Genetic and environmental effects on isometric muscle strength and leg extensor power followed up for three years among older female twins

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Genetic and environmental effects on isometric muscle strength and leg extensor power followed up for three years among older female twins. J Appl Physiol 106: 1604–1610, 2009. First published February 19, 2009; doi:10.1152/japplphysiol.91056.2008.—The purpose of this study was to examine changes in the contribution of genetic and environmental effects to isometric knee extensor strength and leg extensor power among 63- to 76-year-old female twins over a 3-yr follow-up. At baseline in 2000 the sample comprised 206 monozygotic (MZ) and 228 dizygotic (DZ) twin individuals, and at follow-up in 2003 the sample comprised 149 MZ and 164 DZ twin individuals. Genetic modeling showed that genetic effects explained 58% (95% CI: 46–68%) of the variance in muscle strength at baseline and 56% (95% CI: 41–68%) at follow-up, with no occasion-specific genetic effect. Nonshared environmental effects accounted for 42% (95% CI: 32–54%) of the variation at baseline and 15% (95% CI: 7–26%) at follow-up. In addition, new nonshared environmental effects explained the remaining variance, 29% (95% CI: 22–37%) of muscle strength at follow-up. For muscle power, the same genetic effects accounted for 67% (95% CI: 57–74%) of the variation at baseline and 48% (95% CI: 34–61%) at follow-up. Nonshared environmental effects in common at both measurement points explained 33% (95% CI: 25–43%) of the total variation at baseline and 11% (95% CI: 5–21%) at follow-up. The remaining variance of muscle power at follow-up was accounted for by time-specific environmental effects. Results indicated that the contribution of genetic effects to isometric muscle strength was stable, whereas for leg extensor power the proportion of genetic effects decreased during the follow-up. We observed new specific environmental effects underlying follow-up muscle strength and power, which effects could be due to the onset of new disease processes or changes in lifestyle.

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Frederiksen et al. (7), genetic effects accounted for 52% of the variation in handgrip strength and were constant across the age groups. The purpose of the present study was to examine changes in the contribution of genetic and environmental effects to maximal isometric knee extensor strength and leg extensor power among 63- to 76-yr-old female twins over a 3-yr follow-up. On the basis of earlier results we hypothesized that the contribution of genetic effects would decrease and the contribution of environmental effects would increase during the 3-yr follow-up. We also hypothesized that new genetic and environmental effects for muscle strength and power would occur during the follow-up.

MATERIALS AND METHODS

Participants. This study is part of the Finnish Twin Study on Aging (FITSA), a study of genetic and environmental effects on the disablement process in older female twins. A detailed description of the study design and recruitment has been reported elsewhere (33). Briefly, participants were recruited from the Finnish Twin Cohort, which comprises all same-sex twin pairs born before 1958 with both co-twins alive in 1975 (10, 11). An invitation to take part in the present study was sent to 178 monozygotic (MZ) twin pairs, 212 dizygotic (DZ) twin pairs, and 24 twin pairs of previously undetermined zygosity, aged 63–76 yr (Fig. 1). To be recruited for the baseline measurements in the year 2000, both individuals in the pair had to agree to participate. At the follow-up measurements in 2003, we did not require both individuals in the pair to participate. The zygosity of the twins was initially determined in 1975 with a validated questionnaire (28), and in the present study this was confirmed with a battery of 10 highly polymorphic gene markers in DNA extracted from a venous blood sample.

The final sample at the baseline measurements in 2000 comprised 206 MZ and 228 DZ twin individuals, and at the follow-up in 2003 the sample was 149 MZ and 164 DZ twin individuals (Fig. 1). On arrival at the laboratory, the participants provided written informed consent. The baseline and follow-up study protocols were approved by the Ethics Committee of the Central Hospital District of Central Finland.

Measurements. Maximal voluntary isometric knee extensor strength was measured from the side of the dominant hand in a sitting position with an adjustable dynamometer chair (Good Strength, Metitur). The measurement was done at a knee angle of 60° from full extension. The ankle was fastened by a belt to a strain gauge system. After familiarization with the measurement, three to five maximal efforts, each separated by a 1-min rest, were conducted. The data were digitized into newtons (N), recorded, and stored on a computer with the Good Strength software package (Metitur). For each subject the best performance with the highest value was accepted as the result. At our laboratory, the coefficient of variation between two consecutive measurements of isometric knee extensor strength conducted at an interval of 1–2 wk was 6% among older people (24).

Leg extensor power was measured with the Nottingham Leg Extensor Power Rig (2) in an upright sitting position with arms folded across the chest and the active leg toward the push pedal while the free leg rested on the floor. The push pedal was located in front of the seat, which made the direction of movement almost horizontal. The leg on the dominant hand side was measured first, followed by the other leg. The subject was instructed to push the pedal as hard and as fast as possible. Two to three practice trials were allowed for the participants to familiarize themselves with the method. The measurement was repeated until no further improvement occurred, but at least five times. The intertrial rest period was 30 s. For each subject, the highest value was used in the analysis. The results were recorded, computed, and expressed in watts (W) with the Leg Rig software package (PC214E). The coefficient of variation of leg extensor power measured in our laboratory was 8% (33).

Statistical methods. Equality of the means and variances between the MZ and DZ twins and phenotypic correlations between isometric knee extensor strength and leg extensor power in 2000 and 2003 were calculated and tested with Stata statistical software (31).

Intraclass correlation coefficients (ICC) were computed for the MZ and DZ twin pairs separately to estimate the level of within-pair similarity and the ratio of MZ and DZ correlations. The correlation pattern obtained reveals whether genetic or nongenetic familial factors play a role in explaining the variability in the trait. Comparison of the relative sizes of the MZ and DZ ICC at baseline and at follow-up, and of cross-twin cross-trait correlations over time, indicates whether there may be changes in genetic and environmental effects over time. ICC and Pearson’s correlations were computed with SPSS software (30). Comparisons of twin correlations provide initial information regarding the type and magnitude of genetic and environmental variance, and these comparisons were also used as the bases of the genetic modeling.

The mathematical modeling of the data is based on the fact that there are two types of twins. MZ twins share all of their genes (100%), while DZ twins share on average 50% of their segregating genes. Consequently, in DZ twin pairs genetic effects contribute to both similarity and differences, whereas among MZ twin pairs they only contribute to similarity. Greater similarity between MZ twin pairs compared with DZ twin pairs is evidence for genetic influence on the trait (17, 22).

In genetic modeling, the observed variance in a phenotype is decomposed into four sources of variance: additive genetic (A), nonadditive genetic (D), shared environment (C), and nonshared environment (E) (22). The expected correlations for A, D, and C between the twins of a MZ pair are 1.0, while for DZ pairs the expected correlation for A is 0.5, for D 0.25, and for C 1.0. By
definition, E effects are uncorrelated in both MZ and DZ twins, and E also includes random effects. On the basis of these expectations different models (ACE, ADE, AE, CE, and E) are fitted to the data, the aim being to obtain the most parsimonious and best-fitting model to explain the observed pattern of twin similarity in MZ and DZ pairs. D and C cannot be estimated simultaneously when only data on twin pairs raised together are available (17). In the present analyses, D was not included in the models, because the phenotypic correlations between the twin sisters suggested no contribution of D (17).

To evaluate whether two different measurement points (baseline 2000 and follow-up 2003) share a genetic component or whether the genetic effects are measurement point specific, a bivariate Cholesky decomposition model was used. In such an ACE model (Fig. 2), genetic effect A1 is common to both measurement points, while genetic effect A2 loads only on the later measurement point. The shared environmental (C1, C2) and nonshared (E1, E2) environmental effects have similar patterns of loadings. In the present study, the analysis was started with the hypothetical full ACE model including all the plausible parameters. With a bivariate genetic analysis, it is possible to separate new genetic and environmental effects specific for follow-up measurement from effects that are common to both measurement points. To obtain a more parsimonious model, the full model was modified by dropping the weakest (i.e., parameter estimate zero or very small) nonsignificant parameters one at a time, until the model with the best fit was obtained. Biological plausibility was also used as a guideline in selecting the parameters to be dropped (16, 17).

The genetic analyses were carried out by using raw data input with full information maximum likelihood in Mx (16, 17), which permits a guideline in selecting the parameters to be dropped (16, 17).

RESULTS

Descriptive statistics. At the baseline in 2000, the mean (SD) age was 68.3 yr (SD 3.7) for MZ individuals (n = 296) and 68.9 yr (SD 3.1) for DZ individuals (n = 228). The mean weight and height for MZ individuals at the baseline were 69.6 kg (SD 11.8) and 158.0 cm (SD 6.4), respectively. The corresponding values for DZ individuals were 70.6 kg (SD 12.2) and 159.1 cm (SD 5.8), respectively. MZ and DZ twin individuals did not differ statistically significantly in any aspects. The Table 1. When we studied the changes in the entire sample, we observed that the mean isometric knee extensor strength of individuals for whom data were obtained at both measurement points (n = 280) was 301.3 ± 78.2 N (SD) at baseline and 285.8 ± 79.6 N at follow-up 3 yr later. The baseline value for individuals who participated only in the baseline measurement was 271.3 ± 90.4 N (n = 136). The mean leg extensor power of persons with both measurement points (n = 278) was 104.2 ± 32.9 W at baseline and 102.0 ± 30.1 W at follow-up. Individuals who participated only in the baseline measurement had lower leg extensor power (92.3 ± 35.8 W, n = 141) than those who continued in the study. In MZ individuals, the individuals who had results at baseline and follow-up had statistically significantly greater isometric knee extensor strength compared with individuals who had only baseline results (P = 0.02). A statistically significant difference was also seen in leg extensor power between individuals who had baseline and follow-up results compared with individuals who had only baseline leg extensor power results (P = 0.01, Table 1).

Correlations. The test-retest Pearson’s correlation between baseline and follow-up was 0.74 (P < 0.001) for leg extensor power and 0.79 (P < 0.001) for isometric knee extensor strength. For isometric knee extensor strength the MZ and DZ ICC and the ratio of the correlations (R) at baseline (ICCMZ = 0.50, ICCDZ = 0.32, R = 1.56) and at follow-up (ICCMZ = 0.55, ICCDZ = 0.30, R = 1.83; Table 2) were similar, indicating stable genetic effects. For leg extensor power the ratio of the MZ and DZ ICC at follow-up was smaller (R = 0.81) than at baseline (R = 1.45), indicating a possible decline in the influence of genetic effects on the phenotype. In addition, we tested the correlation between changes in both muscle phenotypes over time. For the total sample the correlation between change in both muscle phenotypes over time was r = 0.194, for MZ twin individuals r = 0.190, and for DZ twin individuals r = 0.168.

Genetic modeling. The genetic modeling was started with the full ACE model, in which all the parameters were included. To obtain a more parsimonious model, the full model was modified by dropping the weakest (i.e., parameter estimate zero or very small) nonsignificant parameters one at a time, until the model with the best fit was obtained. In the final reduced Cholesky decomposition model for isometric knee extensor strength, 58% (95% CI: 46–68%) of the variance at baseline was explained by genetic factors (Fig. 3). Three years later the corresponding value was 56% (95% CI: 41–68%), with no occasion-specific genetic effect. Nonshared environmental effects in common for both measurement points accounted for 42% (95% CI: 32–54%) of the variation at baseline and 15% (95% CI: 7–26%) at follow-up. In addition, new nonshared environmental effects, specific to the follow-up, explained the remaining variance, 29% (95% CI: 22–37%) of muscle strength in 2003. The nonshared environmental effects contain the measurement error and are thus always included in models. This final reduced ACE model fitted the isometric knee extensor strength data well (−2LL = 7871.86, df = 694, Akaike’s information criterion (AIC) = 6,483.86, P value of the χ² difference between the models = 0.990; Table 3). The genetic correlation between baseline and follow-up measurement in isometric knee extensor strength was 1.0 (95% CI:
The environmental correlation between the two time points was 0.59 (95% CI: 0.46–0.70).

For leg extensor power, the genetic modeling was started with the full ACE model, including all the plausible parameters (−2LL = 6,590.01, df = 693, AIC = 5,204.01; Table 3). In the final reduced ACE model for leg extensor power (−2LL = 6,590.01, df = 694, AIC = 5,202.01, P value of the difference between the models = 1.00; Table 3) genetic effects accounted for 67% (95% CI: 57–74%) of the variation at baseline (Fig. 4). At follow-up 48% (95% CI: 34–61%) of the variation was accounted for by the same genetic effects. Nonshared environmental effects in common for both baseline and follow-up explained 33% (95% CI: 25–43%) of the total variation at baseline and 11% (95% CI: 5–21%) at follow-up. The remaining variation of leg extensor power at follow-up was accounted for by shared (15%; 95% CI: 8–23%) and nonshared (26%; 95% CI: 19–34%) environmental effects specific to the follow-up. For leg extensor power the genetic correlation between baseline and follow-up measures was 1.0 (95% CI: 0.998–1.0) and the environmental correlation was 0.54 (95% CI: 0.50–0.67).

**DISCUSSION**

The results of the present study showed that the contribution of genetic effects to isometric knee extensor strength was

<table>
<thead>
<tr>
<th>Isometric knee extensor strength in 2000</th>
<th>Isometric knee extensor strength in 2003</th>
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<tbody>
<tr>
<td>0.761 (0.678–0.825)</td>
<td>0.313 (0.640–0.825)</td>
</tr>
</tbody>
</table>

Fig. 3. The most parsimonious Cholesky decomposition model for isometric knee extensor strength at baseline in the year 2000 and at follow-up in 2003. The model consists of additive genetic effect A1 and nonshared environmental effect E1 in common for the measurement points 2000 and 2003. In addition, isometric knee extensor strength measured in 2003 has its own nonshared environmental effect E2. The coefficients shown are standardized parameters (95% confidence interval) of the genetic and environmental latent factors.
stable, whereas in leg extensor power the proportion of genetic effects decreased from 67% to 48% during a 3-yr follow-up in older female twins. Occasion-specific genetic effects were not found. Instead, new environmental effects specific to the follow-up were observed for both muscle phenotypes. The initial hypotheses of the present study were that the contribution of genetic effects would decrease and the contribution of environmental effects for muscle strength and power would occur during the follow-up. These hypotheses were only partly supported by the results.

Earlier studies provided only limited information about whether the contributions of genetic and environmental effects on muscle are stable over time or whether they change with aging. Carmelli and Reed (4) did not observe any new genetic effects on grip strength in their cohort of older male twins followed up for 10 yr. Instead, they observed a new shared environmental effect specific to follow-up. The present results for leg extensor power are in line with those of Carmelli and Reed. Shared environmental effects are factors that influence family members similarly, making them more alike. These include rearing environment and health behaviors that have been adopted during childhood and continued throughout the life span, such as physical activity and eating habits (17). However, such effects can also have their origin in adulthood. For example, if twins choose the same occupation, their working physical activity may be similar. Shared environmental effects are determined as family resemblances that are not explained by genetic factors. Familial effects refer to the sum of variation of genetic effects and shared environmental effects. In our study, the proportion of familial effects on leg extensor power did not show any change at the 3-yr follow-up (63%) compared with baseline (67%). Although the present study was population based and included a relatively large baseline sample of older, independently living female twin pairs, at follow-up the sample size may be limited to discriminating genetic effects from shared environmental effects. All in all, the changes observed in the genetic components were not particularly large in either of our phenotypes. Consequently, the results may be interpreted as suggesting stability rather than change in genetic effects. However, we cannot rule out the possibility that, relatively speaking, environmental effects become more important with age when morbidity and its consequences become more prevalent. It is also possible that 3 yr may be too short a period to show changes in the contribution of genetic and environmental effects, especially among women without severe disabilities.

Some earlier studies examined the heritability of changes in muscle strength phenotypes over time. In the family study by Katzmarzyk et al. (12) the heritability of the change score in hand grip strength was 32%, while Zhai et al. (36) found a heritability of 64% for the change in lower limb strength in middle-aged people. These findings provide no information about stability and change in genetic effects on muscle strength with aging; however, they suggest that there may be an age-related genetic susceptibility to functional decline. The contribution of nonshared environmental effects on individual differences in leg extensor power and maximal isometric muscle strength was 30–40% at both baseline and follow-up. New nonshared environmental effects specific to the follow-up were observed for both phenotypes. These effects may consist of new disease processes, injuries, or changes in lifestyle.

### Table 3. Results of longitudinal Cholesky decomposition models for isometric knee extensor strength and leg extensor power

<table>
<thead>
<tr>
<th>Model</th>
<th>2-LL</th>
<th>df</th>
<th>AIC</th>
<th>Δ-2LL</th>
<th>Δdf</th>
<th>ΔAIC</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isometric knee extensor strength</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>7,871.84</td>
<td>692</td>
<td>6,487.84</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>8,158.63</td>
<td>695</td>
<td>6,768.63</td>
<td>286.79</td>
<td>3</td>
<td>280.79</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CE</td>
<td>9,169.86</td>
<td>695</td>
<td>7,779.65</td>
<td>1,297.81</td>
<td>3</td>
<td>1,291.81</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced ACE</td>
<td>7,871.86</td>
<td>694</td>
<td>6,483.86</td>
<td>0.02</td>
<td>2</td>
<td>−3.98</td>
<td>0.99</td>
</tr>
<tr>
<td>Leg extensor power</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>6,590.01</td>
<td>693</td>
<td>5,204.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>6,868.44</td>
<td>696</td>
<td>5,476.44</td>
<td>278.43</td>
<td>3</td>
<td>272.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CE</td>
<td>7,603.96</td>
<td>696</td>
<td>6,211.96</td>
<td>1,013.95</td>
<td>3</td>
<td>1,007.95</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced ACE</td>
<td>6,590.01</td>
<td>694</td>
<td>5,202.01</td>
<td>0.00</td>
<td>1</td>
<td>−2.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

A, additive genetic effects; C, shared environmental effects; E, nonshared environmental effects; −2LL, −2 times log likelihood; Δ−2LL, difference in −2LL compared with ACE model; df, degrees of freedom; Δdf, difference in df compared with ACE model; AIC, Akaike's information criterion; ΔAIC, difference in AIC compared with ACE model.

![Fig. 4. The most parsimonious Cholesky decomposition model for leg extensor power at baseline in the year 2000 and at follow-up in 2003. The model consists of additive genetic effect A1 and nonshared environmental effect E1 in common for the measurement points 2000 and 2003. In addition, leg extensor power measured in 2003 has its own shared C1 and nonshared environmental E2 effects. The coefficients shown are standardized parameters (95% confidence interval) of the genetic and environmental latent factors.](http://jap.physiology.org/Downloadedfrom/10.22036/20170428/10022032247.html)
Muscle strength and power are multifactorial phenotypes, and they are a result of interaction between environmental and genetic effects. Some people may have poorer “starting values” and thus are at increased risk for muscle impairments or sarcopenia. However, modification of environmental effects, such as physical activity and nutritional intake, has a key role in maintaining muscle performance at an adequate level in old age. Because environmental effects explained almost half of the variation in both phenotypes, our results also emphasize the importance of physical activity, training, and interventions in maintaining and improving muscle functions at older ages. In the present study, the sample consisted of quite healthy older women without severe disabilities. The physical activity level remained relatively stable during the 3-yr follow-up. Only a minor decrease in physical activity level could be seen, which might have an effect on the individual level but not cause significant changes in physical activity on the group level during the follow-up.

The age-related decline in muscle strength and power accelerates after age 65 yr (8, 9, 29). The decline in muscle strength and power is due not only to structural and functional changes in the neuromuscular system but also to diminished use of muscles. Decreased muscle strength causes mobility limitation and disability and restricts independent life (5, 23). In the present study, isometric knee extensor strength decreased by 1.7% annually and leg extensor power by 0.7%. The decline was similar in both MZ and DZ individuals. The decrease in isometric muscle strength is consistent with that found in earlier longitudinal studies (8, 9). Our findings on the change in leg extensor power differ from the 2–4% annual decrease suggested in an earlier cross-sectional study (29). One explanation for the only minor annual decline in muscle power could be that in both isometric knee extensor strength and leg extensor power there are many subjects whose result at follow-up measurement was 20–30% poorer compared with baseline measurement, but there were also subjects whose follow-up result was much (even 80–100%) better than their baseline result. In our study, changes over 3 yr ranged between 118% increase and 76% decrease in isometric muscle strength. In leg extensor power changes ranged between 61% increase and 77% decrease during that 3-yr follow-up. However, in leg extensor power compared with isometric muscle strength, there were more subjects whose follow-up result was better than their baseline result. These large individual differences between baseline and follow-up measurement produced an average decline in muscle power of only 0.7%/yr.

Also, differences in test performance could have an effect on the results. The leg extensor power test is a more difficult and more demanding performance for the subjects compared with isometric muscle strength measurement. Therefore at baseline, when the tests were first introduced to participants, maximum performance was probably not gained. At follow-up, when people were more familiar with the test, they reached a result closer to their full capacity. In the leg extensor power test, the participant needs to push as hard and as fast as possible toward a moving pedal, while in the isometric muscle strength measurement the participant needs to concentrate solely toward producing as much tension as possible against a static resistance. In addition, dynamic tests such as the leg extensor power test may cause more pain or, in particular, fear of pain and thus prevent the subject from reaching maximum even though pain was not reported.

It is possible that dropout from the study may have influenced the results. The baseline values of those who dropped out were lower. At baseline, both individuals in the pair had to agree to participate, whereas at follow-up the participation of both individuals in the pair was not necessary. Consequently, individuals with poor health status or experiencing a steeper decline in muscle function and consequent mobility limitation are more likely to have declined to participate in the follow-up assessments. This, however, is a common occurrence in longitudinal studies among older people and may result in underestimation of individual changes. When the individuals with poorer muscle strength or health status drop out from follow-up measurement, the sample may become more homogeneous, which may influence the contributions of the genetic and environmental effects. Consequent to selective dropout of the pairs with one member having poor health, the remaining pairs differ less for muscle strength and power than is the case in the population at large. The overestimation of the twin similarity might increase the apparent size of the shared environmental effect and at the same time decrease the proportion of the genetic effect. Alternatively, if both cotwins have the same disease-related cause of dropout, this would lead to the opposite effect on variance components, because such pairs may be more often MZ than DZ if the disease has a genetic underpinning. However, disease-discordant pairs are much rarer than disease-discordant pairs for nearly all conditions. For example, for a disease with a very high heritability (80%) such as schizophrenia, the concordance for the disease is only 50%; i.e., half of the pairs are discordant; common cancers have low heritabilities (14), likewise coronary heart disease in older subjects (15).

The measurements used in the present study are commonly used in studies on the functional capacity of older people, and they have also proven to be suitable and safe among older persons (1, 3, 24). Comparing the coefficients of variation of isometric knee extensor strength (6%) and leg extensor power (8%) with the contributions of specific nonshared environmental effect $E_2$ (isometric knee extensor strength 29%, leg extensor power 26%) it could look as though the test-retest reliability of the tests is low. However, the nonshared environmental effect contains measurement error but it contains also other environmental factors such as diseases, accidents, and other individual-specific effects, which increase the contribution of $E_2$. The present study was population based and included a relatively large sample of older twin pairs. However, this study included only female twin pairs without severe disabilities at baseline, which limits the generalizability of the results. Because heritability estimates can differ by age group and sex, the results can only be generalized to older community-living women.

In conclusion, the results of this study suggest that the same genetic effects were underlying individual differences in isometric knee extensor strength at baseline and follow-up. The results also showed that the extent to which genetic differences contributed to differences in muscle strength did not change. For leg extensor power the situation was similar, except that a small decrease in the relative proportion of genetic effects was observed over the 3-yr follow-up. This should, however, be interpreted with caution because no change was found in
overall familial effects. We observed new environmental effects specific to the follow-up phenotypes. These results provide a basis for further studies on genetic influences on the phenotypes relevant to the disablement process in old age.

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We are grateful to the twin pairs who participated in the laboratory examination in 2000 and 2003.

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