 | 0.517 | 0.181 | 0.091 | 0.214 | 0.894 | 0.883 | |
| skinfold | u=0 | 1 | 20.780*** | 0.612 | 1.022 | | 0.204 | 0.236 | 0.518 | 0.000 | 0.83 | |
| | b=0 | 1 | 21.170*** | 0.634 | 1.025 | 0.526 | 0.217 | 0.245 | | 0.000 | 0.84 | |
| | h=z=0 | 2 | 22.690*** | | | 0.481 | 0.172 | 0.092 | 0.217 | 0.910 | 0.90 | |
| | i=v=1 | 2 | 6.107* | 0.777 | 0.688 | 0.322 | 0.133 | 0.087 | 0.224 | | | |
| lg calf | Full | 8 | 7.448 | 1.000 | 0.276 | 0.736 | 0.372 | 0.000 | 0.244 | 0.690 | 0.94 | |
| skinfold | u=0 | 1 | 27.730*** | 0.573 | 0.998 | | 0.186 | 0.215 | 0.527 | 0.000 | 0.83 | |
| | b=0 | 1 | 30.636*** | 0.601 | 0.999 | 0.514 | 0.185 | 0.213 | | 0.000 | 0.84 | |
| | h=z=0 | 2 | 25.386*** | | | 0.390 | 0.170 | 0.083 | 0.253 | 0.923 | 0.97 | |
| | i=v=1 | 2 | 7.546* | 1.000 | 0.276 | 0.317 | 0.142 | 0.081 | 0.000 | | | |
| lg Sum 4 | Full | 8 | 8.773 | 0.998 | 0.431 | 0.818 | 0.459 | 0.000 | 0.000 | 0.548 | 1.14 | |
| skinfolds | u=0 | 1 | 26.298*** | 0.659 | 1.023 | | 0.187 | 0.241 | 0.530 | 0.000 | 0.83 | |
| | b=0 | 1 | 29.032*** | 0.686 | 1.025 | 0.517 | 0.197 | 0.244 | | 0.000 | 0.84 | |
| | h=z=0 | 2 | 32.105*** | | | 0.465 | 0.163 | 0.090 | 0.227 | 0.919 | 0.90 | |
| | i=v=1 | 2 | 8.836* | 0.998 | 0.431 | 0.322 | 0.129 | 0.087 | 0.000 | | | |
| Centripetal | Full | 8 | 10.526 | 0.640 | 0.640 | 0.458 | 0.180 | 0.078 | 0.223 | 0.906 | 0.91 | |
| Fat Ratio | u=() | Ī | 23.888*** | 0.424 | 0.973 | | 0.249 | 0.159 | 0.503 | 0.000 | 0.86 | |
| (CFR) | b=0 | 1 | 18.424*** | 0.234 | 0.000 | 0.412 | 0.907 | 0.000 | 0.000 | 0.102 | 0.68 | |
| (~) | h=z=0 | 2 | 16.013*** | | | 0.397 | 0.162 | 0.079 | 0.226 | 0.939 | 0.94 | |
| | i=v=1 | 2 | 10.526** | 0.640 | 0.640 | 0.312 | 0.137 | 0.077 | 0.227 | | | |

(d.f.=degrees of freedom; *: $p \le 0.05$; **: $p \le 0.01$; ***: $p \le 0.001$).

For parameters see Figure 1. For hypotheses, see Table 2.

The full model of transmission was accepted and the other four specific more reduced models, where four different hypotheses were introduced, were also tested. The estimations of the eight path coefficients obtained, both in the full and in the more reduced models of transmission also appear in Table 4. The four reduced models were

rejected for all traits. Consequently, the full model of familial transmission constituted the most parsimonious model for the transmission of the studied traits.

The estimations of the h parameter for skinfolds ranged between 0.64 (CFR index) and 1.00 (subscapular and calf skinfolds) under the full model of familial transmission. Regarding cultural transmissible environments from the father and the mother (ff and fm, respectively), evidence of a clear preponderance of the paternal component was observed for all traits when the full model of transmission was applied (Table 4). However, evidence of this cultural paternal influence on phenotype was not apparent in the phenotypic correlations between each parent and the children (Table 3), since the resemblance was higher between mother-child than between father-child, with the exception of the CFR index.

Discussion

The aim of the present study was to quantify the genetic and environmental sources of variation acting on the familial transmission of a series of body fatness and distribution indicators with multifactorial inheritance. Data were fitted to a general lineal model based on that proposed by Cloninger et al. (1979a,b). Comparison of the results of the present study with those of other reports in the literature is a difficult task for two main reasons. Firstly, studies using a BETA model of familial transmission of anthropometric traits are scarce. In contrast, this model has been frequently used in studies of variables such as the Intelligence Quotient (IQ, Cloninger et al. 1979a), schizophrenia (Rao et al. 1981) or some variables of physical fitness (Pérusse et al. 1987). Moreover, researchers traditionally tend to use the TAU model of familial transmission, despite the fact that it displays less information than the BETA model, which was used here (i.e. Devor et al. 1986). Secondly, estimations of transmissible components are specific for each population (Pérusse et al. 1987) and are thus strictly speaking, not comparable.

The full model of familial transmission was accepted for all traits. We also tested four reduced models of transmission, but all of these were rejected for the studied variables. The results concerning the rejection of the model without social homogamy between mates confirmed the initial estimations of the socio-economic and cultural level of the parental sample, obtained through a questionnaire and a further categorisation according to the Census of the Bilbao City Council. Generally, phenotypic homogamy and social homogamy used to be associated in such a way that social homogamy is associated with phenotypic assortative mating. In our population, this did not happen since even if social homogamy between mates were observed, no evidence of phenotypic homogamy was found. This may be due to the homogeneity of social level in our sample, in such a way that phenotypic homogamy between mates did not seem to be influenced. Such an influence was found to be low and not statistically significant.

In a previous study of the same population, using a different sample, a high phenotypic similarity of bony traits between mates was shown (Salces et al. 2004); similarity between fatness variables was observed to a different degree. This fact is consistent with data obtained from other western populations, particularly the Spanish sample studied by Sánchez-Andrés (1992). However, other non-western populations, such as the Indian one studied by Byard et al. (1985), showed the existence of a high similarity between mates for adiposity traits, probably due to social homogamy and cohabitation, since diet and common lifestyle were determinant in the observed resemblance for weight

after marriage. The same was not observed in our population. The reduced model of transmission proposing the non-existence of genetic factors on phenotype (h=z=0) was also rejected for the 6 body fatness traits, indicating the importance of genetic factors on the transmission of this kind of variable across generations. In contrast, Bouchard et al. (1988) noted the larger influence of genotype on internal fat, while superficial (subcutaneous) fat was noted to be mostly determined by non-genetic factors.

Traditionally, human genetics does not take into account the relative importance of environment on the transmission of phenotypes of a multifactorial nature. As noted before, this environment can be of two types: a transmitted environment from parents to offspring, and a non-transmitted intra-generational environment, which can affect individuals of the same generation, i.e. siblings. The latter type of environmental influence is frequently omitted from consideration by researchers, while the former is more frequently taken into account in studies of the maternal and/or paternal influences on phenotypes with multifactorial inheritance (Boldsen and Mascie-Taylor 1990). The estimated values of the ff and fm path coefficients in our sample are indicative of the existence of maternal or paternal effects on the transmission of the studied traits. The major part of the cultural transmissible influence for all the variables was clearly derived from the father (ff > fm), according to the full model of transmission. These results support the existence of a paternal effect on the determination of the quantity and distribution of subcutaneous fat. However, the results concerning phenotypic resemblance observed for these variables of subcutaneous tissue, revealed that only correlations for the CFR were higher between offspring and the father than between offspring and the mother. Overall, our results indicate that the quantity of body fat is more influenced by maternal effects, while its anatomic distribution is more affected by paternal influences.

The phenotypes of parents and children are determined not only by genetic factors, but also by transmissible cultural and environmental factors. Parents and children resemble each other because they share several genetic and cultural factors, while some specific environmental factors are independent. A number of authors (e.g. Rao et al. 1976) have noted that cultural factors may act directly on the phenotype of children and, for this reason, they represent them in path diagrams as a path from the phenotype of the father and mother to the index of the child. However, it is unlikely that two parents, with different genetic and cultural values, would have the same cultural influence on their offspring (see Cloninger et al. 1979a). At the same time, this direct path from parental phenotypes to that of the offspring would imply a non-exclusively cultural transmission, as well as the existence of a correlation between non-transmissible environments from parents to offspring. Using the PATHMIX method (Cloninger et al. 1979a,b), we did not find evidence in favour of such an implication, since a BETA model allows specification of the composition of the variance in a population in equilibrium, as if assortative mating were present.

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