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Genetic and Environmental Transactions Linking Cognitive Ability, Physical Fitness, and Education in Late Life

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Abstract

Cognitive ability and physical fitness are important to the ability to live independently in late life. Both are also related to level of attained education, with better educated older adults tending to display better cognitive ability and better late-life physical health. Chronic illnesses that affect both physical and cognitive function, lifetime cognitive ability that facilitates healthy lifestyle choices, and general biological aging processes have been offered as 3 explanations for the late-life physical–cognitive correlation. Education is generally assumed to provide a protective environment. The authors used a sample of 1,053 twin pairs aged 70 and over and gene–environment moderation models to explore 5 hypotheses that could help to disentangle the genetic and environmental transactions involving physical and cognitive functions and education. Results provide some support for all 3 explanations for the physical–cognitive correlation and indicate the ways in which better education may support better function and lack of education may undermine it.

Keywords

late-life cognitive ability; educational attainment; physical fitness; genetic and environmental influences

Cognitive ability and physical fitness are important to the ability to live independently in late life. They also tend to be correlated among older adults (Deary, Whalley, Batty, & Starr, 2006; Johnson, Deary, McGue, & Christensen, in press). Moreover, both are also related to level of education, with the better educated tending to display both better cognitive ability (Ceci & Williams, 1997) and better late-life physical health (Adler et al., 1994). Cognitive and physical impairments in old age are major sources of economic expense, anxiety, and

deterioration in quality of life for the older adults as well as for their younger relatives who often end up caring for them. Understanding the reasons for the cognitive–physical correlation and the roles played by education in supporting these two crucial life functions would be an important advance in aging research and could lead to new approaches to minimize deterioration in function and the burdens it creates in an aging society.

There are several reasons that cognitive and physical functions may be correlated in old age. First, researchers know that many of the chronic physical illnesses common in old age such as diabetes and heart disease have cognitive as well as physical consequences (Cosway, Strachan, Dougall, Frier, & Deary, 2001; 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); the development of the physical illness no doubt leads to declines in cognitive as well as physical function. To the extent that these illnesses are more common among those with less education, one might expect that education may act to preserve both cognitive and physical function in late life by providing resources to reduce the incidence or severity of these illnesses. At the same time, causation may flow in the other direction: The rather stable lifetime level of cognitive function (Bouchard, 1995; Deary, Whiteman, Starr, Whalley, & Fox, 2004) may contribute to the establishment of health and lifestyle habits that maintain both cognitive and physical functions with age. To the extent that this is true, education may play a similar role. In addition, there may be some form of constitutional integrity that is affected by general bodily processes of aging (Baltes & Lindenberger, 1997; H. Christensen, MacKinnon, Korten, & Jorm, 2001; Lindenberger & Baltes, 1997; Li & Lindenberger, 2002). One might expect education to have little effect on such a process, unless the constitutional integrity also acted to increase educational attainment, which usually takes place in youth to young adulthood.

These possibilities are by no means mutually exclusive, and distinguishing among them and/or evaluating their relative contributions is difficult. This is because the possibilities involve individual life choices both exerted and having consequences over long periods of time, as well as genetic predispositions that are manifested over similarly long periods of time. One approach that can help to clarify the processes involved is to examine the genetic and environmental transactions, or forms of interplay among genetic and environmental influences (Johnson, 2007), that contribute to the traits of interest and the associations among them. Cognitive and physical function and education all show genetic as well as environmental influences (Bouchard & McGue, 1981; Carmelli, Swan, & Cardon, 1995; K. Christensen, Frederiksen, Vaupel, & McGue, 2003; K. Christensen et al., 2000; McClearn et al., 1997; McGue & Christensen, 2001) in their own rights. In addition, however, they show genetic covariance (Bartels, Rietveld, Baal, & Boomsma, 2002; Carmelli et al., 1995; Johnson, McGue, & Iacono, 2006, 2007), suggesting that common genetic influences are important, at least to the pairs of traits. Though the existence of common genetic influences does not help to resolve questions regarding the directions and magnitudes of causal factors, it does establish that to some degree they operate in tandem at a level that is intrinsic to the individual—whatever association exists is not imposed completely by environmental circumstances.

The multivariate examination of genetic and environmental influences can be further extended in a way that begins to illuminate the nature of the causal structure of the genetic and environmental transactions involved. Most models evaluating multivariate genetic and environmental influences rely on the assumptions that genetic and environmental influences are independent of each other and constant for all members of the population under examination. There is increasing evidence that these assumptions are often violated (Johnson, 2007) and that the ways in which they are violated can be informative about the

underlying processes involved. One example relevant to cognitive and physical functions in old age involves genetic and environmental links between physical health and income. Johnson and Krueger (2005) found that genetic influences on chronic physical health problems were stronger in those with lower incomes, despite a smaller genetic correlation between physical health and income in low-income environments. In combination, these two observations of stronger genetic influences on physical health in low-income environments yet a smaller genetic correlation suggest that the low-income environment acted to increase expression of preexisting genetic vulnerabilities to illness that might have gone unexpressed in more favorable environments. These observations also suggest that the same genetically influenced individual characteristics that contributed to higher income also served to minimize expression of genetic vulnerabilities to physical illness in the higher income environment. These characteristics, however, were apparently less effective in preserving physical health in the low-income environment.

Other relevant examples involve changes in genetic and environmental influences on educational attainment in young people with level of cognitive ability. Johnson, Deary, and Iacono (2008) and Johnson, Deary, Silventoinen, Tynelius, and Rasmussen (2008) carried out similar investigations of the associations between these traits in samples in the United States and Sweden. They found substantial genetic and shared environmental influences common to educational attainment. With greater cognitive ability, genetic influences on educational attainment were more readily expressed in both Sweden and the United States. In contrast, shared environmental influences on educational attainment were much more important in the United States when cognitive ability was low but in Sweden when cognitive ability was high. In combination, these observations suggest that high cognitive ability had an effect on educational attainment similar to the effect of low income on physical health: The same genetically influenced individual characteristics that contributed to low cognitive ability also restricted educational attainment. At the same time, at least in the United States, shared environmental influences, probably in the form of family background, acted to compensate for the genetically influenced limitations when cognitive ability was low. This was not the case in Sweden, where shared environmental influences acted in concert with genetic influences.

In the present study, we made use of a population-representative sample of twins over age 70 to explore the genetic and environmental transaction processes linking physical fitness, cognitive function, and education in order to begin to distinguish among the three possible explanations for the correlation between cognitive and physical function in old age. To do so, we investigated five hypotheses. Figure 1 shows how these hypotheses are related to the proposed explanations for the association between cognitive and physical functions in old age. The hypotheses were as follows.

1. If chronic illnesses common in old age first create deterioration in physical function that then leads to deterioration in cognitive function, genetic and/or environmental influences on physical function should moderate genetic and/or environmental influences on cognitive function. To the extent that the process involved is similar to that linking income and physical health, lower physical function should be associated with greater variance in, or expression of influences on, cognitive function. There should also be substantial genetic correlation between physical and cognitive functions.
2. If genetically influenced chronic illnesses common in old age simultaneously create deterioration in physical and cognitive functions and/or if there is some form of general bodily aging that affects both forms of function, there should not be moderating effects of genetic influences either of cognitive function on physical

function or vice versa. There should, however, be substantial genetic correlation between the two forms of function.

3. If lifetime cognitive ability supports the development and maintenance of health and lifestyle habits that preserve both physical and cognitive functions in late life, genetic and/or environmental influences on cognitive function should moderate genetic and/or environmental influences on physical function, with lower cognitive function associated with greater variance in physical function. The way in which genetic and environmental correlations change with level of cognitive ability should help to clarify the way that cognitive function is involved in preserving physical function. If the correlations are high when cognitive function is low, then there is evidence that the common influences are more powerful in bringing about deterioration in function. If the correlations are high when cognitive function is high, then the reverse is the case: There is evidence that the common influences are more powerful in protecting against deterioration in function.
4. If education provides resources to prevent the incidence or minimize the severity of chronic illnesses that affect either or both physical and cognitive functions, it should moderate the genetic and/or environmental influences on particularly physical function so that it shows less variability when education is high. This would also mean greater genetic and/or environmental correlation between education and physical functions when education is high.
5. If the effects of education are similar to those of higher cognitive ability in facilitating the development and maintenance of health and lifestyle habits that preserve either or both physical and cognitive functions in late life, there should be moderating effects of education on the genetic and environmental influences on cognitive function, but their direction may vary. If education has greater power to inspire better health and lifestyle habits than current cognitive function, then there should be less variability in cognitive function when education is high. In this case, correlations between education and cognitive function should be greater when education is high. On the other hand, if lack of education is more important in creating poor health and lifestyle habits, then there should be less variability in cognitive function when education is low. In this case, correlations between education and cognitive function should be greater when education is low.

To investigate these hypotheses, we made use of a model that was developed by Purcell (2002) to quantify the genetic and environmental influences on a particular trait explicitly in the context of correlation with and moderation by genetic and environmental influences on an associated trait. It is only one of many possible models of the kind of gene–environment interplay in which we were interested, and we used it in a way that detected only linear moderating effects. Still, the results of this model can extend researchers' understanding of the underlying processes involved in the associations in which we were interested in the same way that photographing a building from the front provides considerable information about its structure. Though photographing it from overhead may provide very different information that was missed in the frontal photograph, this rarely invalidates the information provided by the frontal photograph. Moreover, most nonlinear effects are at least captured by linear models so examination of linear effects would appear to be the place to begin this kind of investigation. To show how the model works, we define several quantitative genetic concepts and describe how they form the foundation for the model we used. We begin by describing the much simpler standard quantitative genetic model for a single trait, using cognitive function as an example. As we add description of bivariate models, we use education as an example of the other variable involved. In our actual analyses, however, we considered several possible combinations of cognitive and physical function and education related to our hypotheses.

The model relies on the idea that the phenotypic (observed) variance (V_p) in a trait is a linear function of additive genetic (A^2) and shared (C^2) and nonshared (E^2) environmental variance components ($V_p = A^2 + C^2 + E^2$). Shared environmental variance components reflect influences that act to make cotwins similar to each other but which distinguish among twin pairs. Most of the classic examples of shared environmental influence involve childhood experiences such as family socioeconomic status and parental efforts to equalize educational opportunities for their offspring. Examples in later life are more difficult to develop but could include life-long effects from childhood or shared cultural influences such as religious practices or dietary habits. Nonshared environmental variance components include measurement error, but they also reflect influences that act to make cotwins different from each other or at least fail to make them similar. Important examples might be different career experiences, different levels of experience with travel abroad, and spouses or partners with very different characteristics. The distinction between shared and non-shared environmental influences can be subtle. For example, twins may have experienced the same event (e.g., military service), but that event is only a shared environmental influence to the extent that it made the twins similar on the cognitive function we measured later. To the extent that the twins reacted to this event differently in ways that affected later cognitive function, the event is a nonshared environmental influence. This standard model for a single variable for a single twin is generally depicted as shown in Figure 2.

Under this model, we assumed that each of the variance components is both constant and independent of the others throughout the represented population. Moreover, the model makes no provision for the possibility that the influences on cognitive ability are related to those on any other trait, in particular education or physical function. As we were interested here in exploring possible associations of the genetic and environmental influences on cognitive function with genetic and environmental influences on education and physical function, we required that these assumptions be relaxed, and some provision be made for associations between genetic and environmental influences on cognitive function and education or physical function. To do this, we used an adaptation of the model that allowed us to estimate both the extent to which genetic and environmental influences on one trait were also involved in another and the extent to which that trait might exert moderating effects on the components of variance in the other. It was necessary to do this for pairs of variables due to software limitations in parameterizing all three variables at once in this manner.

Beyond the possibility of examining transactions between genetic and environmental influences on two variables, the moderating model we used introduces the potential to develop evidence for causal interpretations involving the specific kinds of genetic and environmental transactions revealed. Moderation of the variance components of one variable such as cognitive function with level of some other variable such as education or physical function is an example of a gene–environment interaction. A gene–environment interaction can also be thought of as differential genetic expression in different environments or as differential genetic sensitivity to the environment. Complementing a gene–environment interaction, substantial genetic influence common to two traits is an example of genetic correlation. This can also be thought of as genetically influenced exposure to different environments. There can be analogous moderating effects and correlations for shared and nonshared environmental influences. Genetic and environmental correlations range from -1 to 1 in the manner usual to correlations.

As described by Purcell (2002), we adapted the standard quantitative genetic model shown in Figure 2 to include provision for these phenomena in two ways. First, we introduced the second variable, for example, education, along with paths that allowed for influences common to education and cognitive function as well as influences unique to cognitive

function alone. And second, we explicitly measured the possibility that the variance components unique to cognitive function varied as continuous linear functions of education. We also explicitly measured the extent to which education moderated whatever influences were common to education and cognitive function. The adapted model is shown in Figure 3. The order of the variables is important: When we assigned one variable to be the moderator and the other to be the primary trait, we were electing to examine the effects of the moderator on the primary trait and not vice versa, yet this choice can be arbitrary. In many situations, actual moderating effects could act in either or both directions, and the direction of effects is important in understanding the underlying processes. In the particular case of education and cognitive function in this older adults sample, however, it made sense to consider only education's moderating effects on cognitive function, as cognitive function reflected performance capacity at the time of the assessment, while education was generally attained well before the assessments. With respect to cognitive and physical functions, however, the situation is not as straightforward. We reasoned that it was just as likely that genetic and environmental influences on cognitive function might moderate those on physical function as vice versa. For these two traits, therefore, we considered moderating effects in both directions.

Under the example model, the paths expressing the genetic and environmental influences on education (indicated as a triangle to emphasize its distinctive moderating role) are considered constant as in Figure 2. Though of course we measured them, we did not explicitly label these paths in Figure 3 in order to keep the focus on the paths involving moderating effects. The paths that are labeled in the figure thus express the genetic and environmental influences on cognitive function. These paths are all linear functions of the form $x + (b \times M)$, where x and b are regression coefficients and M is the level of education. In Figure 3, there are two sets of paths contributing genetic and environmental influences to cognitive function to reflect the two possible kinds of moderating effects we measured: those common to education as well, and those unique to cognitive function. The ways in which education could moderate the variance in cognitive function was measured by six paths, but we also tested the importance of the moderating effects overall by assessing model fit when all six moderating effects were constrained to zero. The resulting best fitting model thus provided estimates of the genetic and environmental influences common to education and cognitive function and the extent to which these varied with education, and estimates of the genetic and environmental variances unique to cognitive function and the extent to which these varied with education. Estimates of the genetic correlation and the environmental correlations were the square roots of the proportions of the total influences on cognitive function that were common to it and education. In most bivariate models of genetic and environmental influences, these correlations are constant in the same manner as are the variance components in Figure 2. When education significantly moderated the genetic or environmental influences on cognitive function, however, the corresponding genetic or environmental correlations could also vary as functions of education. We used these moderating and correlation effects to test our five hypotheses.

Method

Sample

Participants were twin pairs from the intake groups of the Longitudinal Study of Aging Danish Twins (LSADT; K. Christensen, Holm, McGue, Corder, & Vaupel, 1999; McGue & Christensen, 2007). LSADT is a cohort-sequential study that began in 1995. The sample was 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, 1983), based on residency and survival status determined just prior to the time of recruitment from the Danish Central Person Registry. The initial cohort

assessed in 1995 was born prior to 1920 and thus was at least 75 years of age. They were reassessed at 2-year intervals. Additional cohorts were added at the 1997, 1999, and 2001 assessments. Twins born between 1920 and 1923 (thus at least 73 years of age) were added in 1997, twins born between 1924 and 1928 (thus at least 70 years of age) were added in 1999, and twins born between 1929 and 1930 (thus at least 70 years of age) were added in 2001. Participation rates ranged from 75% to 80% across the four waves. More complete details of the sample ascertainment procedures were given by K. Christensen et al. (1999). They also reported a comparison of participants and nonparticipants in the 1995 survey. The survey indicated that participants were slightly but significantly more likely than nonparticipants to be male and to have been hospitalized between 1977 and 1994. The two groups did not, however, differ significantly from each other in terms of age or twin zygosity. Though the LSADT as a whole is longitudinal, for these analyses we did not make use of its longitudinal features.

In total, 4,731 individual twins completed an LSADT intake assessment over the four waves of assessments, though some of the interviews were completed by proxy. Twins were recruited for the study without regard to cotwin survival status, so the total sample included many incomplete pairs even at intake. Among intake participants, there were 2,304 individual twins from 1,152 pairs, and 2,427 single twins. For this study, we made use of the 1,053 (464 monozygotic [MZ], 619 dizygotic [DZ]) pairs, both of whom participated at intake and at least 1 of whom completed at least the physical fitness assessment. Most of these (94%) also completed the cognitive assessment. Among intake participants, the primary reason for not completing either the physical fitness or cognitive assessments was that they were participating via proxy due to severe physical or cognitive impairment. In these cases, a spouse, relative, or close friend answered interview and survey questions on behalf of the participant, and the participant completed only those functional assessments of which he or she felt capable. Additionally, a few participants did not complete the cognitive assessment likely due to concerns about poor performance. Our sample thus underrepresented individuals with significant physical or cognitive impairments. This was reflected in the .25 standard deviation difference in cognitive ability favoring those whose cotwin was a study participant over those who participated as single twins, though this difference also reflected mortality. There were analogous differences of 0.31 standard deviation in physical fitness and 0.07 standard deviation in education. Mean age for the participants we used was 76.00 ($SD = 4.62$). They included 794 men and 1,312 women.

Procedure

Most of the assessments were completed in the participants' primary residences between February and April of the scheduled year. They were carried out by about 100 interviewers from the Danish National Institute for Social Research, which has substantial experience interviewing older adults (Kjoller, 1996; Platz, 1989, 1990). Many of the same interviewers participated in multiple assessment waves. All completed a detailed training program in the 2 months prior to the survey period and were closely monitored during it. Cotwins were separately interviewed by different interviewers. The interviews lasted 60–75 min and included assessments of background demographics, physical health, functional status, cognitive ability, and depression symptomatology.

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% (Christiansen et al., 2003; Hauge, 1981).

Measures

Cognitive ability—We assessed cognitive ability using the Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975) and five individual cognitive measures. For this study, we were interested in measures that reflected prime of life as well as current general cognitive ability rather than specific aspects of cognitive ability or presence of later life cognitive impairment per se. We thus used a composite of the five individual cognitive scores and did not use the Mini-Mental State Examination scores. We selected the cognitive components assessed to represent tasks that are sensitive to normative age changes but that can be briefly and reliably assessed by lay interviewers. Specifically, the tasks included a fluency task, forward and backward digit span, and immediate and delayed recall of a 12-item list. The fluency task required the participant to name as many animals as possible in a 1-min interval. The intercorrelations among the individual components ranged from .31 to .46 (McGue & Christensen, 2001). We calculated the cognitive composite by summing the five standardized components. Our measure of general cognitive ability thus heavily emphasized memory performance in general yet did not distinguish among specific aspects of memory. At the same time, it was much more sensitive to age-related changes than many measures of general cognitive ability that have been used in other studies, particularly those that use tasks such as word reading and vocabulary that largely reflect educational attainment.

Physical fitness—The study assessment in 1995 and 1997 included the Avlund instrument (Avlund, Davidson, & Schulz-Larson, 1995; K. Christensen et al., 2000; Schulz-Larson, Avlund, & Kreiner, 1992). This is a self-report measure that discriminates levels of functional abilities among community-dwelling older adults through questions about tiredness and the ability to carry out functional tasks independently or with assistance. This self-report approach has generally been found to be reliable and valid (Jette, 1987; Kane & Kane, 1981). The Avlund instrument was extended to include assessment of need for equipment or aids to carry out functional tasks, based on findings that such equipment can improve independent functional abilities in older adults (Manton, Corder, & Stallard, 1993). It also included all the items from the Katz index of Activities of Daily Living (Katz, Ford, & Moskowitz, 1963), along with questions about the abilities to see, hear, and carry out more demanding activities such as running.

The 26 items of the Avlund instrument were rated on a 1–4 scale, with 4 = *can do without fatigue*, 3 = *can do with minor difficulties*, 2 = *can do with aid or major difficulties*, and 1 = *cannot do*. They were factor analyzed in the original intake LSADT sample assessed in 1995. As reported by K. Christensen et al. (2003), a two-factor solution was developed. Substantial loadings on the first factor came from questions dealing with the ability to run, walk in good and bad weather, climb stairs, get outdoors, do light and hard exercise, and carry 5 kg. Substantial loadings on the second factor came from questions involving personal care such as the abilities to dress and wash oneself, use the toilet, get in and out of bed, remove and put on socks and shoes, and cut fingernails and toenails. Scales formed from the questions loading on these factors had internal consistency reliabilities of .93 and .91, respectively, for both men and women. They were correlated .77. The 1997 assessment yielded very similar results. In this study, we focused on the scale from the first factor, which we term *physical fitness*. Scores were the averages of the scores from the relevant questions and so ranged from 1 to 4. The scale from the second factor showed very little variance in the younger participants and was dropped from the assessment in 1999.

Education—The participants answered two questions with respect to education. Both were asked with respect to the household breadwinner during most of the participant's lifetime, so, particularly for women, the responses did not necessarily reflect the participant's actual

personal level of education. Because the sample contained more women than men and most participants of both sexes had married during their lifetimes, only about 43% of the participants were probably primary breadwinners. Still, participants from other birth cohorts of the Danish Twin Registry who reported level of education for both themselves and their spouses produced a spousal correlation of .58, suggesting that the questions about breadwinner education probably reflected also participants' levels of education pretty well. The first question asked about level of education in 2-year increments ranging from less than 7th grade to 11th grade or more. The second question asked about vocational education and included response categories ranging from none through *course for semi-skilled workers'* and *training as an apprentice* to number of years of theoretic and academic education in increments of 2 years ending with 4 or more. We combined the information in these two questions to form a scale ranging from 1 to 8, in which 1 = *less than 7th grade*, 2 = *completion of 7th or 8th grade*, 3 = *completion of 9th–10th grade and/or completion of a course for semiskilled workers*, 4 = *completion of 11th grade*, 5 = *completion of an apprentice program*, 6 and 7 = *intermediate levels of academic education beyond 11th grade*, and 8 = *completion of 4 or more years of academic education*.

Descriptive statistics for the variables used are shown in Table 1.

Data Adjustment and Analytical Implementation

The physical fitness variable was negatively skewed. To reduce this skew to reasonable levels, we squared it prior to analysis. The education variable was positively skewed. We thus log-transformed it prior to analysis to reduce this skew. The cognitive composite variable was very close to normally distributed, so we did not transform it. Age and sex effects contribute to twin similarity because twins are the same age and often of the same sex. In samples such as this one consisting of mixed ages and sexes, these effects may act inappropriately to inflate the similarity between cotwins (McGue & Bouchard, 1984). Moreover, we were not interested in examining change over time or the specific effects of age or gender within these data. We specifically wanted to treat the data as coming from an older adults sample without further regard to these demographic variables. Thus, to avoid distortions in estimates of genetic and shared environmental influences and to place all the data on the same level with respect to age and sex, we regressed the effects of age, age², sex, Age × Sex, and Age² × Sex from all of the (transformed as appropriate) variables prior to analysis.

We had small amounts of missing data, but the assumption that these data were missing at random was reasonable (Little & Rubin, 1987), given that completion of cognitive assessment was in no way predicated on performance on physical assessment or education or vice versa. We thus used maximum likelihood to fit our models to raw data. We implemented the quantitative genetic models in the computer program Mx (Neale, Boker, Xie, & Maes, 2003), specifying them as recommended by Purcell (2002). All variables were standardized to place them on the same measurement scale.

We allowed parsimony to dictate the results presented because of the complexity of the quantitative genetic models with which we worked. To do so, we dropped all terms indicating moderating genetic and/or environmental effects from the models when we could do so without significant change in model $-2 \times \log$ likelihood. We did this both sequentially for individual moderating effects and in groups of nonsignificant moderating effects, with identical results. We evaluated the appropriateness of our results using Akaike's information criterion (Akaike, 1983) and the Bayesian information criterion (Raftery, 1995), information theoretic fit statistics. Because they provide objective criteria for selecting models that minimize the amount of information required to express the data, these kinds of information-theoretic fit statistics make it possible to determine the most parsimonious or efficient

representation of the data. The point of dropping nonsignificant moderating terms was not to deny the potential existence of smaller moderating effects on the paths that happened not to be significant in this particular sample. Rather, it was to focus attention on the paths and thus the social forces that were most important in these data.

Results

Descriptive Statistics

Table 2 shows the zero-order correlations among the variables used in our study, as background information for the analyses that test our hypotheses. All of the correlations were significant at $p < .01$ even after we adjusted for the paired data, but the correlations between the cognitive composite and both physical fitness and education would at best be considered moderate, and the correlation between physical fitness and education would often be disregarded. The correlations between members of MZ and DZ twin pairs for the study variables are given in Table 3. Genetic influences are indicated when MZ correlations are greater than DZ correlations, and their relative magnitude can generally be approximated for a given variable as twice the difference between the two correlations (Falconer & Mackay, 1996). Shared environmental influences are indicated to the extent that DZ correlations are more than half the MZ correlations. Their relative magnitudes can be approximated for a given variable by doubling the DZ correlation and subtracting the MZ correlation (with a lower bound of zero). The effects of assortative mating (by the parents of these older adults twins) are the same as those of shared environmental influences, and the two could not be distinguished in these data. Nonshared environmental influences are indicated to the extent that MZ correlations are less than 1.00. All three of the cognitive composite, physical fitness, and education variables showed only small genetic and shared environmental influences, primarily because MZ twin correlations were relatively low compared to those from other studies. For the cognitive composite and education variables, DZ correlations were high enough to indicate substantial shared environmental influences or assortative mating.

Moderating Effects of Cognitive Ability on Physical Fitness, Physical Fitness on Cognitive Ability, and Education on Both

Table 4 shows the model fit statistics determining the most parsimonious versions of the models estimating the genetic and environmental influences on the cognitive composite and physical fitness variables in the presence of potential reciprocal moderating effects and moderating effects of education. We first consider the reciprocal effects of physical fitness on the cognitive composite and vice versa. These models test our first three hypotheses. Physical fitness did not moderate the genetic or environmental influences on the cognitive composite. That is, there was no evidence that the genetic or environmental influences on the cognitive composite varied across the range of physical fitness. This provided evidence in support of our second hypothesis of simultaneous deterioration in cognitive and physical function due to general aging processes but contradicted our first hypothesis that deterioration in physical function contributes directly to deterioration in cognitive function. In contrast, the cognitive composite moderated the genetic influences both common and unique to physical fitness, and both the shared and nonshared environmental influences that were unique to it. Moreover, these moderating paths were necessary and sufficient. None could be dropped without significant decrease in model fit, but adding additional moderating paths did not significantly improve model fit. This provided evidence supporting our third hypothesis that lifetime cognitive ability supports the development and maintenance of a lifestyle that helps to preserve physical health.

Table 4 also shows the model fit statistics determining the most parsimonious versions of the models estimating the extent to which education moderated the genetic and environmental influences on the cognitive composite and physical fitness variables. Education moderated the genetic and shared environmental influences unique to the cognitive composite. It also moderated the shared environmental influences common to the cognitive composite and to education. This provided evidence supporting our fifth hypothesis that education acts like cognitive ability to support the development and maintenance of a lifestyle that helps to preserve good cognitive function. For physical fitness, there were no moderating effects of education. This contradicted our fourth hypothesis that education provides resources to minimize or prevent physical illnesses. These results were determined in the same way as above.

The estimated components of variance, proportions of variance, and genetic and environmental correlations from the most parsimonious models from Table 4 are given in Tables 5 and 6, along with their 95% confidence intervals. Table 5 shows the results for reciprocal effects between cognitive composite and physical fitness (testing the first three hypotheses), and Table 6 shows the moderating effects of education on the two other variables (testing the last two hypotheses). The confidence intervals for the genetic and environmental correlations were very wide. The models have little power to specify these correlations exactly (Carey & DiLalla, 1994), so this is typical for these kinds of correlations. Some of the results shown in Tables 5 and 6 are also displayed graphically in Figure 4. We describe the tabular and graphical results and their relations to our hypotheses in turn for each pair of variables we analyzed.

Figure 4A and Table 5 show the three components of variance in the cognitive composite as functions of physical fitness. Because there was no significant moderation of the cognitive composite by physical fitness, all three of the variance components of variance remained constant across the range of physical fitness. Thus, there was no support for our first hypothesis that deterioration in physical function contributes to deterioration in cognitive function, but there was support for our second hypothesis that cognitive and physical function age simultaneously, at least from the perspective of physical function. This kind of constant variance in one trait throughout the range of another is the situation most commonly assumed in quantitative genetic models, but in this case we tested the appropriateness of this assumption. As both Figure 4A and Table 5 show, variance in the cognitive composite attributable to genetic and shared environmental influences was .23 and .24, respectively, while variance attributable to nonshared environmental influences was .57. Thus, more than half the variance in the cognitive composite was nonshared environmental, and the remainder could be attributed pretty evenly to genetic and shared environmental influences. Table 5 also shows the genetic and environmental correlations. The genetic and shared environmental correlations between the cognitive composite and physical fitness variables were both substantive at .52 and 1.00, respectively, indicating that the same sets of underlying genetic and shared environmental influences contributed substantially to both the cognitive composite and physical fitness variables but that the exact magnitudes of the overlap in these sets of influences were not measured with much precision. The nonshared environmental correlation, again indicating the extent to which the same nonshared environmental influences contributed to both traits, was small to moderate and also statistically significant.

In order to address the possibility that the association between physical fitness and the cognitive composite was primarily driven by effects of cognitive on physical function, in Figure 4B we reversed the roles of these two variables and show the three components of variance in physical fitness as functions of the cognitive composite, thus testing the third hypothesis that lifetime cognitive ability supports the development and maintenance of a

healthy lifestyle that helps to preserve physical function. There were similar moderating effects on all the components of variance, but the ones involving nonshared environmental influences were most dramatic. Each variance component of physical fitness was at its largest when the cognitive composite was low, and they became smaller at its higher levels. Thus, for example, at two standard deviations below the mean of the cognitive composite, the genetic variance in physical fitness was .37 (see Table 5 and Figure 4B), while at two standard deviations above the mean of the cognitive composite it, the genetic variance was .23, and analogous quantities are shown for variance attributable to shared and nonshared environmental influences. The difference in variance was sharpest for nonshared environmental variance in physical fitness, which decreased from 1.14 to .17 across the range of the cognitive composite. The differences in the moderating effects of the cognitive composite on the three components of variance in physical fitness had the effect of making genetic influences a relatively small proportion of total variance at two standard deviations below the mean of the cognitive composite ($.24 = .37 / [.37 + .13 + 1.04]$; see Table 5) but a much larger proportion at two standard deviations above the mean of the cognitive composite ($.23 / [.23 + .02 + .17]$; see Table 5). The proportions of variance attributable to shared and nonshared environmental influences were merely dampened relative to those for the raw variance components.

The cognitive composite's moderating effects were mostly on the variance unique to physical fitness; this meant that, as Table 5 shows, the patterns of the genetic and environmental correlations across the range of the cognitive composite were the opposite of those for the relative variance components. That is, while genetic influences on physical fitness increased in relative importance across the range of the cognitive composite, the genetic correlation dropped from very substantial at .91 when the cognitive composite was two standard deviations below the mean to nonsignificance when the cognitive composite was two standard deviations above the mean. This indicated that genetic influences contributing to both the cognitive composite and physical fitness were tightly linked when cognitive composite scores were low but not when cognitive composite scores were high. Similarly, while nonshared environmental influences on physical fitness decreased in relative and absolute importance across the range of the cognitive composite, the extent to which the same nonshared environmental influences contributed to both traits increased across the range of the cognitive composite. Again, these results supported our third hypothesis that lifetime cognitive ability supports the development and maintenance of a healthy lifestyle that acts to preserve physical function. Links in genetic influences were important in these links when the cognitive composite was low, but links in nonshared environmental influences were important when the cognitive composite was high.

Figures 4C and 4D and Table 6 show the moderating effects of education on the three components of variance in the cognitive composite and physical fitness. Education did not moderate any of the influences on physical fitness, thus providing no evidence for Hypothesis 4 that education provides resources to minimize or prevent difficulties in physical function. Genetic influences were about a quarter of the total, with the remainder being nonshared environmental. The genetic correlation was substantial at .50, but it was the only one that was significant. In contrast, the moderating effects of education on genetic influences on the cognitive composite were dramatic, providing support for Hypothesis 5 that education acts like lifetime cognitive ability to help preserve cognitive function in old age. Education acted to increase the genetic variance in the cognitive composite by a factor of more than five across a two standard deviation range (from .10 to .56; see Figure 4D and Table 6). There was a small offsetting effect on shared environmental influences. Again, because the moderating effects of education were on the genetic influences unique to the cognitive composite, the genetic correlation between the two decreased sharply across the range of education, from .97 when education was two standard deviations below the mean

to .41 when it was two standard deviations above the mean. The shared environmental correlation showed the opposite pattern, going from $-.22$ (and nonsignificant) when education was two standard deviations below the mean to $.75$ when it was two standard deviations above the mean. These patterns of genetic variance supported the version of Hypothesis 5 that lack of education would moderate variance in cognitive function by creating poor health habits. In contrast, the weaker patterns of shared environmental variance supported the version of Hypothesis 5 that greater education would moderate variance in cognitive function by inspiring better health habits.

Discussion

In this study, we used new quantitative genetic methodology to investigate five hypotheses that can help to clarify both the reasons that physical and cognitive functions are correlated in late life and the roles that education may play in those late-life functions and their correlation. To recap, we recounted three reasons for the late-life physical–cognitive correlation: the common incidence of physical illnesses that also have cognitive consequences, the power of lifetime cognitive ability to help develop and maintain good health and lifestyle habits that maintain both forms of function, and a biological process of aging that affects both. As we noted, these possible explanations are not mutually exclusive; there is no reason that all could not operate simultaneously. Consistent with this observation, our results provide support for all three explanations and do not indicate that any could be completely rejected. The ways in which they did so are informative, however, and form the basis of this discussion. In addition, our results indicate that education served primarily to modify health and lifestyle habits that had effects on physical and cognitive functions over the long run rather than to prevent disease incidence or to minimize its effects in the short run. Our methodology made it possible to illuminate the processes involved in rather minimal observed correlations: They ranged from $.08$ to $.32$. Before we discuss the ways in which our results substantiated our specific hypotheses, however, it is important to consider our study's limitations.

Study Limitations

This study was subject to some methodological limitations that should be taken into account when considering the significance and generalizability of the results. First, the sample is representative of the Danish population born between 1911 and 1930. That population is primarily Caucasian, and Denmark is a relatively wealthy nation with good national health care and standards of education. Second, genetic and environmental moderation effects of the kind we explored are often small and difficult to detect (Rowe, Vazsonyi, & Flannery, 1995). Though we clearly had power to identify several interactive effects, there may have been others present that would have required a larger sample size to detect. Third, though our data included both men and women, we did not examine the data for possible sex differences in the associations we explored due to the reduction in power involved in separating our sample by sex. In fact, we explicitly removed the effects of gender differences from the data before beginning our analyses. Future researchers should address the possibility of sex differences in this area. Fourth, our measure of physical fitness was limited to self-report of ability to perform specific activities and did not assess either actual physical aerobic capacity or self-report of frequency of engaging in aerobic physical activities. Fifth, our measure of education was gathered with respect to the breadwinner in the home rather than the participant him–herself. Thus, our education variable probably reflects household socioeconomic status to a greater degree than is usual. Finally, we used only one of many possible models of the interrelations among genetic and environmental influences, and other models may give different and also valid perspectives on the processes

we explored. Because the processes are so closely intertwined, our results must be considered suggestive and corroborative rather than conclusive tests of our hypotheses.

Integrating Our Results and Hypotheses

Our first hypothesis dealt with the possibility that chronic illnesses common in old age might first create deterioration in physical functions that then leads to deterioration in cognitive function. We hypothesized that, were this the case, lower physical function should be associated with greater variance in, or expression of influences on, cognitive function to the extent that the process involved was similar to that linking income and physical health. There should also be substantial genetic correlation between physical and cognitive functions. Though we did observe a substantial genetic correlation of .52 and a small to moderate nonshared environmental correlation of .28 between physical and cognitive function, we did not observe that physical fitness moderated the genetic or environmental influences on the cognitive composite in any way. This provides evidence that illnesses involving physical deterioration that in turn causes deterioration in cognitive function do not contribute substantially to cognitive function in old age, but it does not rule out the kinds of joint effects on physical and cognitive function, either due to chronic illness or general biological aging processes, that formed the basis of our second hypothesis. In fact, it nicely supports this second hypothesis. The nonshared environmental correlations, which showed the same pattern, also support the second hypothesis.

Our third hypothesis involved the possibility that lifetime cognitive ability supports the development and maintenance of health and lifestyle habits that preserve both physical and cognitive functions in late life. To the extent that this is the case, we expected that genetic and/or environmental influences on cognitive function would moderate genetic and/or environmental influences on physical function, with lower cognitive function associated with greater variance in physical function. This hypothesis was rather strongly supported by the observation that total variance in physical function decreased by a factor of more than three across the four standard deviation range of cognitive function we examined. Lower cognitive function was clearly associated with greater variance in physical function. This was driven primarily by greater variance attributable to nonshared environmental influences, which of course include measurement error, but both genetic and shared environmental influences also decreased. The genetic and nonshared environmental influences provided contrasting information about the relative importance of the genetic and nonshared environmental influences common to the two functions as contributing to and protective against deterioration in function. Common genetic influences appeared to contribute to deterioration in function because the correlation was high when cognitive function was low, while common nonshared environmental influences appeared to protect against deterioration in function because the correlation was high when cognitive function was high. This would be the case, for example, if lifetime cognitive ability is directly related to one's power to exert systematic control over one's lifestyle and living conditions. The largest genetic correlation was much greater than the largest nonshared environmental correlation.

Our fourth and fifth hypotheses involved the roles of education in late-life physical and cognitive functions. There was no evidence supporting the fourth hypothesis that education provides resources to prevent the incidence or minimize the severity of chronic illnesses that affect physical function, operationalized as moderating effects of genetic and/or environmental influences on physical function so that they show less variability when education was high. In contrast, there was substantial evidence for our fifth hypothesis that the effects of education are similar to those of higher cognitive ability in facilitating the development and maintenance of health and lifestyle habits that influence both physical and cognitive functions in late life as there were substantial moderating effects of education on the genetic and shared environmental influences on cognitive function. Again, the directions

of the effects indicated contrasting roles for genetic and shared environmental influences common to education and cognitive function. Lack of education seemed to create genetically influenced stress that created poor health and lifestyle habits or limitations in resources to develop good health and lifestyle habits as the genetic correlation was high when education was low. At the same time, high education seemed to facilitate shared environmental influences that made such habits possible as this is where the shared environmental correlation was high. This would be the case, for example, if poor cognitive ability limited one's ability to obtain access to information in adulthood that would be comprehensible if available, but family background helped to provide access to education that included that kind of information as part of cultural background.

Intriguingly, the role of education in late-life cognitive function in Denmark was very similar to the role of cognitive ability in educational attainment in young adults in the United States (Johnson, Deary, & Iacono, 2008). Here, we did not examine the role of cognitive function in education because educational attainment generally preceded our measure of cognitive function by many years. The reverse was true in the study of young adults. Of course, the young United States adult twins were born in a different historical period almost 60 years after the older adult Danish twins and in a different country, but it is reasonable to speculate that similar personality characteristics may be involved in using one's cognitive ability to maximize educational attainment in youth and using one's education to preserve cognitive function in late life. Candidate personality traits would be self-discipline and conscientiousness, curiosity, and openness to information.

In conclusion, we found that transactions between genetic and environmental influences on physical and cognitive functions and education in late life provide evidence supporting all of the commonly offered explanations for the correlation between physical and cognitive functions. Our results support the idea that at least some of the correlation is due to genetic influences associated with illnesses or biological aging processes that cause deterioration in both physical and cognitive functions, but these genetic influences appeared to be held in check by genetically influenced lifetime cognitive ability that could be used to create an environment that protected function. Family background leading to higher education appeared to facilitate this ability, while poor cognitive ability that may also have blocked educational attainment appeared to limit it. These processes should be explored in future research.

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Figure 1. Schematic diagram showing how the tested hypotheses relate to the possible explanations of the link between physical and cognitive functions in old age.

Figure 2.

Standard model of independent genetic and environmental influences. They are all unique to cognitive function because it is measured in isolation from other variables. They are also all constant within the population. Cognitive function is used as an example. A = genetic influences; C = environmental influences; E = environmental influences. (For A, C, and E, the respective path coefficient is designated with the analogous lowercase letter.) U = unique.

Figure 3.

Model of moderation of genetic and environmental influences on the example of cognitive function as moderated by education. Education is represented as a triangle because we are conceptualizing it as an environmental moderating variable with respect to cognitive function. Cognitive function may be influenced by all six of the paths shown, and the extent of these influences may vary linearly with education, noted by M for moderating variable in the paths above. The genetic and environmental paths influencing education are also shown in the figure but are not labeled. A = genetic influences; C = environmental influences; E = environmental influences. (For A, C, and E, the respective path coefficient is designated with the analogous lowercase letter.) b = regression coefficient.

Figure 4.

A: Variance in cognitive ability as a function of physical fitness. B: Variance in physical fitness as a function of cognitive ability. C: Variance in cognitive ability as a function of education. D: Variance in physical fitness as a function of education. In each panel, variance is partitioned by source. A = genetic influences; C = environmental influences; E = environmental influences. (For A, C, and E, the respective path coefficient is designated with the analogous lowercase letter.)

Table 1

Descriptive Statistics and Subsample Difference Tests for Study Variables

Variable	Full sample means		Paired twins' means		
	Unpaired (<i>n</i> = 2,427)	Paired (<i>n</i> = 2,304)	<i>p</i>	Monozygotic (<i>n</i> = 928)	Dizygotic (<i>n</i> = 1,238)
1. Sex (% women)	56	62	<.001	61	62
2. Age	78.8 (5.9)	76.0 (4.6)	<.001	75.9 (4.6)	76.1 (4.7)
3. Cognitive composite	-0.13 (1.00)	0.13 (0.99)	<.001	0.16 (0.96)	0.11 (1.00)
4. Physical fitness	2.9 (0.9)	3.2 (0.8)	<.001	3.2 (0.7)	3.1 (0.8)
5. Education	2.4 (1.6)	2.6 (1.8)	<.001	2.7 (1.9)	2.5 (1.7)

Note. Standard deviations are in parentheses. Cognitive composite was standardized. Physical fitness was scaled so that 4 represented ability to carry out activities without difficulty; 1 represented inability to carry out activities. Education was scaled so that 2 represented completion of Grades 7 or 8, 4 represented completion of Grade 11, 5 represented training as an apprentice beyond completion of Grades 7 or 8, 8 represented completion of 4 years of academic education beyond Grade 11.

Table 2

Zero-Order Correlations Among Study Variables

Variable	1	2	3
1. Cognitive composite	—		
2. Physical fitness	.32	—	
3. Education	.23	.08	—

Note. Education was log-transformed to reduce skewness. All variables were adjusted for age and sex effects. All correlations were significant at $p < .01$, adjusting for the presence of the twin pairs. Correlations were expectation maximized to allow consideration of missing data.

Table 3

Twin Correlations of Study Variables

Variable	Monozygotic	Dizygotic
1. Cognitive composite	.44	.35
2. Physical fitness	.25	.04
3. Education	.52	.35

Note. Correlations are expectation maximized double entered Pearson product–moment correlations.

Table 4
Fit Statistics From the Models of Variance Components Allowing for Gene–Environment Interaction and Correlation

Model	$-2 \times \log\text{-likelihood}$	df	χ^2	Δdf	p	AIC	BIC
Cognitive ability as a function of physical fitness							
All parameters free	11,758.5	6,601				11,780.5	11,828.0
Fix all moderation paths ^a	11,768.3	6,607	9.8	6	<i>ns</i>	11,778.3	11,799.9
Physical fitness as a function of cognitive ability							
All parameters free	10,257.8	5,981				10,279.8	10,327.3
Fix common C and common E moderation paths ^a	10,263.5	5,983	5.7	2	<i>ns</i>	10,281.5	10,320.4
Fix all moderation paths	10,410.2	5,987	146.7	4	<.001	10,420.2	10,441.8
Cognitive ability as a function of education							
All parameters free	10,580.4	5,872				10,602.4	10,649.9
Fix common A and common and unique E moderation paths ^a	10,580.7	5,875	.3	3	<i>ns</i>	10,596.7	10,631.3
Fix all moderation paths	10,602.3	5,878	21.6	3	<.001	10,612.3	10,633.9
Physical fitness as a function of education							
All parameters free	10,632.0	5,956				10,654.0	10,701.5
Fix all moderation paths ^a	10,635.2	5,962	3.2	6	<i>ns</i>	10,845.2	10,666.8

Note. There are possible moderation paths for each of genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E). Fixed moderation paths were constrained to zero, which means that those sources of influence were present but did not vary across the levels of the moderators. AIC = Akaike information criterion; BIC = Bayesian information criterion.

^aBest fitting model.

Table 5

Estimates of Variance Components and Proportions of Variance in Cognitive Ability and Physical Fitness and Their Genetic and Environmental Correlations When Moderating Each Other, at Three Levels of the Moderating Variables

Model result	Moderating trait	Primary trait at level of moderating trait		
		-2 SD	0 SD	2 SD
	Physical fitness	Cognitive ability as moderated by physical fitness		
Variance component				
Genetic	.20 (.11, .27)	.23 (.00, .62)	.23 (.00, .62)	.23 (.00, .62)
Shared environmental	.00 (.00, .06)	.24 (.00, .45)	.24 (.00, .45)	.24 (.00, .45)
Nonshared environmental	.68 (.62, .77)	.57 (.50, .68)	.57 (.50, .68)	.57 (.50, .68)
Proportions of variance				
Genetic	.23 (.15, .29)	.22 (.00, .60)	.22 (.00, .60)	.22 (.00, .60)
Shared environmental	.00 (.00, .05)	.23 (.00, .43)	.23 (.00, .43)	.23 (.00, .43)
Nonshared environmental	.77 (.72, .85)	.55 (.49, .64)	.55 (.49, .64)	.55 (.49, .64)
Correlations with moderator				
Genetic		.52 (-.08, .66)	.52 (-.08, .66)	(-.08, .66).52
Shared environmental		1.00 (-.72, 1.00)	1.00 (-.72, 1.00)	1.00 (-.72, 1.00)
Nonshared environmental		.28 (.15, .34)	.28 (.15, .34)	.28 (.15, .34)
	Cognitive ability	Physical fitness as moderated by cognitive ability		
Variance components				
Genetic	.21 (.06, .41)	.37 (.12, .76)	.19 (.00, .43)	.23 (.00, .62)
Shared environmental	.24 (.08, .37)	.13 (.00, .52)	.02 (.00, .11)	.02 (.00, .27)
Nonshared environmental	.56 (.49, .64)	1.04 (.89, -1.23)	.50 (.43, -.59)	.17 (.09, .26)
Proportions of variance				
Genetic	.21 (.06, .40)	.24 (.08, .49)	.27 (.00, .49)	.55 (.00, .77)
Shared environmental	.24 (.08, .36)	.08 (.00, .32)	.02 (.00, .10)	.04 (.00, .28)
Nonshared environmental	.55 (.50, .63)	.68 (.58, -.78)	.71 (.65, -.79)	.41 (.33, -.49)
Correlations with moderator				
Genetic		.91 (.46, -1.00)	.56 (.11, 1.00)	-.13 (-.59, -.33)
Shared environmental		.13 (-1.00, 1.00)	.37 (-1.00, 1.00)	.37 (-1.00, 1.00)
Nonshared environmental		.16 (.09, .25)	.23 (.17, .52)	.40 (.33, -.47)

Note. The variance components are raw; they do not sum to 1.00. The proportions of variance sum to 1.00. The 95% confidence intervals are given in parentheses.

Table 6

Estimates of Variance Components and Proportions of Variance in Cognitive Ability and Physical Fitness and Their Genetic and Environmental Correlations When Moderated by Education, at Three Levels of Education

		Primary trait at level of education		
Model result	Education as moderator	−2 SD	0 SD	2 SD
Cognitive ability as moderated by education				
Variance components				
Genetic	.35 (.17, .53)	.10 (.00, .47)	.19 (.00, .52)	.56 (.00, .86)
Shared environmental	.16 (.02, .31)	.33 (.00, .67)	.20 (.00, .41)	.21 (.00, .57)
Nonshared environmental	.47 (.42, .54)	.54 (.47, .65)	.54 (.47, .65)	.54 (.47, .65)
Proportions of variance				
Genetic	.36 (.17, .52)	.10 (.00, .42)	.20 (.00, .52)	.43 (.00, .65)
Shared environmental	.16 (.02, .30)	.34 (.00, .61)	.21 (.00, .39)	.16 (.00, .47)
Nonshared environmental	.48 (.42, .54)	.56 (.50, .61)	.59 (.53, .64)	.41 (.36, .46)
Correlations with moderator				
Genetic		.97 (.53, 1.00)	.72 (.27, 1.00)	.41 (−.04, 1.00)
Shared environmental		−.22 (−.73, .33)	.25 (−.27, 1.00)	.75 (−.04, 1.00)
Nonshared environmental		−.11 (−.28, .03)	−.11 (−.28, .03)	−.11 (−.28, .03)
Physical fitness as moderated by education				
Variance components				
Genetic	.34 (.16, .52)	.21 (.00, .58)	.21 (.00, .58)	.21 (.00, .58)
Shared environmental	.17 (.03, .32)	.01 (.00, .07)	.01 (.00, .07)	.01 (.00, .07)
Nonshared environmental	.47 (.42., 54)	.61 (.54, .69)	.61 (.54, .69)	.61 (.54, .69)
Proportions of variance				
Genetic	.35 (.17, .52)	.26 (.00, .62)	.26 (.00, .62)	.26 (.00, .62)
Shared environmental	.17 (.03, .31)	.01 (.00, .06)	.01 (.00, .06)	.01 (.00, .06)
Nonshared environmental	.48 (.42, .54)	.74 (.68, .81)	.74 (.68, .81)	.74 (.68, .81)
Correlations with moderator				
Genetic		.50 (.09, −.74)	.50 (.09, −.74)	.50 (.09, −.74)
Shared environmental		−1.00 (−1.00, .41)	−1.00 (−1.00, .41)	−1.00 (−1.00, .41)
Nonshared environmental		−.06 (−.16, .02)	−.06 (−.16, .02)	−.06 (−.16, .02)

Note. The variance components are raw; they do not sum to 1.00. The proportions of variance sum to 1.00. The 95% confidence intervals are given in parentheses.