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The Apolipoprotein E e4 Polymorphism Is Strongly Associated With Poor Mobility Performance Test Results But Not Self-Reported Limitation in Older People

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Abstract

Background—The apolipoprotein E (ApoE) e4 polymorphism is linked to increased mortality rates, Alzheimer's disease, and cardiovascular disease in older people, but previous studies have largely failed to detect an effect on self-reported mobility disability. We hypothesized that poor performance on mobility-related tests may provide a better measure of effects, and we aimed to estimate the extent to which the ApoE e4 allele increases risks of poor performance on measured mobility and self-reported mobility disability compared to e3/3, in a medium-sized population cohort.

Methods—Data were from 1262 people at baseline older than 65 years from the Longitudinal Aging Study Amsterdam (LASA), followed up for 6 years. Age- and sex-adjusted logistic regression models were used to explore associations.

Results—At baseline, those individuals with an e4 allele had an odds ratio of 2.26 (95% confidence interval, 1.31–3.90) for poor performance on gait speed testing (<0.4 m/s) and 1.94 (95% confidence interval, 1.19–3.16) for five chair stands (≥20 s), compared to those with e3/3 status. At follow-up, associations between e4 status and incident poor performance on the chair stand test was significant. Associations with self-reported inability or need for help walking for 5 minutes or for climbing 15 steps were nonsignificant throughout.

Conclusions—The ApoE e4 polymorphism is associated with a substantial excess of mobility limitation. The impact is detectable by performance testing, but not by self-reports. Poor results on mobility performance tests may provide a phenotype of ageing.

THE apolipoprotein E (ApoE) e4 polymorphism has been associated with increased risk of cardiovascular disease, stroke, Alzheimer's disease, and impaired cognitive function (1). ApoE e4-associated increases in mortality rates have also been reported, especially at younger ages. Multiple mechanisms may contribute to the effects of ApoE, including its roles in total cholesterol and low density lipoprotein metabolism (2), which are mediated through allele-specific differences in the clearance of different lipoproteins.

Given the major disease and mortality rate effects of ApoE e4, significant impacts on functional ability in old age could be expected. Indeed, Gerdes and colleagues (3) have advocated ApoE as a "frailty" gene, yet thus far little evidence of major effects on disability has appeared. Albert and colleagues (4) studied 218 nondemented elderly persons aged 58–93 years, and reported an association between the e4 allele carriers and poorer scores on activities of daily living.

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However, Bader and colleagues (5) found no association of ApoE genotypes with activities of daily living in 162 Italian octogenarians and nonagenarians. Carmelli and colleagues (6) studied 390 males with a mean age of 72 years and observed a nonsignificant effect of ApoE genotypes on tested lower extremity function. Blazer and colleagues (7) similarly reported that e4 was not associated with incident self-reported functional declines as a main effect, in 1529 participants of mean age 77.8 years, although interactions were found in women. In further work in the Duke Established Populations for Epidemiologic Studies of the Elderly (EPESE) site, Blazer and colleagues (8) also showed that there was no association between e4 status and any of five dimensions of quality of life, and argued that their findings "challenge the uncritical assumption that the presence of this susceptibility gene in the population implies an excess burden of poor quality of life."

Restricted (walking) mobility is an early and relatively culture-free marker of the development of disabilities. In the Women's Health and Aging Study (9) and the 1984 Supplement on Aging (10), well over 90% of respondents with any disability reported problems with mobility. In addition, there is good evidence that poor performance on gait speed and chair stand tests in older respondents is associated with increased risk of subsequent disability and mortality (11,12). By contrast, questionnaires eliciting self-reported mobility disability inevitably incorporate the effects of environment and attitude (13,14), and are subject to "category response shifts" (15,16).

In this study we examined the impact of the ApoE e4 polymorphism on mobility in old age in a large population-based study, using self-report and performance tests, to establish both subjectively and objectively whether e4 is associated with excess mobility impairment in older people.

Methods

Sample

The Longitudinal Aging Study Amsterdam (LASA) is a population-based study of persons aged 55–85 years. Sampling and data collection procedures have been described elsewhere (17,18). In summary, a random age- and sex-stratified sample was drawn from the population registries in three geographic areas of The Netherlands. Sampling fractions were related to expected mortality at 5 years of follow-up, to ensure sufficient sample sizes for longitudinal analyses within age and sex strata.

At baseline (1992), there were 1489 people who had passed their 65th birthday for whom ApoE determination was available and of whom 1262 were ApoE e3/3 (n = 903) or e3/4 or e4/4 (referred to as "e4" below, n = 359). Of these, varying numbers provided performance test or disability data, ranging from 1253 for difficulty going up stairs, to 1118 for chair stand performance (Table 2).

Of the 1262 people typed as ApoE e3/3 or e4 at baseline, 326 had died by the 6-year follow-up. Of those surviving, 735 were interviewed, with an additional 42 patients and 36 proxies providing some data by telephone. The remainder either refused or were not contactable.

Numbers available for incidence analyses varied from 765 for reported ability to walk for 5 minutes to 612 with chair stand test data, after removing those persons with disability or poor performance at baseline. The details of the full follow-up have been described elsewhere (19). The local medical ethics committee approved the study, and consent was given by respondents at baseline.

Functional Status Measures

For self-reported mobility, a question on medium distance mobility asked "can you walk outside during 5 minutes without stopping?" with response categories "without difficulty," "with some difficulty," "with much difficulty," "only with help," and "not able to do." A similar question on climbing 15 steps without stopping was also asked. For analysis, the last two responses were grouped into an "inability" category, with the remainder labelled "able."

The instructions for the walking speed and five chair stands tests were similar to those used in the EPESE (11). A gait speed test measured the time taken to walk a 3-meter course twice, walking as fast as possible and without help. For analyses of gait speed, 0.4 m/s was used as an established benchmark for poor tested performance (20), yielding a prevalence of 5.4% (weighted). The five chair stands test measured the time to stand from a sitting position five times, without using one's arms. A cutoff of ≥20 seconds was chosen, to yield a prevalence of poor performance roughly similar to that for gait speed (7.5%).

Overall cognitive function (21,22) was measured with the Mini-Mental State Examination. Information processing speed was measured with the Coding Task; declines in this measure over time have been shown to be associated with ApoE e4 (22,23). The task consisted of three identical trials, in which the respondent had to combine two characters according to a given example, working as quickly and accurately as possible. The score on each trial was the number of completed characters, with a mean score averaging three trials.

Disease algorithms (decision trees) were used to classify cardiovascular conditions, including angina pectoris, myocardial infarction, congestive heart failure, arrhythmia, peripheral arterial disease, and cerebrovascular disease separately. Data were obtained from interview self-reports of chronic diseases, inspections of medicine bottles, and medical records of general practitioners (24). The presence of any cardiovascular disease was categorized as "Definite" (at least one cardiovascular disease), "Possible" (at least one cardiovascular disease is possible, but no definite diagnosis) or "No" (no possible or definite cardiovascular diagnosis).

The number of major chronic conditions was based on the presence of diabetes mellitus, arthritis, malignancies, chronic nonspecific lung disease, heart disease, peripheral arterial disease, or stroke, and could vary from 0 to 7.

ApoE

The ApoE phenotypes were determined from the serum blood samples, to investigate the association between ApoE and cognitive decline in the LASA (21,22). Most blood samples were taken at baseline (n = 1132 in this analysis), but some samples were collected 3 years later (n = 357). Serum samples were frozen at −80°C until determination of ApoE phenotype by isoelectric focusing of delipidated serum samples, followed by immunoblotting. Our experience in the laboratory was similar to that of Kardaun and colleagues (25) who, using the same technique, reported that phenotype–genotype correlations were high for the e4 allele; in our study, 90% of phenotype-identified e3/3 individuals were also genotyped as e3/3, with percentages for e3/4 and e4/4 of 81% and 87%, respectively. Most misclassification was linked to the identification of the e2 allele. The distribution of the ApoE phenotypes was in Hardy–Weinberg equilibrium (ApoE e2/2, 0.7%; e2/3, 11.1%; e3/3, 61.5%; e2/4, 2.7%; e3/4, 21.3%; e4/4, 2.7%). In this analysis, the e3/4 group (unweighted n = 317 at baseline) plus e4/4 (n = 42) were combined (and termed "e4") and compared to the e3/3 group (n=903). Analyses of the effects of e2/2 (n = 11) or 2/3 (n = 146) were underpowered.

Statistical Analysis

For analysis, data were weighted to correct for the differing sampling fractions by age and sex to yield general population estimates. All data were analyzed in Stata 8 (StataCorp LP, College Station, TX), and 5% significance levels were used. Logistic regression models explored associations between poor mobility and ApoE status.

Results

The baseline sample (Table 1) had a mean age of 74.9 years (standard deviation [*SD*] 5.8). There were no significant differences by ApoE status in sex, cognition measures, or disease status. Over the 6 years of follow-up, a higher percentage of people in the e4 group died compared to the e3/3 group (age- and sex-adjusted odds ratio [OR] = 1.47; 95% confidence interval [CI], 1.05–2.05).

At baseline, the prevalences of self-reported inability to walk for 5 minutes and to climb stairs was similar in the ApoE groups (Table 2). However, poor performance on the gait speed and chair stands tests were significantly associated with e4 status (for poor gait speed, age- and sex-adjusted OR = 2.26; 95% CI, 1.31–3.90; for poor chair stands tests, OR =1.94; 95% CI, 1.19–3.16). Adjusting for other factors known to influence gait speed measures such as height and weight made little difference (baseline gait speed OR = 2.35; 95% CI, 1.33–4.15 for e4 and poor performance).

At 6-year follow-up, examining incident-reported inability to walk for 5 minutes (excluding those unable at baseline) showed no significant difference by ApoE group, and there were neither differences in climbing 15 stairs nor in incident poor gait speed performance (Table 3). However, a significant difference was present for poor performance on five chair stands.

Missing Values and Sensitivity Analysis

To assess the effects of missing data, we recomputed the logistic models assuming that all those eligible nonparticipants in the performance tests were poor performers. The results remained similar: For poor gait speed at baseline in the e4 group the adjusted OR = 1.61 (95% CI, 1.07–2.41) and for chair stands OR =1.45 (95% CI, 1.03–2.05). For the 6-year follow-up, the significant finding for e4 status and slow performance on chair stands also remained significant (OR = 1.61; 95% CI, 1.04–2.50).

Interactions between sex and ApoE status were explored for all associations, but none proved significant. Recalculating the models adding the 193 people with e2/2 and e2/3 status to the e3/3 group did not change the findings. Also, restricting the analyses to only those persons from whom a blood sample was taken at baseline did not change the findings.

As e4 status is associated with cognitive impairment and cardiovascular disease, models were tested to explore whether poor physical performance was secondary to these conditions (Table 4). The ORs for e4 status and poor performance at baseline were little affected, with gait speed and chair stands remaining statistically significant. The association between incident poor performance on chair stands and ApoE e4 also remained significant after adjustment (OR = 2.02; 95% CI, 1.12–3.65).

Discussion

The LASA provides a rare opportunity to examine the effects of the ApoE e4 (vs e3/3) polymorphism in a large population-based sample of older people. The LASA has both self-reported disability data plus performance testing. In addition, data are available at baseline and for a 6-year follow-up.

The results show that, although self-reported inability to walk for 5 minutes or to climb 15 stairs without help were not associated with e4 status (compared to e3/3), relatively strong associations were found for poor tested performance on gait speed or five chair stands. At follow-up, incident poor performance on five chair stands was also associated with e4 status.

In assessing this result several issues need to be considered. Some of the blood samples were obtained at the 3-year follow-up rather than at baseline, but analyses using only baseline blood samples showed similar findings. Levels of attrition over 6 years in an ageing study are inevitably high, mainly due to mortality. Attrition from other sources also occurred but, as described, sensitivity analyses in which all the missing participants were assumed to have poor performance status did not change the findings.

Another potential problem with these analyses is that they could merely be detecting the effects of Alzheimer's disease. Some of the previous reports specifically excluded this group, so as to assess the morbidity burden from the other effects of ApoE e4. In this study, excluding those persons with low Mini-Mental State Examination scores did not change the overall pattern of results (data not shown). Correcting for measures of cognition also did not change the results materially, and even models also including markers of cardiovascular disease and numbers of comorbidities (Table 4) still demonstrate the association between e4 and tested performance. Clearly the effects of e4 must be mediated by pathologies that limit function, but it is unclear which disease intermediates are most important. Better measures of the various components of cognitive function (including executive function) and disease will be needed to explore the underlying mechanisms of these mobility impairments.

Why then are these findings apparently at odds with the previous literature? Most of the previous literature is based on self-reported data, and our findings of no association between e4 status and self-declared inability to walk for 5 minutes or to climb 15 stairs is consistent with those previous studies. Carmelli and colleagues (6) did examine data from gait speed and five chair-stand tests, although in a substantially smaller sample. In addition, they used a score of 0–4 to summarize the performance on the gait speed and five chair stand tests, rather than examining poor performance. In our analyses, the differences at baseline in mean gait speed were not significant (e3/3 gait speed mean 0.770 m/s vs 0.769 m/s for e4, *t* test of difference=0, *p* value 0.56). Despite this, there were large differences in the proportions walking very slowly, the "pathological" tails of the distributions.

Given the previous reports of lack of association (7) between the e4 allele and disability and the claimed unimportance of e4 to quality of life at the population level (8), this evidence of a substantial burden of physical impairment in old age in the e4 group is important. This correction of the literature indicates first that self-reported limitations may be insufficiently accurate for studying the effects of genetic factors in ageing and, second, that poor performance on the established tests could be used for this purpose. Further work is clearly needed on whether there are systematic explanations for the failure to detect ApoE e4 effect with self-reports. Given the high heritability of the gait speed and chair stand measures (26), the case for using them as ageing phenotypes in the search for relevant genes and polymorphism is greatly strengthened.

Conclusions

In a medium-sized population based study, substantial cross-sectional and longitudinal associations were evident between ApoE e4 status and poor tested mobility performance in older people. These associations were absent for self-reports of mobility limitations, which may be insufficiently specific to reflect the effects of genetic factors.

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Table 1

Percentage (95% CI) of the Sample at Baseline by Attribute and ApoE Status, With Age-Adjusted and Sex-Adjusted ORs

Variables	e3/3 *N* = 903	e4 *N* = 359	Adjusted OR	95% CI
Sex, %				
Male	43.8	43.8	1	
Female	56.2	56.2	1.01	(0.77–1.33)
Age group, %				
65–69 y	35.9	39.7	1	
70–74 y	30.3	30.5	0.91	(0.64–1.29)
75–79 y	21.8	16.8	0.69	(0.49–0.99)
80+ y	12	13.1	0.98	(0.69–1.41)
Mini-Mental State Examination				
Score (baseline), mean (*SD*)	26.8 (2.8)	26.6 (3.2)	0.96	(0.92–1.01)
Information processing speed, mean (*SD*)	22.3 (7.0)	22.3 (7.6)	0.99	(0.98–1.02)
Cardiovascular disease				
None	70.3	71.8	1	
Definite	20.5	19.1	0.92	(0.66–1.31)
Possible	9.2	9.1	0.98	(0.62–1.56)
No. of diseases, mean (*SD*)	1.1 (1.0)	1.0 (1.1)	0.9	(0.78–1.03)
Died before follow-up (6 y later)	19.1	23.9	1.47	(1.05–2.05)

Note: CI = confidence interval; ApoE = apolipoprotein E; OR = odds ratio; *SD* = standard deviation.

Table 2

Percentage (95% CI) of the Sample by Attribute and ApoE Status at Baseline, With Age-Adjusted and Sex-Adjusted ORs

Baseline Functioning	No. of Observations	e3/3		e4			Age- and Sex-Adjusted ORs of e4 With Poor Functioning	
		%	95% CI	%	95% CI	X^2 of Difference *p* Value	OR	95% CI
Self-reported inability to walk for 5 min	1219	3.5	(2.5–4.9)	3.0	(1.6–5.5)	0.658	0.90	(0.44–1.87)
Self-reported inability to climb 15 stairs	1953	11.2	(9.2–13.6)	11.0	(8.1–14.8)	0.936	1.02	(0.67–1.55)
Gait speed (≤0.4 m/s)	1185	4.2	(3.1–5.8)	8.3	(5.7–11.9)	0.006	2.26	(1.31–3.9)
Five chair stands (≥20 s)	1118	6.3	(4.7–8.3)	10.7	(7.7–14.6)	0.015	1.94	(1.19–3.16)

Note: CI = confidence interval; ApoE = apolipoprotein E; OR = odds ratio.

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Table 3

Incident Poor Performance or Death at 6-Year Follow-Up in Those Free of Each Respective Disability or Impairment at Baseline

Variables	No. of Observations	e3/3		e4			Age- and Sex-Adjusted OR of e4 With Poor Functioning	
		%	95% CI	%	95% CI	X^2 of Difference *p* Value	OR	95% CI
Self-reported inability to walk	765	9.7	(7.5–12.5)	8.7	(5.7–13.2)	0.656	0.94	(0.54–1.64)
Self-reported inability to climb 15 stairs	711	14.2	(11.4–17.6)	14.2	(9.9–19.9)	0.985	1.08	(0.65–1.80)
Gait speed ≤0.4 m/s	687	12.6	(9.9–15.9)	12.9	(8.7–18.7)	0.921	1.14	(0.67–1.96)
Five chair stands ≥20 s	612	10.3	(7.7–13.7)	17.2	(11.7–24.6)	0.034	1.89	(1.08–3.31)

Note: CI = confidence interval; OR = odds ratio.

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Table 4

Logistic Models of Physical Performance, Adjusted for Age, Sex, Mini-Mental State Examination (MMSE) Score, and Information Processing Speed Score

	Gait Speed at Baseline (≤0.4 m/s vs Faster)		Chair Stands at Baseline (≥20 s vs Faster)	
Variables	**OR**	**(95% CI)**	**OR**	**(95% CI)**
Baseline				
Apo e4 vs e3/3	2.72	(1.47–5.04)	2.01	(1.18–3.43)
Information processing speed score	0.94	(0.89–0.99)	0.92	(0.88–0.96)
MMSE score	1.02	(0.88–1.18)	1.03	(0.90–1.18)
Cardiovascular disease				
None	1		1	
Definite	1.49	(0.68–3.28)	1.00	(0.52–1.93)
Possible	1.18	(0.47–2.99)	0.68	(0.28–1.65)
No. of diseases	1.86	(1.44–2.39)	1.67	(1.30–2.16)
Incident (at 6-year follow-up)				
Apo e 43/44 vs 33	1.23	(0.66–2.27)	2.02	(1.12–3.65)
Information processing speed score	0.97	(0.92–1.01)	0.98	(0.93–1.02)
MMSE score	0.94	(0.85–1.04)	0.99	(0.88–1.12)
Cardiovascular disease				
None	1		1	
Definite	0.66	(0.31–1.41)	1.22	(0.54–2.74)
Possible	1.18	(0.43–3.21)	1.82	(0.66–5.00)
No. of diseases	1.65	(1.17–2.34)	1.15	(0.78–1.69)

Note: OR = odds ratio; CI = confidence interval; Apo = apolipoprotein.