In old age, cognitive and physical functions are correlated. Knowing the correlations between genetic and environmental influences underlying this correlation can help to clarify the reasons for the observable (phenotypic) correlation. We estimated these correlations in a sample of 1,053 pairs of twins from the Longitudinal Study of Aging Danish Twins. Cognitive function was measured using forward and backward digit span, immediate and delayed memory, and fluency tasks. Physical function was measured using self-report of ability to carry out physical activities including walking, running, and climbing stairs. The phenotypic correlation between latent variable representations was .46 (95% confidence interval 0.27–0.65). The genetic correlation was .56 (95% confidence interval 0.15–1.00) and the nonshared environmental correlation .48 (95% confidence interval 0.35–0.61).

We discuss several ways of interpreting these correlations.

Key Words: Genetic and environmental correlation—Late-life cognitive ability—Physical fitness.

In old and even middle age, physical and cognitive functions are correlated (Carlson et al., 1999; Deary, Whalley, Batt, & Starr, 2006; Malmström, Wolinsky, Andresen, Miller, & Miller, 2005; Tabbarah, Crimmins, & Seeman, 2002), but the reasons for their correlation are not understood. Impairments of both physical and cognitive functions with old age are major sources of economic expense, anxiety, and deterioration in quality of life, not only for the elderly people themselves but also for their younger family members who often end up supporting them. Understanding the ways in which the physical–cognitive function correlation emerges would be a fundamental advance in aging research because we could then identify new, more likely successful approaches to minimize the impairments associated with old age, thus reducing the social burden this deterioration presents.

The correlation between physical and cognitive functions may arise for at least three reasons. First, it may occur because deteriorations in physical function due to incipient or manifest illness also undermine cognitive function. For many conditions common in old age such as cardiovascular disease and diabetes, this is clearly the case (Cosway, Strachan, Dougall, Frier, & Deary, 2001; Hassing, Grant, et al., 2004; Hassing, Hofer, et al., 2004; Henderson, Allen, Deary, & Frier, 2003; Price et al., 2006; Rafnsson, Deary, Smith, Whiteman, & Fowkes, 2007; Rafnsson, Deary, Smith, Whiteman, & Rumley, et al., 2007; Schram et al., 2007; Strachan, Deary, Ewing, & Frier, 1997; Strachan, Frier, & Deary, 2003), and the effects can be moderate (e.g., correlations on the order of −.4 between cognitive function and duration of diabetes controlling for age; Cosway et al., 2001) or even large (e.g., correlations on the order of −.6 with the removal of outlying data points; Cosway et al., 2001). We are beginning to have some understanding of the mechanisms involved in the contribution of physical illness to deterioration in cognitive function: for example, many chronic disease conditions are associated with increasing levels of proinflammatory markers (Ferrucci et al., 2005), which in turn contribute to cognitive decline (Rafnsson, Deary, Smith, Whiteman, Rumley, et al., 2007; Schram et al., 2007).

This situation, however, may not completely explain the correlation. A second possibility is that the relatively stable lifetime trait of cognitive function may act to support lifestyle choices and the development of skills that maintain or underpin physical function. Thus, lower mental test scores in childhood are associated with earlier mortality and excess morbidity in later life (Hart et al., 2003). Third, perhaps neither physical nor cognitive functions exert causal influences on the other: Each might be affected by more general processes of bodily aging. Evidence for these two factors suggests a common cause hypothesis of aging (Baltes & Lindenberger, 1997; Christensen, Mackinnon, Korten, & Jorm, 2001; Li & Lindenberger, 2002; Lindenberger & Baltes, 1997).

Studies that can distinguish among these possibilities are rare because the possibilities are not mutually exclusive. An exception was a report in which good physical function did appear to have some specific effect in maintaining good cognitive function, as it was shown to contribute 3.3% of the variance in cognitive ability at age 79 after controlling for cognitive ability in childhood (Deary, Whalley, et al., 2006). Although this may sound small in terms of accounting for variance, if real it is larger than many effects generally considered very important. For example, variation in possession of an e4 allele of the APOE gene accounts for...
ences predominate in explaining change or deterioration in relatively important to initially measured levels of both cognitive and physical functions in old age, in that the absence of physical deterioration also appeared to be associated with absence of deterioration of cognitive function. The result is important because the control measure of baseline cognitive ability was assessed in childhood and clearly was not affected by cognitive aging, although even here it is still possible that relative cognitive decline in later life preceded and contributed to physical decline in some individuals in the sample. The data necessary to make this kind of control are rare, however, so other approaches must be used to understand the etiology of the correlation between cognitive and physical function in old age.

Although it cannot distinguish among the three possibilities, the multivariate twin study can provide unique insights into the possible associations between physical and cognitive functions by providing fundamental information about the extent to which there may be genetic and shared and nonshared environmental influences common to the two domains of function. This is commonly done through estimation of the genetic and environmental correlations. For example, in related domains, Svedberg, Bardage, Sandin, and Pedersen (2006) estimated the genetic and environmental correlations between self-rated health and risk factors such as pain, lack of exercise, smoking, and obesity. They concluded that there were genetic and environmental influences common to self-rated health and some of the risk factors, indicating that individuals make behavioral choices that have long-term effects on self-rated health. In another example, Ortega-Alonso et al. (2006) examined the genetic and environmental correlations between walking speed and endurance in older women. Although genetic influences were relatively minor for these two traits, they were completely correlated. In contrast, the more important shared and nonshared environmental influences were much less correlated. This hinted at a common physiological basis but much more independent environmentally derived influences.

There has also been much important work describing the developmental patterns of genetic and environmental influences on cognitive and physical functions in late life separately using in particular the Swedish Adoption/Twin Study of Aging (e.g., Finkel, McArdle, Reynolds, & Pedersen, 2007; Finkel, Pedersen, et al., 2003; Finkel, Reynolds, McCardle, Gatz, & Pedersen, 2003) and the Longitudinal Study of Aging Danish Twins (LSADT; Christensen, Fredriksen, Vaupel, & Megue, 2003; Christensen, Gaist, Vaupel, & McGue, 2002; McGue & Christensen, 2002). In general, this work suggests that genetic influences are relatively important to initially measured levels of both cognitive and physical function but that environmental influences predominate in explaining change or deterioration in function with increasing age. To date, however, the extent to which the same genetic and environmental influences may contribute to levels of cognitive and physical function in old age has not been reported. Moreover, the extent to which the same genetic and environmental influences may contribute to their developmental patterns in old age has not been explored. Addressing these issues would help to clarify the degree to which the cognitive and physical impairments common in old age result from normal processes of aging or increasing vulnerability with age to chronic illness. The first step in this process is to estimate the genetic and environmental correlations between physical and cognitive functions in late life.

We measured these correlations using intake participants from the LSADT (Christensen, Holm, McGue, Corder, & Vaupel, 1999), one of the very few data sets available to date that can provide such information.

**Methods**

**Participants**

LSADT is a cohort-sequential study drawn from the older cohorts of the Danish Twin Registry, which includes twins born in Denmark between 1870 and 1910, and same-sex pairs born in Denmark between 1911 and 1930 (Hauge, 1981; Holm, 1981). All twins were at least 70 years of age. Participation rates were 75%–80% across four recruitment waves. We made use of the 1,053 same-sex pairs (464 monozygotic [MZ], 619 dizygotic [DZ]) in which both participated at intake and at least one completed at least the physical fitness assessment. These pairs spanned the full birth-year range of the Danish Twin Registry. Most of them (94%) also completed the cognitive assessment. Among intake participants, the primary reasons for not completing either the physical fitness or cognitive assessments were that they were participating via proxy due to severe physical or cognitive impairment or refused to complete one assessment or the other, possibly out of fear of poor performance. Our sample is thus reasonably representative of relatively healthily aging Danish twins. This does not rule out the existence of chronic disease conditions that did not preclude task performance in the sample, but information about the extent to which such conditions were present in the sample was not compiled. Mean age for the participants we used was 76.00 years (SD = 4.62). They included 794 men and 1,312 women.

**Procedures and Measures**

The assessments were administered by approximately 100 interviewers employed by the Danish National Institute of Social Research, which has extensive experience in survey administration to elderly Danes (Kjoller, 1996; Platz, 1989, 1990). The interviewers completed a detailed training program 2 months prior to survey administration and were
closely monitored during the assessment period. Most assessments were conducted in the twins’ homes or other places convenient to the twins between February and April of the scheduled year and lasted 60–75 min. They included assessment of demographic background, medical status, physical and cognitive functioning, and depression symptomatology. Co-twins were assessed independently by different interviewers.

Twin zygosity was determined on the basis of a self-report questionnaire completed by members of the Danish Twin Registry when most of the twins were in middle age. The method used has been validated against methods based on genetic markers, with error rates of less than 5% (Hauge, 1981). Cognitive function was assessed using standardized scores on five individual cognitive measures selected to represent tasks that are sensitive to normative age changes but that can be briefly and reliably assessed by lay interviewers. The specific tasks used were developed for the LSADT. They included a fluency task called categories, which required the participant to name as many animals as possible in 1 min; forward and backward digit span; and immediate and delayed recall of a 12-item list. The immediate and delayed recall tests were separated by about 15 min, during which the participants completed other tasks not assessing cognitive ability (McGue & Christensen, 2001). All tests were intercorrelated; the average correlation was .34. As the two digit span scores and the two recall scores were more highly correlated with each other than with the others, we summed their standardized scores to form digit span and recall composites, as did McGue and Christensen.

Physical fitness was assessed using 11 items from the Avlund scale (McGue & Christensen, 2000; Avlund, Davidsen, & Schulz-Larsen, 1995), a reliable and well-validated (Schulz-Larsen, Avlund, & Kreiner, 1992) self-report measure that discriminates levels of functional abilities among community-dwelling elderly adults through questions about tiredness and the ability to carry out functional tasks independently or with assistance. The 11 items assess the ability to run, walk various distances in good and bad weather, climb stairs, get outdoors, do light and hard exercise, and lift and carry 5 kg. Principal axis factor analysis indicated a factor representing movement through space (eight items, Cronbach’s α = .94) and a factor representing lifting and carrying (three items, Cronbach’s α = .72). We formed composite variables representing each factor by averaging the respective item scores and standardized the resulting variables. Following this, we regressed the effects of age and sex from all five cognitive function and physical fitness variables to avoid overstatement of twin similarity (McGue & Bouchard, 1984).

Statistical Analyses

We used a standard quantitative genetic model to estimate genetic and shared and nonshared environmental influences on cognitive function and physical fitness and the extent to which these influences were correlated. The model relies on the assumption that the variances in cognitive function and physical fitness can each be allocated to independent components attributable to additive genetic (commonly termed “A”), shared environmental (termed “C”), and nonshared environmental (termed “E”) influences. The variance decomposition is based on the fact that MZ twins have the same genome, whereas DZ twins share only 50% of their segregating genes if there is no assortative mating. Shared environmental influences refer to effects of environmental factors such as long-lasting effects of their common rearing and adult experiences such as common religious faith, level of attained education, or shared activities that act to make co-twins similar. The model relies on the assumption that such effects are equally important to MZ and DZ twin similarity. Nonshared environmental influences contribute to trait variance but not to twin similarity. They account for environmental differences between co-twins and for measurement error.

In addition to allocating the variances in cognitive function and physical fitness to genetic and environmental influences, the model can be used to allocate the covariances between one twin’s level of cognitive function and the co-twin’s level of physical function in a directly analogous manner through what is termed a Cholesky decomposition. This allows for estimation of the extents to which common sources of genetic and shared and nonshared environmental influences may contribute to the two traits. It is usually accomplished through the calculation of the genetic and shared and nonshared environmental correlations between them (often termed rA, rC, and rE). These correlations range from −1 to 1 in the manner usual to correlations.

To implement this basic model in our data, we allowed the three cognitive function variables (fluency, digit span, and recall) and the two physical fitness variables (movement and carrying) to define latent variables representing these two constructs. We then used the Cholesky decomposition to estimate the links among the components of variance attributable to the genetic and shared and nonshared environmental forms of influence on these latent variables, in a form called a Cholesky factor model (e.g., Johnson, Bouchard, Segal, & Samuels, 2005; Johnson, McGue, & Iacono, 2005), which is distinguished from the more commonly used model referred to as a “Cholesky model.” When fitting a Cholesky model, links among all observed variables are estimated. When fitting a Cholesky factor model, only the links among the components of variance on the latent phenotypic variables are estimated. It is thus a multivariate version of the common factor model (Neale & Maes, in press). This has the advantage of minimizing the effects of measurement error and systematic variance unique to each variable by focusing the analysis on the variance common to the variables contributing to each factor. The model we used is shown in Figure 1. We estimated it using the raw
data file and maximum likelihood analysis as implemented in Mx (Neale, Boker, Xie, & Maes, 2001) as the data were approximately normally distributed. In contrast to many studies making use of models of this type, we did not attempt to constrain any of the paths to 0 and we report results from fitting the full model in the following. Our model thus includes estimates of even small effects.

**Results**

**Twin Similarity and Phenotypic Correlations**

We estimated twin correlations in two ways. First, we estimated standard double-entered Pearson correlations for MZ and DZ twins separately, considering only twin pairs with full data for the measures in question. Second, we estimated the correlations simultaneously using all available data. As shown in Table 1, the results were very similar, and because the MZ correlations were greater than the DZ correlations, all indicated the presence of genetic influences. Because the DZ correlations were greater than half the MZ correlations for the cognitive function variables, shared environmental influences were indicated. This was not the case for the physical fitness variables.

Table 2 shows the correlations between pairs of observed (phenotypic) variables and their cross-twin correlations, or the correlations between one trait in one twin and another trait in the other twin. The cognitive function variables were moderately correlated, whereas the physical fitness variables were strongly correlated. The correlations between the cognitive function and physical fitness variables would generally be considered small. They were, however, highly statistically significant ($p < .001$). The cross-twin correlations generally suggested the presence of common genetic influences on the two sets of traits because MZ cross-twin correlations were greater than DZ cross-twin correlations.

**Quantitative Genetic Modeling**

Figure 2 shows the parameter estimates and the 95% confidence intervals for the latent variable structural model resulting from fitting the model shown in Figure 1. Paths shown in Figure 1 but not in Figure 2 had parameter estimates of 0. This does not mean, in particular, that there were no genetic influences on fluency or movement. Rather, it means that the genetic influences on fluency were completely absorbed by the latent cognitive function variable.

![Figure 1. Model for one twin linking cognitive function and physical fitness. “A” refers to additive genetic influences, “C” to shared environmental influences, and “E” to nonshared environmental influences. The other twin was modeled similarly, and genetic influences on the two were linked 1.0 in monozygotic twins and .5 in dizygotic twins. Shared environmental influences were linked 1.0 in both.](https://academic.oup.com/psychsocgerontology/article-abstract/64B/1/65/619440)

<table>
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<tr>
<th>Standard Double-Entered Pearson’s Correlation</th>
<th>Correlations Based on Full Available Data</th>
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<td>MZ</td>
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<td>Fluency</td>
<td>.35</td>
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<td>Digit span</td>
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<td>Memory</td>
<td>.33</td>
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<td>Movement</td>
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*Note: MZ = monozygotic; DZ = dizygotic.*
and the genetic influences on movement were completely absorbed by the latent physical fitness variable. Genetic and shared and nonshared environmental influences accounted for 43% (e.g., $A^2 + C^2 + E^2$) from Figure 2), 17%, and 40% of the total latent variable variance of cognitive function, and 29%, 0%, and 71% of physical fitness.

The phenotypic (full) correlation between the latent physical and cognitive function traits was .46. The genetic correlation between the latent physical and cognitive function traits was .56. The nonshared environmental correlation was .48. All were substantial and significant. Because there was no variance in latent physical fitness attributable to shared environmental influences, it could have no shared environmental correlation with cognitive function. These results were not solely dependent on the latent variable modeling process. Although they showed considerable variation from measure to measure, the raw cross-measure phenotypic correlations showed evidence of similar levels of genetic correlation, as the MZ twin cross-trait correlations were generally higher than the DZ twin cross-trait correlations (Table 2). The presence of genetic and shared environmental influences on the variances unique to fluency, digit span, memory, and lifting as shown in Figure 2 indicates that the latent cognitive function and physical fitness variables did not account for all the systematic variance in the individual measures contributing to them. Because all the factor loadings were substantial, however, the latent variable variances can be considered to represent the core, underlying constructs of cognitive function and physical fitness, at least as tapped by the measures used.

A limitation of this study is the selection effect that could result from underrepresentation of cognitively impaired persons in our sample, due either to their inability to complete the cognitive assessment or to their refusal to attempt it. To examine the possible implications of this at the level of the full population, we reestimated the model parameters

![Figure 2](https://academic.oup.com/psychsocgerontology/article-abstract/64B/1/65/619440)

Figure 2. Parameter estimates from model linking cognitive function and physical fitness. “A” refers to additive genetic influences, “C” to shared environmental influences, and “E” to nonshared environmental influences. These parameters are squared to provide proportions of variance. Ninety-five percent confidence intervals for results of interest are given in parentheses. Only paths with non-zero parameters are shown.
including the participants who had refused the cognitive assessment but provided physical function data by assigning them very low arbitrary values for each of the cognitive measures. The results were very similar to those presented. Our results could also be affected by the inclusion of those with cognitive impairment. To examine the possible implications of this, we also reestimated the model parameters, dropping those scoring in the bottom 10% on any of the cognitive measures. This resulted in a somewhat higher genetic correlation of .83 and a nonshared environmental correlation of .41.

**DISCUSSION**

In summary, as hypothesized, we observed substantial correlations linking genetic influences on cognitive function and physical fitness in a large and representative sample of healthily aging Danish twins. We also observed substantial correlations linking nonshared environmental influences on the two traits. Together, the two observed correlations point in a novel way to the existence of aging processes that tend to undermine cognitive and physical function in tandem, and indicate that these processes arise through both genetic and environmental influences. The correlations also point to the possibility that environmental circumstances and lifestyle choices throughout life (that may also arise through genetically influenced characteristics) have consequences for both cognitive and physical function in late life.

Despite its strengths in terms of size and population representativeness of the sample, our study is subject to some limitations that should be considered in interpreting its findings. Most notably, the ambiguity regarding the extent to which the participants may have suffered from clinical cognitive impairment that could be categorized as dementia makes it difficult to be sure to what segment of the population our results apply. This is, however, a problem with all research involving elderly samples, and there is no reason to believe that our findings suffer more from it than would data from other samples. In addition, our measures of physical function were based on self-report rather than observations of actual functional capabilities, and our cognitive measures show greater evidence of shared environmental influences than do others in the literature for adult samples. It is not clear what specific effects on our results these limitations may have had.

The latent phenotypic correlation of .46 that we observed is higher than most of the phenotypic correlations that have been reported in the literature. These correlations, however, have been between individual measured variables, each of which is subject both to measurement error and systematic variance unique to that specific measure of function. The process of constructing our latent cognitive and physical function variables based on their covariances effectively removed both of the sources of unique variable variance, which would explain the higher correlation we observed. Our estimates should thus more accurately reflect the correlations between the core constructs of physical and cognitive functions, to the extent they were reflected in the measures we used.

These data parse the genetic and environmental links between physical and cognitive functions in old age. The substantial correlations that we measured can be interpreted along three lines. First, many of the diseases prevalent in late life such as cardiovascular disease and diabetes are both under genetic influence and have cognitive as well as physical manifestations involving functional limitations (Cosway et al., 2001; Henderson et al., 2003; Price et al., 2006; Rafnsson, Deary, Smith, Whiteman, & Fowkes, 2007; Rafnsson, Deary, Smith, Whiteman, Rumley, et al., 2007; Schram, et al., 2007; Strachan et al., 1997, 2003). The genes involved can be expected to contribute to both the physical and cognitive manifestations of disease as it emerges, thus creating the genetic correlation we observed. That is, the physical limitations and cognitive effects of such diseases emerge at least partly as a result of the expression of highly prevalent genetic vulnerabilities to particular diseases with multifaceted effects. Second, lifelong cognitive function is subject to substantial genetic influence (Bouchard & McGue, 1981; Deary, Spinath, & Bates, 2006). Because of this, to the extent that lifelong cognitive function contributes to the creation of healthy lifestyle that in turn benefits late-life physical function, the genetic influences on lifetime cognitive function will influence physical function as well and thus contribute to the emergence of a genetic correlation between them, particularly in later life.

Finally, there is evidence of genetic contributions to more general aging-related processes—such as oxidative stress and Hypothalamic-Pituitary-Adrenal axis dysregulation (Harris et al., 2007)—which might affect both physical and cognitive functions and thus contribute to the emergence of a genetic correlation in later life. Moreover, there is the possibility that some form of constitutional “soundness” may affect both cognitive and physical function throughout the life span (Prokosch, Yeo, & Miller, 2005). Although we cannot use the presence of the genetic correlation alone to distinguish among these interpretations, its presence reinforces the importance of lines of research exploring sources contributing to both cognitive and physical decline in old age to our developing understanding of the processes involved in aging and the functional limitations experienced by many elderly adults. In addition, the possibility that cognitive and physical functions may be genetically correlated throughout the life span should be investigated in younger samples. This could provide important information about the extent to which aging could reflect fundamental biological processes present from much earlier developmental periods or even from conception.

The similarity of the genetic and nonshared environmental correlations suggests that genetic and nonshared...
environmental developmental processes produce similar patterns of variation that contribute to both. Where personal choice is involved in these processes, such as in education and lifestyle factors influencing health, the nonshared environmental influences may be correlated with and act to support the genetic association. That is, the nonshared environmental influences contributing to the nonshared environmental correlation may be correlated with the genetic influences contributing to the genetic correlation. This would be the case if there are genetic influences on the particular nonshared environmental influences that contribute to both physical and cognitive functions in old age. Such a gene–environment correlation would occur if the personal choices contributing to life circumstances of the aging individual were also subject to genetic influence (Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990).

Regardless of whether this possibility is true, these results also point to the importance of the nonshared environmental influences linking these two crucial human processes. Possible contributors to the substantial nonshared environmental correlation we observed include health and lifestyle choices such as diet and exercise, and drinking and smoking that may also be influenced by lifelong cognitive function, as well as experiences such as occupational stress and injury, marital instability, financial difficulties, and problems involving family relationships. That is, any environmental circumstance that contributes to any of the three lines of interpretation of the genetic correlation outlined previously may contribute to the nonshared environmental correlation we observed. Of course, these same factors may also link the genetic and nonshared environmental sources of influence themselves, creating the kind of mutual support between the genetic and nonshared environmental influences creating the correlations referred to previously. This should be pursued in future investigations of purely environmental effects, as well as in investigations of gene–environment interplay such as gene–environment correlation and interaction (Johnson, 2007). The present study, therefore, contributes substantial and novel information on the sources of the physical–cognitive correlation in older people.

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CORRESPONDENCE

Address correspondence to Wendy Johnson, PhD, Department of Psychology, University of Minnesota, Twin Cities Campus, 75 East River Road, Minneapolis, MN 55455. Email: wendy.johnson@ed.ac.uk

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