Isometric muscle strength and muscle power are both strong predictors of functional status, particularly among older people (3,20). However, it has been suggested that poor muscle power, rather than strength, is more closely correlated with the risk for mobility limitation (3). Furthermore, interventions aiming to improve power have been suggested to be more efficient in improving mobility of older individuals than traditional strength training programs (13).

Maximum isometric muscle strength has been defined as the maximum voluntary contraction performed at a specific joint angle against an unyielding resistance. Muscle power is the product of force generation and speed of muscle contraction, so it refers to the ability of the neuromuscular system to produce the greatest possible force as fast as possible.

Maximum isometric muscle strength decreases after the age of 30, and the decrease accelerates around the age of 60 due to diminished use, and due to structural and functional changes in the neuromuscular system with aging. Isometric muscle strength decreases on the average 1–2% per year (6,22), whereas the decline in maximal muscle power may be steeper. Cross-sectional data suggest deterioration in maximal power of 2–4% per year (28). The steeper decline in power than strength may, at least partly, be due to greater effects of aging on Type II (fast-twitch) than Type I (slow-twitch) fibers (10) and the decreased conduction velocity of motoneurons innervating Type II fibers (32).

Data about the heritability of lower extremity muscle strength and power among older people are limited. Arden and Spector (1) estimated that among 45- to 70-yr-old female twins, 46% of the variance in leg extensor power was accounted for by genetic effects. For maximum isometric knee extensor strength, genetic effects account for approximately one third of the interindividual variation of the trait (17,29).

Muscle strength and power are highly correlated (28). However, previous studies have investigated the heritability of isometric muscle strength and leg extensor power separ-
rately. Currently, it is not known whether there are genes having a general effect on muscle function in old age, or whether the contribution of genes is specific for each type of force production. This issue can be investigated using multivariate genetic models. Previously, a study among 10-yr-old male twin pairs and their parents suggested that the contribution of genetic effects could depend on the type and velocity of contraction (29).

The purpose of the present study was to examine the relative contribution of genetic and environmental effects on maximum leg extensor power and to investigate whether leg extensor power and maximum voluntary isometric knee extensor strength share a genetic component, which would indicate that they are at least partly regulated by the same genes.

METHODS

Approach to the problem and experimental design. Differences between individuals may be due to environmental or genetic factors or to both of these factors. The classical twin study provides an opportunity to differentiate sources of familial resemblance that may arise from shared genes, shared environments, or both. The mathematical modeling of data is based on the fact that two types of twinning occur. Monozygotic (MZ) twins share all of their genes (100%), whereas dizygotic (DZ) twins share on the 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 the genetic influence on the trait (19). In the present study, additive genetic effect (A), shared environmental effects (C), and nonshared environmental effects (E) were viewed as the independent variables thought to underlie differences in the measured traits, maximal voluntary isometric knee extensor strength, and leg extensor power which were the dependent variables (15).

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. Detailed selection procedures, determination of the zygosity and description of the participants have been described elsewhere (31). Briefly, the participants were recruited from the Finnish Twin Cohort Study, which consisted of 13,888 adult twin pairs, first studied in 1975 (8,9). Among them were originally 1260 respondent female twin pairs born in 1924–1937. Of this group, an invitation to take part in the present study was sent in the year 2000 to 178 MZ, 212 DZ, and 24 twin pairs with uncertain zyosity (XZ). To be recruited to the study, both individuals in the pair had to agree to participate. The reasons for nonparticipation were that one or both sisters were unwilling to take part in the study (50 MZ, 51 DZ, and 5 XZ twin pairs) or had poor health status (28 MZ, 52 DZ, and 5 XZ twin pairs) or had died after vital status was last updated for all cohort members (2 MZ, 3 DZ, and 1 XZ). The zyosity of the twins was determined at the baseline study in 1975 by a validated questionnaire (23,24), and in the present study it was assessed using DNA extracted from a venous blood sample by a battery of 10 highly polymorphic gene markers. The final sample of this study was 101 MZ and 116 DZ twin pairs.

Before the laboratory examinations, the subjects provided a written informed consent. The study was approved by the ethics committee of the Central Hospital District of Central Finland. Both of the individuals in the pair came to the laboratory at the same time and received their individual test schedules. First, all subjects underwent a 30-min clinical examination. Self-report of acute and chronic diseases, medication, and the present physical activity had been obtained earlier, and was confirmed by the physician during the clinical examination. Those who reported having used hormonal replacement therapy for more than 10 yr during the last 15 yr and had continued doing so over the age of 60 were considered as hormonal replacement therapy users. Those who reported taking systemic corticosteroid treatment (injection or tablets) currently or had used them for more than a 5-yr period during the last 10 yr were considered as corticosteroid users.

The participants were classified as sedentary, moderately active, or active on the basis of their physical activity self-report. Physical activity was measured using the scale developed by Grimby (7) with slight modifications. Those reporting no other activity but light walking ≤2× wk⁻¹ were rated as sedentary. Those reporting walking or other light exercise at least 3× wk⁻¹, but no exercise more intense than that, were rated as moderately active. If a participant reported moderate or vigorous exercise at least 3× wk⁻¹, she was rated as active.

Measurements. Leg extensor power of single leg was measured using 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 makes the direction of movement almost horizontal. For each subject, the distance between the seat and the pedal was individually determined based on leg length. When the subject was settled into the back of the seat, the distance between the push-pedal and the seat was adjusted by placing one foot on the fully depressed footplate and pushing the seat back slowly until the leg was fully extended (2). First, the dominant leg was measured, followed by the nondominant leg. The dominant leg was determined as the leg of the dominant hand side for both muscle power and maximum isometric muscle strength measurements. The subject was instructed to push the pedal as hard and fast as possible. Two to three practice trials were allowed for the participants to familiarize themselves with the method. Five to nine maximal efforts per leg, separated by 30-s rests, were conducted. For each subject, the best performance with the highest value was accepted as the result.

The average power of each push was calculated from the final velocity of the flywheel and the amount of rotation during the push, together with the moment of inertia of the
The maximum voluntary isometric knee extensor strength was measured on the dominant side in a sitting position using an adjustable dynamometer chair (Good Strength, Metitur LTD, Palokka, Finland). The measurement was done at the knee angle of 60° from full extension. The ankle was fastened by a belt to a strain-gauge system. After familiarization to the measurement procedure, three to five maximal efforts, each separated by a 1-min rest, were conducted. The data were digitized into newtons, recorded, and stored on a computer using Good Strength software package (Metitur LTD, Palokka, Finland). For each subject the best performance with the highest value was accepted as the result.

The leg extensor power was measured by three and isometric knee extensor strength by two trained measurers who worked on alternate days. Verbal encouragement was given in every test. The test-retest intraclass correlation coefficient (ICC) in leg extensor power measurement was 0.922, and the coefficient of variation between test-retest was 8%. The Pearson correlation coefficient for maximum voluntary isometric knee extensor strength was 0.965, and a coefficient of variation was 6%, between two consecutive measurement occasions with a 1- to 2-wk interval among 80-yr-old men and women (21). The Nottingham Leg Extensor Power Rig (2) and the dynamometer chair used here (21) have previously been shown to be reliable and valid as well as acceptable and safe also for older people.

### Statistical methods.

In the genetic modeling, the total variance in a trait is decomposed into components representing the genetic and environmental variances. Typically, genetic effects are classified into additive genetic effects and nonadditive genetic effects (D). Additive genetic effects refer to the individual alleles summed over the contributing loci and nonadditive effects refer to interactions between alleles at the same loci or different loci. Environmental effects are classified into shared environmental effects and nonshared environmental effects. Shared environmental effects are common to both twins. Such effects may, for example, be related to rearing environment, where certain factors have affected both individuals in the same way in their childhood and then tracked over to adulthood behaviors. Nonshared environmental effects are exposures that are not shared by the members of a pair, such as diseases and accidents that affect only one sibling, and thereby contribute only to within-pair differences in a trait. In addition, nonshared environmental effects contain the measurement error and are thus always included in models. The expected correlation between additive genetic effects in MZ and in DZ twins, respectively, is 1.0 and 0.5 and between nonadditive genetic effects 1.0 and 0.25. For shared environmental effects, the correlation is 1.0 and for nonshared environmental effects zero for both MZ and DZ twins (15).

The different possible combinations of variance components A, D, C, and E in the genetic models are ACE, ADE, AE, CE, and E. Nonadditive genetic effects and shared environmental effects cannot be estimated simultaneously, when only data on twin pairs raised together is available. In genetic modeling, the aim is to build up a model, which fits the data well with the least possible explanatory components.

To quantify the respective genetic and environmental contributions to leg extensor power and isometric knee extensor strength, they were first modeled separately using univariate genetic analyses. Next, to evaluate whether leg extensor power and isometric knee extensor strength share a genetic component or whether the genetic effects are specific for each measurement, a bivariate Cholesky decomposition model was used. In such an ACE-model (Fig. 1), genetic effect $A_1$ is shared by strength and power, whereas genetic effect $A_2$ loads only onto power. The shared environmental ($C_1$, $C_2$) and nonshared ($E_1$, $E_2$) environmental effects have similar patterns of loadings. In the present study, the analysis was started with the hypothetical full ACE bivariate model including all plausible parameters. To get 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 reached. Biological plausibility was used as a guideline as well in selecting the parameters to be dropped.

Means and standard deviations (SD) were computed using the Mx program (version 1.52a; (14,15). Age-adjusted intraclass correlation coefficients were computed for the MZ and DZ twin pairs separately to estimate the level of within-pair similarity. The genetic analyses were carried out using raw data input with full information likelihood in Mx (14,15), which permits twin pairs with incomplete data to be included. The goodness of fit of the ACE and reduced ACE-models was assessed comparing the fit of

![Figure 1](http://www.acsm-msse.org)

**FIGURE 1**—The full Cholesky decomposition ACE-model for the maximum isometric knee extensor strength and the leg extensor power with additive genetic effects ($A_1$, $A_2$), shared environmental effects ($C_1$, $C_2$), and nonshared environmental effects ($E_1$, $E_2$). Shown $a_{11}$-$a_{22}$, $c_{11}$-$c_{22}$, and $e_{11}$-$e_{22}$ are standardized path coefficients from $A$, $C$, and $E$.  

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the model to the fit of the saturated model. In the saturated model means are modeled in a similar way as in the ACE-model, whereas the covariance matrices are unconstrained and all variances and covariances in MZ and DZ twins are estimated. The other models, for instance, AE, CE, E, and reduced ACE-model, were compared with the full ACE-model. Nested submodels were compared with the difference $\chi^2$-tests (15).

Acceptable leg extensor power result was obtained for 195 MZ and 224 DZ twin individuals. Isometric knee extensor strength result was obtained for 195 MZ and 221 DZ twin individuals. Age has been taken into account as a covariate in the genetic modeling. The effect of age explained 6% of the total variance in leg extensor power and isometric knee extensor strength in MZ and DZ twins.

RESULTS

Descriptive statistics. All the participants were community-dwelling women who were able to move independently. Typically, their physical activity level consisted of household and gardening as well as walking for fitness. Half of the sample (50%) was classified as moderately active, 28% as sedentary, and 22% as active based on the physical activity self-report. Neither the use of medications nor the level of current physical activity differed systematically between MZ and DZ twins. Table 1 summarizes the physical characteristics of the subjects and the results of leg extensor power and isometric knee extensor strength measurements. No statistically significant differences between the MZ and DZ twin individuals were observed in means or variances of age, body height or weight, body mass index, or leg extensor power and isometric knee extensor strength. The prevalence of coronary heart disease (12%), asthma (8%), cerebrovascular disease (7%), diabetes (6%), knee (29%), hip (14%), or foot and ankle osteoarthritis (12%) did not differ systematically between MZ and DZ twins. Altogether, 22% of the subjects were hormonal replacement therapy users and 4% corticosteroid users.

Correlation analysis. In the leg extensor power, the age-adjusted ICC for MZ twins was 0.623 (95% confidence interval (CI) 0.500–0.746) and for the DZ twins 0.418 (95% CI 0.265–0.572), suggesting influence of additive genetic effects and shared environmental effects to individual differences. In the isometric knee extensor strength, the age-adjusted ICC for MZ twins was 0.459 (95% CI 0.300–0.617) and for DZ twins 0.282 (95% CI 0.108–0.456), also suggesting contribution from familial factors to the variability.

Genetic modeling. Results of the univariate genetic analysis of leg extensor power indicated that in the ACE-model, the additive genetic effects accounted for 30% (95% CI 0–66%), and shared environmental effects 29% (95% CI 0–57%) of individual differences, whereas remaining variance was due to nonshared environmental effects (41%, 95% CI 31–54%). The fit of the AE- or CE-models ($P = 0.095$ for difference with the ACE-model) was not significantly worse than theoretically more acceptable ACE-model; however, the E-model was clearly not as good. In the AE-model, additive genetic effect accounted for 61% (95% CI 50–70%) of the variance and the remaining variance was explained by nonshared environmental effect. In the CE-model, 52% (95% CI 41–61%) of the variance was explained by shared environmental effects with remaining variance being due to nonshared environmental effects.

For isometric knee extensor strength, the AE-model was considered the most acceptable model for the data. In the AE-model the additive genetic effects accounted for 49% (95% CI 34–61%) and nonshared environmental effects 51% (95% CI 39–67%) of the total variance in strength.

Also, the CE-model was statistically appropriate for the data. In the CE-model, the shared environmental effect accounted for 36% (95% CI 23–47%) of the total variance in strength. The remaining 64% (95% CI 53–77%) of variance was due to the nonshared environmental effect. However, the AE-model was selected as the final model, because dropping A from the ACE-model worsened the model more than dropping C (Table 2).

The Cholesky decomposition modeling was started with the full ACE-model with all parameters included. Even though the full ACE-model fitted the data well ($\chi^2 = 10.49$, $df = 11$, $P = 0.49$), there were several statistically nonsignificant parameters. The model was modified by dropping the nonsignificant parameters with zero or small coefficients one by one, until a more parsimonious and theoretically acceptable model was reached (Table 3). In the final reduced ACE-model, isometric knee extensor strength and leg

### Table 1. Physical characteristics and results of the leg extensor power and the maximum isometric knee extensor strength measurements of monozygotic (MZ) and dizygotic (DZ) twin individuals; means and standard deviations (SD).

<table>
<thead>
<tr>
<th>Variables</th>
<th>MZ</th>
<th>DZ</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>202</td>
<td>68.16</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>202</td>
<td>69.47</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>202</td>
<td>158.21</td>
</tr>
<tr>
<td>BMI (kg·m$^{-2}$)</td>
<td>202</td>
<td>27.89</td>
</tr>
<tr>
<td>Leg extensor power (W)</td>
<td>195</td>
<td>102.96</td>
</tr>
<tr>
<td>Isometric knee extensor strength (N)</td>
<td>195</td>
<td>297.08</td>
</tr>
</tbody>
</table>

No statistically significant differences were observed between the MZ and DZ twin individuals.

### Table 2. Results of the univariate genetic analyses for the leg extensor power and the maximum isometric knee extensor strength.

<table>
<thead>
<tr>
<th></th>
<th>Difference $\chi^2$ Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\Delta LL$</td>
</tr>
<tr>
<td>Leg extensor power</td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>4058.12</td>
</tr>
<tr>
<td>AE</td>
<td>4060.91</td>
</tr>
<tr>
<td>CE</td>
<td>4060.92</td>
</tr>
<tr>
<td>E</td>
<td>4025.46</td>
</tr>
<tr>
<td>Isometric knee extensor strength</td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>4801.95</td>
</tr>
<tr>
<td>AE</td>
<td>4802.03</td>
</tr>
<tr>
<td>CE</td>
<td>4805.45</td>
</tr>
<tr>
<td>E</td>
<td>4833.62</td>
</tr>
</tbody>
</table>

A, additive genetic effects; C, shared environmental effects; E, nonshared environmental effects; $\Delta LL$, $-2$ times log-likelihood; $\Delta LL$ differences in $-2LL$ compared with ACE-model; $df$, degrees of freedom; $\Delta df$, differences in $df$ compared with ACE-model.
extensor power had an additive genetic effect in common (A1) explaining 48% (95% CI 33–60%) of the total variance in strength and 32% (95% CI 17–46%) in power (Fig. 2, Table 3). This model implies that the genetic effects on isometric knee extensor strength and leg extensor power were fully correlated (rg = 1.0). The two traits also had a nonshared environmental effect in common (E1), which accounted for 52% (95% CI 40–67%) of the total variance in strength and 4% (95% CI 1–10%) in power. The nonshared environmental effect between isometric knee extensor strength and leg extensor power correlated moderately (re = 0.33). Leg extensor power had also a specific shared environmental effect (C2) explaining 28% (95% CI 17–39%), and a nonshared environmental effect explaining 36% (95% CI 28–46%) of the total variance in power. This final reduced ACE-model fitted well for the data ($\chi^2 = 12.32, df = 14, P = 0.58$ compared with the saturated model).

**DISCUSSION**

The major finding in this study was that leg extensor power and maximum voluntary isometric knee extensor strength shared a genetic component in common, which accounted for 32% of the total variance in power and 48% in strength. The results suggest that regardless of the type of contraction, the same genes regulate muscle functions, at least when measured on largely the same muscle groups. As far as we know, this is the first study to examine maximum isometric muscle strength and muscle power combined in the same genetic model.

In the present study, the reduced ACE Cholesky decomposition model was selected as the best model to explain the data. The main reasons for choosing the reduced ACE-model was that it fitted the data well, was theoretically plausible, provided estimates consistent with earlier studies (1,5), and logically followed from the results of the univariate genetic models.

Genetic effects may be mediated largely through similar mechanisms on strength and power. For example, lean body mass, muscle size, and muscle fiber characteristics all correlate with muscle function, and are known to be under strong genetic regulation. Earlier studies have shown that genetic effects accounted for 52–84% of the variation in lean body mass among older female twins (1,16). In muscle cross-sectional area, 66–92% of the variation was explained by genetic effects, depending on the muscle and the age group studied (11,30). There is some evidence to suggest that also neural factors regulating physical movements are at least moderately regulated by genes. In the study by Simonen et al. (25), where testing was based on the speed of hand

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**TABLE 3. Proportions of variances explained by the path coefficients in the Cholesky decomposition model for the maximum isometric knee extensor strength (strength) and the leg extensor power (power).**

<table>
<thead>
<tr>
<th>Path</th>
<th>ACE (95% CI)</th>
<th>Reduced ACE (95% CI)</th>
<th>AE (95% CI)</th>
<th>CE (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strength</td>
<td>Power</td>
<td>Strength</td>
<td>Power</td>
</tr>
<tr>
<td>a11</td>
<td>0.65 (0.11–0.77)</td>
<td>—</td>
<td>0.69 (0.58–0.78)</td>
<td>—</td>
</tr>
<tr>
<td>a21</td>
<td>—</td>
<td>0.36 (0.18–0.74)</td>
<td>—</td>
<td>0.56 (0.42–0.68)</td>
</tr>
<tr>
<td>a22</td>
<td>—</td>
<td>0.42 (0.00–0.64)</td>
<td>—</td>
<td>0.59 (0.00–0.83)</td>
</tr>
<tr>
<td>c11</td>
<td>0.25 (0.00–0.61)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>c21</td>
<td>—</td>
<td>0.54 (0.00–0.75)</td>
<td>—</td>
<td>0.53 (0.41–0.62)</td>
</tr>
<tr>
<td>c22</td>
<td>—</td>
<td>0.00 (0.00–0.60)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>e11</td>
<td>0.72 (0.63–0.83)</td>
<td>—</td>
<td>0.72 (0.63–0.82)</td>
<td>—</td>
</tr>
<tr>
<td>e21</td>
<td>—</td>
<td>0.24 (0.13–0.36)</td>
<td>—</td>
<td>0.21 (0.10–0.32)</td>
</tr>
<tr>
<td>e22</td>
<td>—</td>
<td>0.58 (0.51–0.67)</td>
<td>—</td>
<td>0.60 (0.53–0.68)</td>
</tr>
</tbody>
</table>

**Difference $\chi^2$ tests**

- $\Delta$-2LL = 8737.00
- $df$ = 825
- $\Delta$df = 3
- $P$ = 0.008

A, additive genetic effects; C, shared environmental effects; E, nonshared environmental effects; a11, a21, a22, standardized path coefficient of phenotype on effect A; c11, c21, c22, standardized path coefficient of phenotype on effect C; e11, e21, e22, standardized path coefficient of phenotype on effect E; $\Delta$-2LL, difference in −2LL compared with ACE-model; $\Delta$df, difference in df compared with ACE-model.

---

**FIGURE 2—The most parsimonious Cholesky decomposition model for the maximum isometric knee extensor strength and the leg extensor power consists of two effects in common for isometric muscle strength and leg extensor power: additive genetic effect (A1) and nonshared environmental effect (E1). In addition, leg extensor power has its own shared environmental effect (C2) and nonshared environmental effect (E2). The percentages (95% confidence intervals) are the proportions of genetic and environmental effects.**

---

**A1**

Isometric knee extensor strength

48% (33–60%)

32% (17–46%)

52% (40–67%)

4% (1–10%)

36% (28–46%)

**C2**

Leg extensor power

28% (17–39%)

**E1**

Nonshared environmental effect

**E2**

Nonshared environmental effect

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and foot response to an external stimuli, genetic and shared environmental effects explained a substantial proportion of individual differences among middle-aged men.

In the present study, shared and nonshared environmental effects explained over half of variability in leg extensor power and isometric knee extensor strength. Nonshared environmental effect in common for power and strength probably consists of factors that have an effect on the force production. Leg extensor power had also its own specific nonshared environmental effect explaining 36% of the total variance, which may indicate features specific in explaining velocity of movement. The specific shared environmental effect on leg extensor power may be a result of sisters resembling each other in their physical activity habits. Earlier studies have shown that physical activity behavior is influenced by familial factors and that the adoption of a physically active lifestyle in childhood strongly influences the physical activity level throughout the lifespan (18,26).

One explanation for the observed difference in the contributions of genetic and environmental effects on leg extensor power and isometric knee extensor strength could be offered by their differing sensitivity to age. Aging affects particularly the largest and fastest conducting motor units, potentially decreasing velocity, and consequently also power more than strength (32). The fiber type distribution is known to explain interindividual differences in power (27), whereas the same relationship was not being found between strength and fiber composition (12). As age has a greater influence on the faster motor units, the variance between individuals in power may increase, and thus the relative proportion of genetic and environmental effects may change. No previous studies measuring power at two time points exist; however, the age effect of heritability has been shown for strength (5). During a 10-yr follow-up the heritability of isometric handgrip strength decreased from 35% to 45% among male twins aged on average 63 yr, whereas the same relationship was not being found between strength and fiber composition (12). As age has a greater influence on the faster motor units, the variance between individuals in power may increase, and thus the relative proportion of genetic and environmental effects may change. No previous studies measuring power at two time points exist; however, the age effect of heritability has been shown for strength (5). During a 10-yr follow-up the heritability of isometric handgrip strength decreased from 35% to 45%, whereas the shared environmental effects increased from 39% to 45% among male twins aged on average 63 yr at the baseline (4). In our study, the age-induced variability may be somewhat diminished. The requirement that both individuals of the pair had to participate may have resulted in the exclusion of twin pairs in which one or both sisters had poor health or mobility. This may have resulted in overestimation of the twin similarity by increasing the proportion of the shared environmental effect and decreasing the relative contribution of genetic effects.

The present study is a population-based study and includes a relatively large sample of older, independently living female twin pairs. However, if the demands of twin analysis were taken into account, the sample size may be limited in discriminating genetic effects from the shared environmental effects. In the present study, the issue of statistical power was investigated by creating covariance matrices according to the full theoretical Cholesky decomposition model presented in Figure 1. When the full Cholesky decomposition model except for one of the parameters was fitted, the power was 0.50 for the parameters of the A23 and C23 components and 1.0 for the parameters of E23. The power for the A1 component was 1.0. Statistical power for the final model was investigated by computing covariance matrices for the reduced ACE-model (Fig. 2). When the reduced ACE-model except for one of its parameter was fitted, the estimated power was 1.0. However, a slight limitation in the ability to distinguish shared environmental effects from genetic effects was observed, which is a limitation inherent in modest-sized studies based on twins reared together (15). Future studies should secure larger samples or include other types of relatives to ensure the clear identification of the proportion of the genetic and shared environmental effects.

In conclusion, muscle power and isometric muscle strength of women aged 63 yr and over shared a genetic effect in common, which could be mediated through physiological mechanisms that are under strong genetic regulation. This indicates that heredity regulates muscle performance in older women. Half of the variation was explained by environmental effects, which emphasizes the importance of the physical activity, training, and interventions to maintain and improve muscle functions in older ages.

We are thankful to the twin pairs who participated to the laboratory examination.

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