Heritability of maximal isometric muscle strength in older female twins

Kristina Tiainen,1 Sarianna Sipilä,1 Markku Alen,2 Eino Heikkinen,1,3 Jaakko Kaprio,4,5 Markku Koskenvuolo,6 Asko Tolvanen,7 Satu Pajala,1,3 and Taina Rantanen1,3

1Department of Health Sciences, 2The Finnish Centre for Interdisciplinary Gerontology, and 3Department of Psychology, University of Jyväskylä, FIN-40014 Jyväskylä; 4Pearunka Medical Rehabilitation Center, FIN-41340 Laukaa; 5Department of Public Health, University of Helsinki, FIN-00014 Helsinki; 6Department of Mental Health, National Public Health Institute, FIN-00300 Helsinki; and 7Department of Public Health, University of Turku, FIN-20014 Turku, Finland

Submitted 26 February 2003; accepted in final form 3 September 2003


The purpose of the present study was to examine genetic and environmental effects on maximal isometric handgrip, knee extension, and ankle plantar flexion strength. In addition, we wanted to investigate whether the strength of these three muscle groups shares a genetic component or whether the genetic effect is specific for each muscle group. Muscle strength was measured as part of the Finnish Twin Study on Aging in 97 monozygotic (MZ) and 102 dizygotic (DZ) female twin pairs, aged 63–76 yr. The MZ and DZ individuals did not differ from each other in age, body height, weight, or self-related health. The age-adjusted pairwise (intraclass) correlations of the MZ and DZ twins were, respectively, 0.462 and 0.242 in knee extension, 0.435 and 0.345 in handgrip, and 0.512 and 0.435 in ankle plantar flexion strength. The multivariate genetic analysis showed that handgrip and knee extension strength shared a genetic component, which accounted for 14% (95% confidence interval: 4–28%) of the variance in handgrip strength and 31% (95% confidence interval: 18–45%) of knee extension strength. The influence of genetic effects on ankle plantar flexion strength was minor and not significant. Furthermore, these three muscle groups had a nongenetic familial effect in common and nonshared environmental effects in common. The results suggested that muscle strength is under a genetic regulation, but also environmental effects have a significant role in explaining the variability in the muscle strength.

MATERIALS AND METHODS

Participants. This study is part of the Finnish Twin Study on Aging, a study of genetic and environmental effects on the disablement process in older female twins. The participants were recruited from the Finnish Twin Cohort Study, which was launched in 1974 and is located at the Department of Public Health at the University of Helsinki. The Finnish Twin Cohort consists of 13,888 adult twin pairs of known zygosity at the baseline study in 1975 by a validated questionnaire, which included a series of questions on the twins’ similarities and dissimilarities during childhood (27). According to the questionnaire, the twin pairs were classified as monozygotic (MZ), dizygotic (DZ), and uncertain zygosity (XZ). The method categorized 92.7% of the pairs as MZ or DZ with 1.7% probability of misclassification (26, 27).

In the Finnish Twin Cohort (13, 14), there were 1,260 respondent female twin pairs born in 1924–1937 and first studied in 1975. Of this group, an invitation to take part in the present study was sent to 178 MZ, 212 DZ, and 24 XZ twin pairs selected solely on the basis of age and...
zygosity. 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), 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). Ninety-eight MZ, 106 DZ, and 13 XZ twin pairs participated in the laboratory examinations. With the use of DNA extracted from a venous blood sample, zygosity was assessed by a battery of 10 highly polymorphic gene markers among XZ twin pairs. As a result, four XZ twin pairs were classified as MZ and nine as DZ. These analyses were carried out by the paternity testing laboratory of the National Public Health Institute in Helsinki, Finland.

Measurements. Twin pairs arrived from all over Finland the evening preceding the laboratory measurements and stayed overnight in a hotel. 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 with ECG and blood pressure measurements. A physician interviewed and examined the participants. Self-reports of acute and chronic diseases and medication had been obtained earlier and were confirmed by the physician during the clinical examination. Those who reported having used hormonal replacement therapy (HRT) for more than 10 yr during the last 15 yr and had continued doing so over the age of 60 yr were considered HRT users. Those who reported recent systemic corticosteroid treatment (injection or tablets) currently or had used them for over 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 by using the scale by Grimby (11) with slight modifications. Those reporting no other activity but light walking two or fewer times a week were rated as sedentary. Those reporting walking or other light exercise at least three times a week, but no exercise more intensive than that, were rated as moderately active. If a participant reported moderate or vigorous exercise at least three times per week, she was rated as active.

After the clinical examination, laboratory measurement battery took place. The maximal isometric muscle strength measurements were performed on the dominant side in a sitting position by using an adjustable dynamometer chair (Good Strength, Metitur, Palokka, Finland). The tests were done by two trained physiotherapists who worked on alternate days. The dynamometer was calibrated every morning before the measurements. Handgrip strength was measured with a dynamometer fixed to the arm of the chair with the elbow flexed at 90°. Knee extension strength was measured at the knee angle of 60° from full extension with the ankle fastened by a belt to a strain-gauge system. In the ankle plantar flexion strength measurement, the ankle was set at an angle of 90° and was fastened by a belt to a strain-gauge system. The leg was elevated to a horizontal position, and the knee was set at an angle of 20° from full extension. The subjects were allowed to familiarize themselves with the method by doing two to three submaximal trials. Three to five maximal efforts, separated by a 1-min rest, were conducted. During the measurements, the subjects were verbally encouraged to produce their maximum. For each subject, the best performance with the highest value was accepted as the result.

In our laboratory, the coefficient of variation between two consecutive measurements performed 2 wk apart has earlier been 6.1% (SD 6.9) for handgrip strength and 6.3% (SD 5.7) for knee extension strength (22). The coefficient of variation in the ankle plantar flexion measurements in the present study was 15.9% (SD 10.7).

Handgrip strength results were obtained for all of the MZ and DZ twin pairs. Acceptable knee extension strength results were obtained for 97 MZ and 102 DZ twin pairs, and ankle plantar flexion strength results for 96 MZ and 102 DZ twin pairs. To increase the power of the analysis, missing values were replaced by values computed by the expectation-maximization method by using SPSS 11.0.1 (31) missing value analysis. Imputation was performed if, due to a technical problem, the subject had only one missing strength result, whereas her sister had an acceptable result in that particular strength measurement. Knee extension strength value was imputed for two DZ twin individuals, and ankle plantar flexion strength value for three MZ and four DZ twin individuals. When the individuals with imputed values were included, handgrip, knee extension, and ankle plantar flexion strength results were obtained for 97 MZ and 102 DZ twin pairs. The genetic analysis was done on these pairs. The results of the analyses performed without the imputed values were essentially the same as for the inclusion analyses.

Before the laboratory examinations, the subjects were informed about the study, and a written consent form was signed. The study was approved by the Committee on Ethics of the Central Hospital of Central Finland.

Statistical methods. Normality of data and equality of the means and variances between the MZ and DZ twins were calculated and tested by using Stata (32), which makes it possible to take into account the fact that the data consist of twin pairs rather than unrelated individuals. The level of statistical significance was set at $P < 0.05$.

Age-related effects were corrected by unstandardized residuals obtained from linear regression on age models. Age-adjusted values were further used for genetic models. Intraclass correlation coefficients were computed for the MZ and DZ twin pairs separately to estimate the level of within-pair similarity. The correlation pattern obtained reveals whether genetic or nongenetic familial factors play a role in explaining the variability in the trait.

In classic twin studies, the aim is to differentiate sources of familial resemblance that can arise from shared genes, shared environments, or both. MZ twins share all of their genes (100%), whereas 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.

Univariate genetic analysis. To quantify the respective genetic and environmental contributions to each of the muscle strength measurements, univariate genetic analysis was used. In genetic modeling, the variance in a trait is decomposed into the genetic and environmental variances. Typically, genetic effects are classified into additive genetic effects (A) and nonadditive genetic effects (D), where A refers to the additive effects of the individual alleles summed over the loci, and D refers to interactions between alleles at the same loci or different loci. Environmental effects are classified into shared environmental effects (C) and nonshared environmental effects (E). 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 (E) 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 measurement error. The correlation between additive genetic effects in MZ and in DZ twins is 1.0 and 0.5 and between nonadditive genetic effects is 1.0 and 0.25, respectively. For shared environmental effect, the correlation is 1.0 for both MZ and DZ twins, and for nonshared environmental effects, it is 0 for both MZ and DZ twins (19).

The different possible combinations of parameters A, D, C, and E in the genetic models are E, AE, AC, ACE, and ADE. Nonadditive genetic effects and shared environmental effects cannot be estimated simultaneously. The genetic and environmental parameters described above were estimated from variance-covariance MZ and DZ data simultaneously using the Mx program (18, 19). In genetic modeling, the aim is to build up a model that fits the data well and has as few explanatory components as possible. To evaluate the goodness of fit of genetic models, $\chi^2$ and its $P$ value were used. The alternative models

J Appl Physiol • VOL 96 • JANUARY 2004 • www.jap.org
were compared by Akaike’s information criterion (AIC = $\chi^2 - 2 \times$ degrees of freedom), which is smaller for better fitting models.

Multivariate genetic analysis. Multivariate genetic analyses were carried out by using the independent pathway model (Fig. 1) to evaluate whether handgrip, knee extension, and ankle plantar flexion strength share a genetic component or whether the genetic effect is specific for each muscle group and to what extent the effects are common or trait specific. The full independent pathway model consists of genetic and environmental effects that are common to ankle plantar flexion, handgrip, and knee extension strength (Ac, Cc, Ec) as well as genetic (As1, As2, and As3, respectively), shared environmental (Cs1, Cs2, and Cs3, respectively), and nonshared environmental (Es1, Es2, and Es3, respectively) effects, which are specific for each strength measure. The analysis was started with the hypothetical full independent pathway model. 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 the time, until the model with the best fit was reached.

RESULTS

Descriptive statistics. No significant differences between the MZ and DZ twins were observed in the means or SDs for age, body height, or weight, or for the muscle strength measurement, except in the variance of handgrip strength, which was 15% larger in the MZ twin pairs than in the DZ twin pairs (Table 1). The effect of age explained 2.0% of the variance in ankle plantar flexion strength, 0.7% in handgrip strength, and 5.6% in knee extension strength. The prevalence of coronary heart disease (12%), asthma (7%), cerebrovascular dysfunction (6%), and diabetes (6%), knee (27%), hip (14%), or foot and ankle osteoarthritis (11%) did not differ systematically between MZ and DZ twin individuals. Altogether, 23% of the subjects were HRT, and 4% were corticosteroid users. On the basis of physical activity self-report, 28% were classified as sedentary, 50% as moderately active, and 22% as active. Use of medications and the level of current physical activity did not differ systematically between MZ and DZ twin individuals.

Correlation analysis. In knee extension strength, the age-adjusted pairwise (intraclass) correlation for the MZ twins ($r_{MZ} = 0.462$) was twice as high as the correlation for the DZ twins ($r_{DZ} = 0.242$), which suggested the contribution of additive genetic effects. Greater age-adjusted, within-pair correlations were also observed in the MZ compared with DZ twin pairs for handgrip strength ($r_{MZ} = 0.435$, $r_{DZ} = 0.345$) and ankle plantar flexion strength ($r_{MZ} = 0.512$, $r_{DZ} = 0.435$).

Univariate genetic analysis. Table 2 shows the variance-covariance matrices for the MZ and DZ twin pairs, which were the basis for the univariate and multivariate genetic analyses. Table 2 also shows the Pearson’s correlation coefficients between the different measures of strength. The results of the univariate genetic analysis are shown in Table 3. For handgrip and ankle plantar flexion strength, the CE model showed the best fit for the data. Shared environmental effects accounted for 40% [95% confidence interval (CI): 27–51%] of the total variance in handgrip strength and 47% (95% CI: 36–58%) of the variance in ankle plantar flexion strength, whereas the remaining variance was due to nonshared environmental effects. For knee extension strength, the most parsimonious model was the AE model, in which additive genetic effects accounted for 46% (95% CI: 31–59%) of the variance, with the remaining variance being due to nonshared environmental effects.

Table 1. Physical characteristics and results of strength measurements of monozygotic and dizygotic twin individuals

<table>
<thead>
<tr>
<th>Variables</th>
<th>MZ</th>
<th>SD</th>
<th>DZ</th>
<th>SD</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>68.0</td>
<td>3.5</td>
<td>68.8</td>
<td>3.1</td>
<td>0.10*</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>69.3</td>
<td>11.5</td>
<td>70.4</td>
<td>11.9</td>
<td>0.43</td>
</tr>
<tr>
<td>Height, cm</td>
<td>158.2</td>
<td>6.4</td>
<td>159.0</td>
<td>5.7</td>
<td>0.32</td>
</tr>
<tr>
<td>BMI, kg/cm²</td>
<td>27.8</td>
<td>4.7</td>
<td>27.9</td>
<td>4.7</td>
<td>0.86</td>
</tr>
<tr>
<td>Handgrip strength, N</td>
<td>190.4</td>
<td>61.5</td>
<td>192.2</td>
<td>52.3</td>
<td>0.79</td>
</tr>
<tr>
<td>Knee extension strength, N</td>
<td>297.8</td>
<td>81.4</td>
<td>285.3</td>
<td>81.8</td>
<td>0.20</td>
</tr>
<tr>
<td>Ankle plantar flexion strength, N</td>
<td>221.1</td>
<td>87.2</td>
<td>217.9</td>
<td>79.7</td>
<td>0.76</td>
</tr>
</tbody>
</table>

Values are means, SDs and P values for equality of means and SD; n = 194 for monozygotic (MZ) and 204 for dizygotic (DZ). BMI, body mass index. *r-Test; n = 97 and 102; P < 0.05.
Multivariate genetic analysis. Because the results of the univariate genetic analysis showed the nonadditive genetic effects (D) to be nonsignificant, it was not taken into account in the multivariate genetic analysis. The analysis was started with the hypothetic full independent pathway model, including all plausible parameters ($\chi^2 = 31.61$, degrees of freedom = 24, $P = 0.137$, Fig. 2). Because several coefficients were statistically nonsignificant, the full 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 ($\chi^2 = 31.93$, df = 30, AIC = $-28.07$, $P = 0.371$, Fig. 3). In the final model, a common additive genetic effect (Ac) accounted for 14% (95% CI: 4–28%) of the variance in handgrip strength and 31% (95% CI: 18–45%) of the variance in knee extension strength. Adding the path from common additive genetic effect to ankle plantar flexion strength suggested a minor and not significant effect and was thus not included in further analyses. A shared environmental effect in common, Cc, explained 48% (95% CI: 36–58%) of the variance in ankle plantar flexion strength, 10% (95% CI: 3–20%) in handgrip strength, and 12% (95% CI: 4–22%) in knee extension strength. In addition, handgrip strength had its own specific shared environmental effect, Cs2, which accounted for 18% (95% CI: 5–30%) of the variance. The nonshared environmental effect in common, Ec, accounted for 6–23% of the total variance in ankle plantar flexion, handgrip, and knee extension strength. In addition, each strength measure had its own specific nonshared environmental effect (Es1, Es2, Es3), explaining 29–52% of the total variance. The specific nonshared environmental effect (Es1) for ankle plantar flexion strength was statistically nonsignificant, but it was left in the final model because it was theoretically believable and at the same level as the nonshared environmental effect for handgrip and knee extension strength.

**DISCUSSION**

The main result of the multivariate genetic analysis performed in this study was that maximal isometric handgrip and knee extension strength shared a genetic component in common, which accounted for 14% of the variance in handgrip strength, and 28% of the total variance. This finding suggests that genetic factors play a role in the variability of these strength measures, and that the use of a multivariate approach can help to disentangle the genetic and environmental contributions to these traits. Further research is needed to explore the specific genetic and environmental factors underlying these correlations, and to identify potential targets for interventions to improve strength and performance.
strength and 31% in knee extension strength. The effect of the genetic component on ankle plantar flexion strength was minor and not significant. In addition, handgrip, knee extension, and ankle plantar flexion strength had both a shared environmental effect and a nonshared environmental effect in common. To the best of our knowledge, this is the first study to include strength measurement results of multiple muscle groups in the same multivariate genetic model.

Fig. 2. The full hypothetical independent pathway model for ankle plantar flexion, handgrip, and knee extension strength. The coefficients shown are standardized parameters (95% confidence interval) of Ac, As1, As2, and As3, Cc, Cs1, Cs2, and Cs3, and Ec, Es1, Es2, and Es3. In this model, several parameters are statistically nonsignificant.

Fig. 3. The most parsimonious model explaining the genetic and environmental effects in ankle plantar flexion, handgrip, and knee extension strength. The coefficients shown are standardized parameters (95% confidence interval) of Ac, Cc, and Ec. In addition, the model includes Cs2 and Es1, Es2, and Es3. The common additive genetic effect accounted for 14% of the variation in handgrip strength and 31% in knee extension strength. The common shared environmental effect explained 48% of the variance in ankle plantar flexion, 10% in handgrip, and 12% in knee extension strength. The common nonshared environmental effect accounted for 23% of the variance in ankle plantar flexion, 6% in handgrip, and 17% in knee extension strength.
Our results indicate that handgrip and knee extension strength are measures under the control of the same genetic component. The association of genes with muscle characteristics is presently under intensive investigation. An effect on muscle has been suggested for several gene polymorphisms. For example, in type I collagen $\alpha_1$ spl polymorphism, the presence of the s allele was associated with lower muscle strength among women over 70 yr of age (34), whereas genotype G/A of the ciliary neurotrophic factor was associated with greater muscle strength, power, and muscle quality compared with the G/G genotype across the adult age span (25).

Also, a polymorphism of the IGF-II gene has been observed to have an effect on adult handgrip strength among men (28). In addition, it has been shown that genetic variation in the angiotensin-converting enzyme gene is associated with muscle strength and training response in some studies (8, 17, 35) but not all (9, 33). The results of the present study imply also that, in older people, muscle strength is most likely regulated by multiple genes, and it may be necessary to measure strength from multiple muscle groups to better capture the entire genetic variability in the trait.

Despite the relatively high MZ correlation in this study, there was no additive genetic effect in the handgrip strength in the univariate analysis. However, the multivariate genetic analysis of the handgrip strength showed a moderate additive genetic effect (14%). The difference between the results of the univariate and multivariate genetic analysis could be a consequence of a low statistical power in the univariate analysis to discriminate the genetic effect from the shared environmental effect. The multivariate analysis, which takes into account all available information, increases the statistical power of the analysis. Consequently, the multivariate modeling in this study resulted in a statistically significant, although moderate, additive genetic effect in handgrip strength. In the present study, the MZ correlation (0.435) was less than twice the DZ correlation (0.345) of handgrip strength, indicating the influence of shared environmental effect. However, the correlations are approximately at the same level as in previous studies, in which the correlation has varied between 0.41 and 0.76 (1, 6, 10, 24) for MZ and between 0.31 and 0.58 (1, 6, 24) for DZ twins. In the large population-based study by Frederiksen et al. (10), the MZ correlations for handgrip were comparable to our results, whereas the DZ correlations were clearly at a lower level (0.15–0.18) than in the present study.

In the present study, the multivariate genetic analyses were carried out by using the independent pathway model. With application to the present data of an alternate multivariate genetic model, the Cholesky decomposition model produced similar results. Previous studies have evaluated the heritability of muscle strength by using univariate genetic models. Our results indicated a somewhat more moderate genetic influence on muscle strength than the previous studies, in which the genetic component explaining the variability in handgrip strength has ranged from 22% (6) to 52% (10). The differences may, at least partially, be explained by differences in environmental variability, measurements used, and modeling approaches. To the best of our knowledge, no previous studies have investigated the genetic contribution to variability in maximal isometric muscle strength of knee extension or ankle plantar flexion, but two previous studies have investigated strength-related traits. Genetic effects accounted for 56% of the variability in chair stand performance among older male twins (5) and 46% in leg extension power among older female twins (1). No earlier studies on the heritability of calf muscle strength were found. In the present study, the CI values around the genetic effect on the handgrip and knee extension strength estimates were rather narrow, indicating a low margin of error in the coefficients.

The minor, statistically nonsignificant genetic contribution to ankle plantar flexion strength could be partly explained by the limited power of our study to detect minor genetic effects. Another potential explanation may be differences in the repeatability of different strength measurements. The coefficient of variation between two consecutive measurements was 6.1% in handgrip, 6.3% in knee extension (22), and 15.9% in ankle plantar flexion strength. Ankle plantar flexion may have been a more unfamiliar movement for the participants than knee extension or squeezing with the hand, thereby causing the greater variability in performance between repeated measurements. However, it is unlikely that this had a major effect on the results. The measurement error increases the relative contribution of the nonshared environmental effects in the model. In the present study, the contribution of nonshared environmental effects did not differ between the strength measurements.

Heritability is a characteristic that also depends on environmental variability and may thus differ between populations and age groups. In general, the methods of measurements used are a further potential cause affecting heritability estimates. In women, for example, menopause, HRT, and better survival into old age may cause more environmental variability, leading to a lower genetic contribution to a trait than in men. It has previously been observed that the relative contributions of genetic and environmental effects to the variability of strength can change over the years. For example, in the study by Carmelli and Reed (6), the heritability of handgrip strength decreased during a 10-yr follow-up, from 35 to 22%, whereas the shared environmental effects increased from 39 to 45% in male twins aged, on average, 63 yr at the baseline.

In the present study, the role of environmental effects in explaining variability in muscle strength was significant. Handgrip, knee extension, and ankle plantar flexion strength had a shared environmental component in common. The requirement of both individuals of the pair to participate in the study may have resulted in the exclusion of more DZ pairs than MZ pairs due to the poor health or mobility of one individual only. Consequently, the DZ pairs in the present study may be less discordant for muscle strength than what the DZ pairs generally are. The overestimation of the twin similarity increases the apparent size of the shared environmental effect and at the same time decreases the proportion of the genetic effect. Another explanation for this common shared environmental effect may be that family members resemble each other in physical activity so that either active or sedentary lifestyles accumulate in the same families (for a review, see Refs. 3, 4). The adoption of a physically active lifestyle in childhood may track over into a physically active lifestyle even in old age (12). Furthermore, evidence has started to emerge suggesting that even prenatal environment and early growth may have an effect on strength in older age. A recent British study showed that birth weight was associated with grip strength among men aged 64–74 yr (28).

In this study, a nonshared environmental effect was found in common for handgrip, knee extension, and ankle plantar flex-
ion strength. In addition, handgrip, knee extension, and ankle plantar flexion strength had their own specific nonshared environmental effects. The contribution of nonshared environmental effects to all three strength measurements was almost the same (52–58%). It has previously been shown that the presence of diseases, such as coronary heart disease, diabetes, or pulmonary disease, is associated with poorer strength (23). In the present relatively healthy study population, almost equal numbers of discordant MZ and DZ twin pairs were observed for coronary heart disease, hip osteoarthritis, knee osteoarthritis, and diabetes, as well as HRT. Diseases, such as diabetes and heart diseases, may have a general effect across all muscle groups, whereas osteoarthritis may affect only the strength of the muscles around the unhealthy joint. Also, factors other than diseases may contribute to nonshared environmental effects. These include, for example, individual living conditions and habits, work exposures, and other factors affecting only one sister. Frederiksen et al. (10) found that excluding subjects with chronic diseases increased the heritability estimate from 0.52 to 0.62.

This study only concerned female twin pairs, which limits the generalizability of the results. However, it is particularly important to study muscle strength in women due to the greater familial effects, thereby potentially placing them at an increased risk of disability in old age.

ACKNOWLEDGMENTS

The content of the manuscript does not represent the opinion of the European Community, and the Community is not responsible for any use that might be made of the information presented in the text.

GRANTS

This study was supported by grants from the Ministry of Education, the Academy of Finland, the Yrjö Jahnsson Foundation, and the Juho Vainio Foundation. E. Heikkinen and T. Rantanen are participants in the Burden of Disease in Old Age (BURDIS) network, which is a project within Key Action 6, The Ageing Population and Their Disabilities, of the European Union. The content does not represent the opinion of the Community, and the Community is not responsible for any use that might be made of the information presented in the text.

REFERENCES


