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# Shorter Adult Stature Increases the Impact of Risk Factors for Cognitive Impairment: A Comparison of Two Nordic Twin Cohorts

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We analyzed the association between mean height and old age cognition in two Nordic twin cohorts with different childhood living conditions. The cognitive performance of 4720 twin individuals from Denmark (mean age 81.6 years, *SD* = 4.59) and Finland (mean age 74.4 years, *SD* = 5.26) was measured using validated cognitive screens. Taller height was associated with better cognitive performance in Finland (β-estimates 0.18 *SD*/10cm, *p* value < .001, for men and 0.13 SD, *p* = .008, for women), but this association was not significant in Denmark (β-estimates 0.0093 *SD*, *p* value = .16, for men and 0.0075 *SD*, *p* value = .016, for women) when adjusted for age and education/social class. Among Finnish participants higher variability of cognitive performance within shorter height quintiles was observed. Analysis using gene-environment interaction models showed that environmental factors exerted a greater impact on cognitive performance in shorter participants, whereas in taller participants' it was explained mainly by genetic factors. Our results suggest that shorter participants with childhood adversity are more vulnerable to environmental risk factors for cognitive impairment.

**Keywords:** twins, genetics, height, cognition, dementia, risk factor

Early life nutritional deficits increase the risk of common diseases, such as cardiovascular disease (Wu & Chen, 2009), hypertension (Sawaya et al., 2005), diabetes (Barker, 2005) and osteoporosis (Nicklas, 2003), in later life. Correlation between childhood living environment and Alzheimer's disease (AD) (Borenstein et al., 2006) suggests that poor childhood living environment may increase the risk of neurodegenerative diseases, too. Indeed, twin study findings support a significant set of environmental factors affecting old age cognitive functioning (Lee et al., 2010).

If early life exposures are associated with old age cognition, different patterns of cognitive performance should be demonstrable between cohorts with different childhood living environments. Notwithstanding the comparable living environments across the Nordic countries these days, there were substantial differences between Denmark and Finland when the cohort participants in this study were growing up. Comparisons of Gross Domestic Product (Maddison, 2003), as well as infant (Amiri et al., 2006; Mitchell, 1978.; Turpeinen, 1979) and child mortality (Gapminder Foundation, 2010) between Finland and Denmark indicate poorer living conditions in Finland during the first half of the 20th century.

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Variation in adult height is mainly due to genetic factors, but it also reflects the impact of numerous environmental exposures in utero, childhood and adolescence (Silventoinen et al., 2003). Indeed, an association between shorter adult height and poor cognitive performance in old age (Abbott et al., 1998; Jeong et al., 2005) or in dementia diseases, mainly AD (Abbott et al., 1998; Beeri et al., 2005; Petot et al., 2007), vascular dementia (VaD) (Beeri et al., 2005), the combination of VaD and other non-AD based pathologies (Gatz et al., 2006) or dementia as a whole (Beeri et al., 2005; Gatz et al., 2006), has been demonstrated. If dementia is associated with shorter height, it may be explained either by a shared genetic or environmental predisposition among short people. Such a predisposition may also increase vulnerability to genetic or later life environmental factors leading to cognitive impairment.

The presence of several risk factors already in early life, especially in developing countries, as well as the increasing proportion of elderly people with varying childhood environmental restrictions and longer life expectancy, all emphasize the importance of studies on environmental factors affecting cognitive performance in old age. The aim of our study was to determine whether an association exists between height and cognitive performance in old age in two large Nordic population-based samples having substantially different childhood living environments. We also investigated whether there was interaction between height and genetic versus environmental components behind this association using quantitative genetic modeling for twin data.

## Participants and Methods

### The Older Finnish Twin Cohort

The participants belonged to the older cohort of the Finnish Twin Cohort Study, which contains all Finnish same-sex twin pairs born before 1958 (Kaprio & Koskenvuo, 2002). Zygosity, height, education and other baseline characteristics were self-reported by postal questionnaires in 1975 and 1981 (response rates 89% and 84%, respectively). Mean age at the time of height reporting was 45.9 years ($SD$ = 5.7) in men and 48.1 years ($SD$ = 6.7) in women, and the correlation between self-reported and measured height was over 0.95 in a subsample (Silventoinen et al., 2000). Moreover, there was full agreement in this subsample between the zygosity determination based on questionnaire items and genetic markers (Sarna et al., 1978).

Respondents were asked to classify their education in one of eight categories, and this was converted to educational years and analyzed as a continuous variable (Silventoinen et al., 2000). In the analyses, education as reported in 1981 was chosen to represent highest educational level. As age-associated shortening was likely to be least when participants were younger, height (cm) was based on the 1975 questionnaire data. If not reported in the preferred year, data was taken from the other survey.

Between 1999 and 2007, all respondents aged 65 or older were invited to participate in a telephone interview to define their cognitive status using a combination of two sensitive and specific telephone screens: a self-report interview referred to as TELE (Gatz et al., 1995) and the Telephone Interview for Cognitive Status (TICS) (J. Brandt et al., 1988), both of which correlate strongly with clinical measurements, such as the Mini Mental State Examination (MMSE; Järvenpaa et al., 2002). The questions included in both screens were asked once and the total score from this 29-item interview formed a linear variable called cognitive score (Laitala et al., 2009).

The telephone interview was completed for 2483 twins of known zygosity (703 monozygotic (MZ) and 1780 dizygotic (DZ) twins) and 123 twins of uncertain zygosity, with an overall response rate of 79 %. Among those who were not interviewed, 127 were not reached by telephone, 412 declined to participate in the interview, 32 died before being contacted, and 133 were not contacted or their interview was not completed. For the MZ twins, the participants and non-participants were found to be comparable with respect to sex, schooling and alcohol use (Järvenpää et al., 2005). Mean age at the time of the telephone interview was 73.6 years ($SD$ = 4.65) in men and 75.4 years ($SD$ = 5.72) in women; 48.0% of the respondents were women.

### The Longitudinal Study of Aging Danish Twins

Danish participants belonged to the Longitudinal Study of Aging Danish Twins (LSADT), which is a sample drawn from older cohorts of the Danish Twin Registry including all Danish twin pairs born between 1870 and 1910 and all same-sex twin pairs born between 1911 and 1930 (Hauge, 1981; Holm, 1983). Baseline characteristics including zygosity were assessed by a postal questionnaire as soon as a twin was traced after the registry was established in 1954 (Skytthe et al., 2006). The comparison of zygosity determination based on questionnaire items and blood group determinants found a misclassification rate of less than 5% (Christiansen et al., 2003).

In 1995, all respondents aged at least 75 years were asked to participate in an interview including MMSE, social classification and height measurement (Christensen et al., 1999). The majority of the assessments were performed in the twins' primary residence by trained interviewers from the Danish National Institute of Social Research. Social classification was based on one's position in the production irrespective of whether this was gained through ownership or attained through education.

A total of 2401 interviews were conducted (77% of the potential study population). Of the remainder, a partial interview due to inability to complete was obtained from 1%, 9% did not wish to participate in the full interview, and no interview information was obtained from 13% of the potential study population. The participants and non-participants were similar in terms of age and earlier



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morbidity based on hospitalization data (Christensen et al., 1999). However, since participants with severe physical or cognitive impairment were interviewed by proxy, completed MMSE was available only from 2161 respondents (63% women), whose mean age at the time of both height and cognitive assessment was 81.6 years (*SD* = 4.6) in men and 82.1 years (*SD* = 4.8) in women.

### Statistics

Participants were divided into sex and country specific height quintiles, and means and variances of cognitive test scores were calculated separately for each quintile. Linear regression analyses, initially adjusted only for age at interview and then additionally for education/social class, were used to determine the association between height and cognition. Regression analyses were also performed for standardized cognitive measurements; that is, per one *SD* change, in order to improve comparability between the study samples. In these analyses, twins were treated as individuals and the dependence of individual observations; that is, clustering within pairs was taken into account to obtain correct confidence intervals using the cluster option in the Stata statistical package 11 for Windows (Williams, 2000).

Since we found no consistent differences in the variances of cognitive score between height quintiles in Danish twins, we continued the genetic analyses only in Finnish twins with the Mx statistical software using the raw data option, which also allows twins to be included without their co-twins (Neale, 2003). The twin design assumes that DZ twins share, on average, 50% of their segregating genes, whereas MZ twins are genetically identical at the sequence level. Genetic resemblance between twins results from genes having an additive effect of their alleles in different loci (A) and an interaction effect between alleles at the same locus, summed over all relevant loci (D). The correlation of genetic effects is 1 within MZ twin pairs, whereas the correlations of A and D effects are 0.5 and 0.25 within DZ twin pairs. Environmental effects are defined as common (C) and unique (E), with respective correlations 1 and 0 within both MZ and DZ twin pairs. C is the sum of environmental factors similarly affecting both co-twins, such as childhood family environment, whereas E represents non-shared environmental factors and includes any measurement error.

To find the initial model, the intraclass correlations of cognitive score within MZ twins were compared to those within DZ twins. Model evaluation is based on comparing a model containing fewer parameters to the model from which it is nested by performing the likelihood ratio $\chi^2$ test and determining *p* values. In addition, moderator variables, such as height in this study, which can affect both means (shorter people may have lower cognitive test score) and variances of trait (taller people may have less genetic and/or environmental variance) can be tested by constructing a gene–environment interaction (G × E) model (Purcell, 2002). Only linear modification effects were fitted in the models, because our data sets are not large enough to test nonlinear effects. Cognitive tests showed some violation from normality, which was more a problem in the Danish (skewness -1.66 and kurtosis 6.63) than in the Finnish sample (-1.38 and 6.15, respectively). However, because the G × E models we used analyse particularly how variance changes as a function of environmental moderator, we decided not to make any transformation to normalize the distribution artificially, because it may have biased the results.

## Results

Height was available from 2566 Finnish and 2154 Danish participants with a completed cognitive interview. In Finland mean height was 173.3 cm (*SD* = 6.0) for men and 160.7 cm (*SD* = 5.6) for women, and in Denmark 170.6 cm (*SD* = 6.7) for men and 160.5 (*SD* = 6.2) for women.

According to linear regression analysis adjusted for age, each 1 cm increase in height improved the cognitive score by an average of 0.17 units (95%CI = 0.12; 0.22, *p* value < .001) in Finnish men and 0.15 units (95%CI = 0.084; 0.21, *p* value < .001) in Finnish women. Among Danish participants, the respective MMSE values were 0.07 units (95%CI = 0.011; 0.13, *p* value = .021) in men and 0.051 units (95%CI = 0.012; 0.090, *p* value = .011) in women. When further adjusted for education/social class, the improved score per 1 cm height increase was 0.11 units (95%CI = 0.065; 0.16, *p* value < .001) in Finnish men and 0.083 units (95%CI = 0.021; 0.14, *p* value = .008) in Finnish women, while among Danish participants the corresponding results were 0.044 units (95%CI = -0.018; 0.11, *p* value = .16) and 0.037 units (95%CI = -0.0018; 0.076, *p* value = .061) in men and women.

When using standardized cognitive measurements, each 10 cm increase in height elevated the cognitive score by an average of 0.28 *SD* (95%CI = 0.20;0.36, *p* value < .001) in Finnish men and 0.24 *SD* (95%CI = 0.13; 0.34, *p* value < .001) in Finnish women. Among Danish participants, the respective values for MMSE were 0.015 *SD* (95%CI = 0.0023; 0.028, *p* value = .021) in men and 0.010 *SD* (95%CI = 0.0024; 0.018, *p* value = .011) in women. When further adjusted for education/social class, the results were 0.18 *SD* (95%CI = 0.10;0.26, *p* value < .001) in Finnish men and 0.13 *SD* (95%CI = 0.034; 0.23, *p* value = .008) in Finnish women, compared to 0.0093 *SD* (95%CI=-0.0037; 0.022, *p* value = .16) and 0.0075 SD (95%CI = -0.00036; 0.015, *p* value = .016) in Danish men and women.

Mean cognitive score increased more among Finnish than Danish participants within quintiles of taller height and there was a consistent decline in variances (standard deviations) with increasing height in Finnish participants. To analyze the causes of consistently decreasing variance among Finnish participants, we fitted genetic models, including G × E models (model fit details in



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**TABLE 1**

Age-Standardized Means, Standard Deviations (SD) and 95% Confidence Intervals (95% CI) of Cognitive Tests in Finnish and Danish Participants According to Height Quintiles[1]

| | Finnish | | | | Danish | | |
|---|---|---|---|---|---|---|---|
| height (*n*) | mean | *SD* | 95% CI | height (*n*) | mean | *SD* | 95% CI |
| Men | | | | | | | |
| 150–167 (210) | 36.28 | 6.10 | 35.45;37.11 | 140–167 (219) | 23.75 | 5.18 | 23.06;24.44 |
| 168–171 (305) | 37.49 | 5.36 | 36.88;38.09 | 168–170 (192) | 24.82 | 4.51 | 24.18;25.46 |
| 172–174 (276) | 38.71 | 5.07 | 38.10;39.31 | 171–173 (118) | 24.99 | 3.31 | 24.39;25.58 |
| 175–177 (203) | 38.55 | 5.81 | 37.75;39.35 | 174–178 (159) | 25.07 | 4.63 | 24.35;25.79 |
| 178–192 (339) | 39.61 | 4.45 | 39.13;40.08 | 179–200 (92) | 25.37 | 5.07 | 24.33;26.41 |
| Women | | | | | | | |
| 145–154 (183) | 36.35 | 6.46 | 35.41;37.30 | 120–156 (371) | 23.90 | 4.41 | 23.45;24.35 |
| 155–159 (299) | 37.18 | 6.64 | 36.43;37.94 | 157–160 (368) | 23.66 | 5.32 | 23.11;24.20 |
| 160–161 (153) | 37.43 | 6.42 | 36.40;38.45 | 161–163 (183) | 24.80 | 4.74 | 24.11;25.49 |
| 162–164 (265) | 37.98 | 5.70 | 36.96;38.34 | 164–166 (227) | 24.43 | 4.39 | 23.86;25.00 |
| 165–180 (333) | 38.43 | 5.23 | 37.87;38.99 | 167–184 (225) | 24.84 | 4.65 | 24.23;25.44 |

Note: [1]Quantiles are not exact fifths, since respondents having the same height could not be assigned to two different quintiles; *n* = number of respondents.

Appendix A Table A1). In both sexes, the overall intraclass correlations of cognitive score within MZ twin pairs ($r = .62$, $n = 159$ twin pairs in men; $r = .49$, $n = 174$ in women) were less than two times higher than in DZ twins ($r = .33$, $n = 256$ in men; $r = .28$, $n = 249$ in women) suggesting a C effect in addition to A and E effects (ACE model) for cognitive score. However, comparison of a model containing A and E effects and their moderators to a model also including a C effect and its moderator (i.e. comparing AE model to ACE model) showed that C and its moderator effects were not significant either in men ($\Delta\chi^2_2 = 1.84$, $p = .40$) or in women ($\Delta\chi^2_2 = .42$, $p = .81$).

Thus, further analyses were carried out using an AE model including height as a moderator of both A and E (i.e. how height affected the magnitude of these components) both in men and women and comparing model fits to ones without the moderator effects. Not including the moderator of E component had a statistically significant effect, meaning that height moderated the magnitude of E component both in men ($\Delta\chi^2_1 = 14.74$, $p = .0001$) and women ($\Delta\chi^2_1 = 20.59$, $p < .0001$). Instead, omitting moderation of A component was not significant either in men ($\Delta\chi^2_1 = 1.73$, $p = .19$) or women ($\Delta\chi^2_1 = .062$, $p = .80$), and thus this simpler model could be accepted as adequately describing the data.

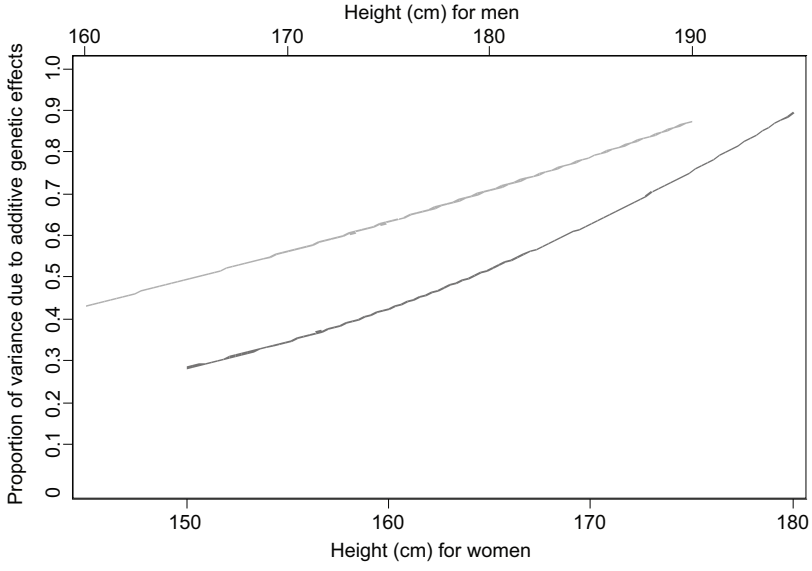


**FIGURE 1**

Proportion of variance attributed to additive genetic effects by height of subjects (upper line = men, lower line = women) based on a moderator variable model fitting in Finnish men and women. The association implies that shorter subjects have more environmental variance whereas genetic variance plays a greater role among taller participants.




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The gene–height interaction model implied that shorter subjects had more environmental variance, which also had a greater impact on cognitive performance among shorter Finnish men and women. Instead, genetic variance played a greater role among taller Finnish participants.

## Discussion

An association between childhood living environment and AD was first observed in studies concerning the clinical picture of AD (Weiner et al., 1996) or combined potential risk factors (Kondo et al., 1994). More recent case-control studies have found that lower mental and linguistic abilities at school age (Whalley et al., 2000) or in early adulthood (Riley et al. 2005), poorer area of childhood residence and higher number of siblings (Moceri et al., 2000), early tooth loss in adulthood (Gatz et al., 2006), as well as shorter adult stature (Gatz et al., 2006; Jeong et al., 2005; Petot et al., 2007), arm length (Huang et al., 2008; Jeong et al., 2005; Kim et al., 2003) and leg length (Huang et al., 2008; Kim et al., 2003; Mak et al., 2006) are each associated with poorer cognitive performance and memory disorders, mainly AD. Most of these case-control studies were conducted in Asian populations, and only three in European (Gatz et al., 2006) or US populations (Huang et al., 2008; Petot et al., 2007).

A few existing follow-up studies have shown that taller height at mean age 53 years diminished the prevalence of poor cognitive performance (Abbott et al., 1998), and that midlife height was inversely associated with AD, vascular dementia and dementia as a whole (Beeri et al., 2005). The participants in previous follow-up studies represent US (Abbott et al., 1998) and Israeli (Beeri et al., 2005) populations, and to our knowledge there have been no follow-up studies of this association in Europeans. A greater genetic variance in adolescent height in Caucasian compared to East Asian populations found in a large international study (Hur et al., 2008) highlights the importance of studies within different populations.

In our study, shorter adult height was associated with lower cognitive performance in both study samples, although the association was more obvious among Finnish than Danish participants. This was supported by both lower test scores among those in shorter height quintiles and regression coefficients, which were significant in Finnish participants when adjusted for both age and education but in Danish participants only when adjusted for age. Moreover, there was a clear decrease in cognitive score variance with increasing height in Finnish participants, but not in Danish participants. This means that among shorter Finnish participants there were more subjects with decreased cognitive performance in addition to those with normal cognition, whereas the prevalence of poorer test scores was lower among taller Finnish participants. Instead, among Danish participants, the prevalence of decreased test scores was comparable in all height quintiles (represented by comparable SD's in all height quintiles in Table 1).

Only a little is known about the genetics underlying the association between height and cognition. In one study, presence of the ApoE epsilon 4 allele obscured the association between height and AD in women (Petot et al., 2007), but the results of other studies are controversial (Huang et al., 2008; Kim et al., 2003; Moceri et al., 2000). GxE interaction in quantitative genetic studies, such as twin analyses, can both elucidate environmental pathways and direct gene-mapping efforts (Purcell, 2002).

According to twin studies, genetic factors have been found to account for both less and more than half of the total variance of cognitive performance in old age (Brandt et al., 1993; Lee et al., 2010; McGue & Christensen, 2001; McGue & Christensen, 2002). However, they also support a significant effect of environmental factors, some of which may be present already in childhood. In general, genetic influence on cognitive functioning has been found to decrease with advancing age (Lee et al., 2010), but there are no previous studies on differences in height. In this study, a greater environmental variance among shorter participants was found, which suggests that restrictive childhood environment (resulting in shorter height) increases vulnerability to environmental factors for poorer cognitive performance. It seems reasonable that restrictions during childhood would decrease the effect of the genetic component similarly to increasing age. Since those factors were found to be unique for each co-twin, and twin siblings usually share the exposures present in childhood, we assume that most of them are encountered later in life. In people who had not experienced restrictions in their childhood and thus had reached an adult height in accordance with their genetic potential, old age cognitive performance was found to be determined to a greater extent by their genotype. However, genetic variance itself did not change with height.

Childhood environmental limitations such as malnutrition (Yehuda et al., 2006), chronic diseases (Malleson, 1991), behavioral disturbances and psychosocial stress (Skuse et al., 1996) contribute to slower growth velocity and may restrict the attainment of height according to genetic potential. Indeed, the steady increase in living standards during the first half of the last century in Finland has been demonstrated to occur in parallel with the increase in heritability of height (Silventoinen et al., 2000) indicating that genetic factors play a greater role in growth in the presence of a favourable childhood environment. Also, the greater height variation in Caucasians compared to East-Asian populations may indicate either stronger influence of childhood factors on height or greater differences in childhood living environments among Caucasians (Hur et al., 2008). Either way, this highlights the usability of height difference as a tool for estimating childhood environment in Caucasian populations.



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The strength of this study is that it is based on a comparison of two Nordic populations with highly different childhood environments. Before the Second World War, there were large differences in living standards between the Nordic countries, among which Finland was the poorest. However, although the gap in average height between Finland and Sweden narrowed after the Second World War, Finnish cohorts born earlier were shorter (Silventoinen et al., 2001), indicating greater environmental stress affecting height in the Finnish population. In the 1920s and 1930s, Gross Domestic Product in Finland was around half of that in Denmark (Maddison, 2003). The difference in childhood conditions is supported by the higher infant (Amiri et al., 2006; Mitchell, 1978.; Turpeinen, 1979) and child mortality (Gapminder Foundation, 2010) rates seen in Finland in the early 20th century. For instance, the probability of death before the age of five was 40% higher in Finland than in Denmark in 1920, and varied from 8% to 95% till 1950, when child mortality was still 46% higher in Finland (Gapminder Foundation, 2010). Thus, we assume that the greater variance in cognitive score among shorter Finnish respondents results from participants experiencing childhood environmental restrictions and thus failing to reach their genetically determined height, which also increases their risk for poor cognitive performance. The lack of this phenomenon in Danish twins is reasonable, since most Danish participants had probably experienced a more favorable childhood environment than the Finns.

Comparing two samples can have several strengths but also some limitations. For instance, measures of education and social class are not fully comparable. Although high SES can be achieved otherwise than via long education, higher social class is most often associated with higher education (Magnus & Mick, 2000; McGarvey et al., 1981; Staff & Mortimer, 2008). Moreover, we believe that SES and length of education both reflect intellectual capacity throughout working life, which makes them more comparable in terms of their influence on old age cognitive performance and emphasizes the importance of taking the effects of both into account in this comparable study.

Both of the telephone interviews, which combination was used in the Finnish sample, have been found to correlate strongly with MMSE, which was used for the Danish participants: Pearson's correlations between MMSE and TELE and TICS (Pearson's $r = .87$ and 0.86, $p < .0001$) were high in AD patients of this same Finnish study population (Järvenpää et al., 2002), and even higher correlations between MMSE and TELE (Gatz et al., 1995) and TICS (Brandt et al., 1988) have been found in other study populations. Telephone screens have some limitations, such as problems with impaired hearing and distractions, and difficulty accessing visuospatial skills and controlling use of information sources and instruments including pens and paper. However, the ease of use and widespread applicability of telephone screening may overcome limitations caused by physical impairment, long travel distances and low motivation of some older people to participate, and this doubtless contributed to the high response in our study. Indeed, the successful use of telephone screening has been documented in several reports apart from our study sample (Laitala et al., 2009). It should be noted that high scores in the cognitive tests used in this study indicate normal cognitive performance, and thus the increasing variance in cognitive test score results from a higher prevalence of poor or decreased cognitive performance.

Although diagnostic classification of old age cognition is reasonable for classic risk factor studies including risk quantification, the use of cognitive performance as a continuous variable in our study allowed us to perform genetic models (because of better statistical power) and to examine the effect of childhood living conditions on the whole range of poor or decreased cognitive ability. Indeed, our findings suggest that shorter participants with restrictions in childhood environment are at higher risk of poorer cognitive performance less severe than dementia, which represents the extreme of cognitive decline. However, since the shorter subjects in our study cohorts had a substantially heterogeneous background, and because poor cognitive performance and dementia are generally manifestations of a multifactorial etiology, clinical conclusions based on the findings of this study should be drawn with special caution.

The height of the Finnish subjects was measured in midlife, on average 27.5 years before the assessment of cognitive performance, when height decreasing conditions such as severe osteoarthritis or osteoporosis are still rare. On the other hand, the height of the Danish subjects was measured at the time of cognitive assessment in old age, so we cannot exclude the effect of age-associated shortening among them. However, the likelihood that more diseased subjects with poorer cognitive performance would have shortened more should strengthen the association between shorter height and cognitive performance and increase the variance in cognitive test performance among shorter Danish subjects, thus making them more similar to the Finnish subjects. Although twins are shorter than singletons at birth, several studies have established that differences in means or variances of height between MZ or DZ twins or twins and singletons in adulthood are only minor (Andrew et al., 2001; Silventoinen et al., 2008).

To conclude, we found a clear and higher decline in cognitive score with decreasing height in a population with a restrictive childhood environment compared to a population without. Further investigation revealed that childhood restrictions leading to shorter height were associated with greater vulnerability to environmental factors on old age cognitive performance. These findings suggest that poor childhood living environments cause wide socioeconomic and public health problems decades later, and that the burden of past environmental restrictions may have an influence far into the future. For instance, older birth




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cohorts in rapidly developing countries are likely to need more health care in their old age. The greater vulnerability of the shortest subjects to environmental risk factors should be considered within classic risk factor studies, and whether they should be treated differently needs to be determined on the grounds of the population in question. Similarly, in genetic research, the larger environmental variation in old age cognitive performance among shorter subjects should be taken into consideration.

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# Appendix A

**TABLE A1**

Model Fit Statistics, Standardized Variance Components and Moderator Effects of Height Using Different Models for Cognitive Scores in the Finnish Men and Women

| Model | Model fit statistics | | | Variance component estimates[1] | | | Moderator effect estimates | | |
|---|---|---|---|---|---|---|---|---|---|
| | Degrees of freedom | $\chi^2$-values | AIC-index | Additive genetics | Common environment | Unique environment | Additive genetics | Common environment | Unique environment |
| Men | | | | | | | | | |
| Full model | 4807.67 | 802 | 3203.67 | 0.48<br>0.21, 0.58 | 0.01<br>0.00, 0.23 | 0.51<br>0.42, 0.63 | -0.02<br>-0.14, 0.09 | -0.19<br>-0.32, 0.32 | -0.12<br>-0.16, -0.06 |
| No common environment | 4809.51 | 804 | 3201.51 | 0.82<br>0.78, 0.85 | — | 0.18<br>0.15, 0.22 | -0.05<br>-0.12, 0.02 | — | -0.12<br>0.17, -0.06 |
| No unique environmental moderator | 4824.24 | 805 | 3214.24 | 0.58<br>0.48, 0.66 | — | 0.42<br>0.34, 0.53 | 0.05<br>-0.01, 0.11 | — | — |
| No additive genetic moderator | 4811.24 | 805 | 3201.24 | 0.6<br>0.53, 0.66 | — | 0.4<br>0.34, 0.47 | — | — | -0.1<br>-0.14, 0.05 |
| Women | | | | | | | | | |
| Full model | 5264.54 | 827 | 3610.54 | 0.41<br>0.32, 0.48 | 0<br>0.00, 0.06 | 0.59<br>0.52, 0.67 | -0.02<br>-0.17, 0.14 | -0.09<br>-0.26, 0.26 | -0.15<br>-0.22, -0.09 |
| No common environment | 5264.96 | 829 | 3606.96 | 0.73<br>0.67, 0.77 | — | 0.27<br>0.23, 0.33 | 0.01<br>-0.08, 0.10 | — | -0.15<br>-0.22, -0.09 |
| No unique environmental moderator | 5285.55 | 830 | 3625.55 | 0.41<br>0.30, 0.50 | — | 0.59<br>0.50, 0.70 | -0.14<br>-0.24, -0.06 | — | — |
| No additive genetic moderator | 5265.03 | 830 | 3605.03 | 0.44<br>0.34, 0.53 | — | 0.56<br>0.47, 0.66 | — | — | -0.16<br>-0.21, -0.10 |

Note: [1]Variance component estimates are standardized and presented for mean height in men (173 cm) and women (161 cm).




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