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Executive Function, Memory, and Gait Speed Decline in Well-Functioning Older Adults

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Background. In community-dwelling older adults, global cognitive function predicts longitudinal gait speed decline. Few prospective studies have evaluated whether specific executive cognitive deficits in aging may account for gait slowing over time.

Methods. Multiple cognitive tasks were administered at baseline in 909 participants in the Health, Aging, and Body Composition Study Cognitive Vitality Substudy (mean age 75.2 ± 2.8 years, 50.6% women, 48.4% black). Usual gait speed (m/s) over 20 minutes was assessed at baseline and over a 5-year follow-up.

Results. Poorer performance in each cognitive task was cross-sectionally associated with slower gait independent of demographic and health characteristics. In longitudinal analyses, each 1 *SD* poorer performance in global function, verbal memory, and executive function was associated with 0.003–0.004 m/s greater gait speed decline per year (p =.03–.05) after adjustment for baseline gait speed, demographic, and health characteristics.

Conclusions. In this well-functioning cohort, several cognitive tasks were associated with gait speed cross-sectionally and predicted longitudinal gait speed decline. These data are consistent with a shared pathology underlying cognitive and motor declines but do not suggest that specific executive cognitive deficits account for slowing of usual gait in aging.

Key Words: Aging—Cognitive function—Gait speed.

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MANY studies have identified an independent relationship of cognitive and motor performance in older adults. Although it is well known that several cognitive processes are associated with walking speed and risk of falls (1–4), studies of attention-demanding "dual tasking" while walking suggest that deficits in attention and executive function may in part explain gait slowing in aging (2,5–8). Considered important for the planning and execution of movements, executive control functions (ECF) and attention demand integrity of frontal–subcortical circuitry also recognized as associated with mobility and balance (9–11). In older adults, this extensive neural network is preferentially vulnerable to ischemic changes within the deep white matter (12), suggesting that cerebrovascular alterations in aging (13) may contribute to declines in both executive and motor functions (10,14) while sparing temporal lobe-mediated long-term memory (15).

Previous studies of older adults in the community have found executive function and processing speed associated with rate of gait speed decline independent of traditional risk factors and chronic conditions (16,17). These findings are consistent with a shared, potentially cerebrovascular etiology of cognitive and mobility declines (10) and suggest that executive and attention deficits may manifest in gait slowing (17), even before such deficits can be detected (18). A recent publication further identified an association of baseline global cognitive function with physical performance change but not of baseline physical performance with cognitive change; these data support the hypothesis that cognitive decline may exacerbate or co-occur with gait alterations in aging (19). Few studies have evaluated whether an association of cognitive function with longitudinal gait decline extends to temporal-mediated long-term memory or is restricted to frontal-associated executive function and attention.

This study evaluates the relationship of cognitive function with usual gait speed at baseline and over 5 years in the Health Aging, and Body Composition (ABC) Study Cognitive Vitality Substudy, in which participants completed a detailed assessment of memory and other cognitive tasks not evaluated in the full Health ABC cohort. Cross-sectional associations of cognitive and gait performance would support a shared pathogenesis of cognitive and gait alterations that is not explained by traditional vascular risk factors or chronic conditions. Associations of specific cognitive tasks with accelerated gait decline would further suggest that deficits in these domains may in part account for gait slowing in aging.

Methods

Population

From 1997 to 1998, the Health ABC Study enrolled 3,075 Medicare-eligible well-functioning men and women aged 70–79 years from Pittsburgh, Pennsylvania and Memphis, Tennessee. The population was 52% women and 42% black with a mean age of 73.6 years. Participants were recruited from Medicare-eligible adults with contact information provided by the Centers for Medicare & Medicaid Services (formerly the Health Care Financing Administration) on a random sample of white and all black beneficiaries in predesigned zip code areas surrounding the study centers. Other household members aged 70–79 years were also eligible for recruitment. Exclusion criteria included reported difficulty walking one quarter of a mile, climbing 10 steps without resting, or performing basic activities of daily living or need for a walking aid.

In Year 3 of Health ABC, the Cognitive Vitality Substudy was initiated. Participants represent approximately the top 20% of performers on an endurance walk test (20) in Year 2 from each of eight groups defined by sex, race, and study site (Memphis or Pittsburgh) and an equal number drawn at random from the remaining members of each group yielding 951 black and white women and men aged 72–81 years who received additional cognitive testing. Substudy participants were slightly younger (75.5 vs 75.7 years), more likely to be female (54% vs 50%), white (65% vs 55%), and have less than 12 years of education (30% vs 23%) compared with the Health ABC participants who were not included in the substudy. Exclusion criteria included self-reported difficulty seeing large print or grasping a pen. The Institutional Review Boards of the University of Pittsburgh, PA, and University of Tennessee at Memphis approved the study, and written informed consent was obtained from each volunteer. Of the 951 participants in the substudy, 920 completed cognitive and gait speed testing at Year 3. Of these participants, we excluded those with either lower extremity revascularization (n = 8) or angioplasty (n = 3), leaving 909 participants for cross-sectional analyses (mean age 75.2 ± 2.8 years, 50.6% women, 48.4% black). Longitudinal analyses included 865 participants who also had at least one gait speed measurement over the subsequent 5-year period (mean age 75.2 ± 2.8 years, 50.5% women, 47.5% black).

Cognitive Tests

Cognitive function was assessed at baseline of the substudy (Year 3). The Modified Mini-Mental Status Examination (3MS) (21) is a commonly used evaluation of global cognitive function, including orientation, attention, calculation, language, and short-term memory. Scores can range from 0 to 100 points, with lower scores indicating poorer performance. The Buschke Selective Reminding Test (22) is a multiple-trial list-learning task used to measure verbal learning and memory. In this task, the examiner presents a list of 12 written words and reads each word aloud. The participant is then asked to recall the words presented. For the next trial, the examiner repeats the words the participant failed to recall and then asks the participant to provide the full list of 12 words. This procedure is repeated five times. Long-term storage is scored as the number of words recalled at least twice in a row that were also recalled in Trial 6. The 15-item Executive Interview (EXIT 15) was developed for the Health ABC Study and constitutes a shortened version of the 25-item Executive Interview (23). The test assesses several executive control functions, such as inhibition of automatic responses, word and design fluency, and sequencing, and is scored from 0 to 30, with lower scores indicating better performance. The Boxes and Digit Copying tests are timed tests of psychomotor speed (24). The participant is asked to complete as many boxes and copy as many digits as possible within 30 seconds for each test. Psychomotor speed is scored as the sum of total boxes and digits completed (ρ = .77). Finally, the Pattern and Letter Comparison tests are timed tests of attention and perceptual speed (24). The participant is asked to determine whether pairs of patterns and letters are the same or different within 30 seconds for each test. Perceptual speed is scored as the sum of correct pattern and letter comparisons (ρ = .64).

Gait Speed

Gait speed was measured as the time needed to walk a 20-m straight course setup along a hallway (25). Participants were asked to stand stationary behind a starting line marked with tape, and at the examiner's command, to walk at usual pace to just past the finish line. Timing was recorded with a stopwatch and began at the first footfall over the starting line and ended with the first footfall over the finishing line. These analyses includes up to five measurements of gait speed collected at Years 3–6 and Year 8 in Health ABC.

Covariates

We considered as covariates variables that were identified in the literature as potential confounders of the relationship

between cognitive function and gait speed or were associated with both cognitive function and gait speed in this cohort with a p value <.15. Selected covariates included demographic variables (age, race, sex, education, and clinic site), vascular risk factors (body mass index, smoking, and physical activity), depressive symptoms, and chronic conditions (prevalent coronary heart disease, cerebrovascular disease, hypertension, diabetes mellitus, and peripheral arterial disease). Presence of chronic conditions was determined from participant reports at the baseline visit. Depressive symptoms were assessed using the Centers for Epidemiological Studies Depression 10-item scale (26). Body mass index was calculated as measured weight in kilograms divided by measured height in meters squared. Walking frequency and duration were determined from responses to questions, which distinguished walking for exercise and other types of walking from a standardized interviewer-administered physical activity battery (27). Participants walking a total of less than 30 min/wk were defined as sedentary. Finally, ankle–arm index was calculated as the ratio of the systolic blood pressure obtained in the ankle to the systolic blood pressure of the right arm. Measures were performed twice, and the results were averaged; the lower average value between the two legs was used to define an individual's ankle–arm index. Peripheral arterial disease was then defined as ankle–arm index less than or equal to 0.9, according to traditional diagnostic criteria (28).

Statistical Analysis

Differences in baseline characteristics across quartiles of gait speed and cognitive function were tested with chi-square tests for categorical variables and analysis of variance or Kruskal–Wallis tests for continuous variables. Associations between cognitive tasks were evaluated using Pearson correlations adjusted for demographics. Linear regression was used to test the cross-sectional association between cognitive function as the independent variable and gait speed as the dependent variable adjusting for demographics, risk factors, depressive symptoms, and chronic conditions. Scores for each cognitive test were divided by their standard deviation to allow direct comparison of regression coefficients across tests. Multivariable models were built using a backward procedure (p out = .5) after entering in cognitive test score and demographics. Final models were additionally adjusted for EXIT 15 score to evaluate whether associations of remaining cognitive tests with gait speed may be explained by executive performance. Low values of the variance inflation factor (<2) excluded the risk of multicollinearity.

Because patterns of specific cognitive deficits may differ in normal aging and preclinical dementia (15), we tested interactions between each cognitive test and cognitive status, categorized as normal cognitive function versus cognitive impairment or decline. For these analyses, cognitive impairment was defined as a 3MS score less than 80 at Year 3 and cognitive decline as a decrease in 3MS score of 5 or more points from Year 1 to Year 3, consistent with previously validated criteria (29).

Linear mixed-effects models were used to evaluate associations of cognitive performance with rate of gait speed decline over 5 years. A simple model for each cognitive task included a random intercept for each participant, a random slope for time, baseline gait speed and demographics as fixed effects, and interaction terms for baseline cognitive score and covariates with time. Full models were built using a backward procedure (p out = .05) to additionally adjust for vascular risk factors and chronic conditions and the interactions of each covariate with time. To evaluate whether executive performance may explain associations of the remaining cognitive tasks with rate of gait speed decline, final models were further adjusted for EXIT 15 score. Analyses were repeated after excluding participants with evidence of cognitive impairment or decline. No structure was imposed on the covariance matrix of the random effects. Analyses were performed in Stata 10 (STATA, Houston, TX).

To confirm that the cognitive tasks were appropriately categorized, we applied principal components analysis to reduce the five tasks into a smaller set of uncorrelated factors that account for most of the observed variance in cognitive scores (30). A varimax rotation of the initial factors was used to obtain interpretable cognitive components, defined according to loadings that relate each cognitive test to each component. Loadings greater than 0.50 were used to identify cognitive tests represented by each component.

Finally, we used random-effects pattern-mixture models (31) to evaluate the influence of missing gait speed data at final follow-up (n = 303, 33.3%) in the analysis of gait speed decline. This method involves stratifying participants by their missing data pattern, then evaluating the influence of each missing data pattern on the outcome of interest. The results of the pattern-mixture model may then be averaged over the missing data patterns to obtain overall estimates that are corrected for missing data patterns. We performed these analyses using adapted code (31) in SAS 9 (SAS Institute, Inc., Cary, NC).

Results

Mean age of the cohort was 75.2 years (*SD* 2.8); 49.4% were men and 48.4% black (Table 1). Mean (*SD*) 3MS score and gait speed were 90.3 (7.9) and 1.20 (0.22) m/s, similar to performances of Health ABC participants not included in the substudy. Participants in lower quartiles of gait speed were more likely to be female, black, sedentary, and have less education, higher body mass index, more chronic conditions, and poorer performance on all cognitive tests (Tables 1 and 2). Cognitive tasks showed low-to-moderate correlation with each other after adjustment for demographics (Table 3).

Table 1. Characteristics of the Cohort by Quartiles (range in parentheses) of Baseline Gait Speed (m/s)

	M ± *SD*, Median (IQR), or %					
	Overall (0.47–1.93) *n* = 909	Q1 (<1.05) *n* = 228	Q2 (1.05–1.21) *n* = 229	Q3 (1.21–1.36) *n* = 226	Q4 (≥1.36) *n* = 226	*p*
Age, yr	75.2 ± 2.8	75.8 ± 2.9	75.1 ± 2.8	75.2 ± 2.7	74.7 ± 2.5	<.001
Male	49.4	36.4	49.8	51.8	59.7	<.001
Black	48.4	73.3	59.4	38.1	22.6	<.001
Education ≥ 12 yr	78.4	65.2	72.4	86.2	89.8	<.001
BMI, kg/m^2	26.9 ± 4.6	28.5 ± 5.7	26.8 ± 4.3	26.6 ± 4.2	25.6 ± 3.3	<.001
Current or former smoker	52.2	47.8	53.7	51.8	55.6	.391
Sedentary	68.2	83.7	71.5	62.5	54.7	<.001
Coronary heart disease	17.1	20.0	16.1	16.9	15.5	.594
Cerebrovascular disease	6.2	9.8	7.1	4.4	3.6	.026
Diabetes	19.5	31.3	21.8	12.4	12.4	<.001
Hypertension	59.4	78.5	60.7	53.1	45.1	<.001
CES-D 10	3 (5)	4 (5)	3 (5)	3 (4)	3 (4)	<.001*
Ankle–arm index < 0.9%	13.9	26.0	15.3	7.1	7.1	<.001

Notes: *p* Values are from chi-square tests of proportions for categorical variables and analysis of variance for comparison of mean values of continuous variables, unless otherwise noted. BMI = body mass index; CES-D 10 = Centers for Epidemiological Studies Depression 10-item scale; IQR = interquartile range.

*Kruskal–Wallis test of equal medians.

In separate linear regression models of each cognitive test, as the independent variable and gait speed as the dependent variable, poorer performance in each test was significantly associated with slower gait speed after adjustment for demographics, risk factors, and chronic conditions (Table 4). Additional adjustment for EXIT 15 score only slightly attenuated coefficients for remaining cognitive tests. Interactions of each cognitive test with cognitive status were nonsignificant. In analyses excluding participants with cognitive impairment or decline, associations were similar to those observed in the whole sample.

In separate mixed models of each cognitive task and gait speed decline, each interaction term for cognitive test score and time represents the calculated contribution of cognitive score to rate of gait speed decline per year (Table 5). Each 1 *SD* poorer performance in global function, verbal memory, and executive function was associated with 0.003–0.004 m/s greater gait speed decline per year ($p = 0.03$–0.05) after adjustment for baseline gait speed, demographic, and health characteristics. Psychomotor and perceptual speed performances were not significantly associated with rate of gait speed decline in simple or full models. The association of verbal memory performance with rate of gait speed decline did not substantially change after further adjustment for EXIT 15 score. Estimates for each cognitive test were modestly attenuated after exclusion of participants with evidence of cognitive impairment or decline.

Principal components analysis identified three independent factors that explained 88% of overall variance in cognitive performance. The factor that explained most of the variance had strong loadings in 3MS and EXIT 15 scores and was identified as an "executive" factor; a second factor loaded heavily on Boxes and Digit Copying and Pattern and Letter Comparison test scores, representing "processing speed"; and a final, "memory" factor loaded strongly on long-term storage scores. Worse scores in each of the three factors were highly correlated with slower baseline gait speed.

Pattern-mixture models of gait speed decline indicated that participants who did not complete gait speed testing at final follow-up had significantly slower gait and faster gait speed decline relative to completers. However, interactions of cognitive test score with time did not vary among completers and noncompleters, suggesting that estimated contributions of cognitive function to annual gait speed decline are not sensitive to missing data.

Discussion

In this community-dwelling cohort, memory and other cognitive tasks were associated with gait speed cross-sectionally and predicted longitudinal gait speed decline

Table 2. Cognitive Performance by Quartiles (range in parentheses) of Baseline Gait Speed (m/s)

	M ± *SD* or Median (IQR)					
	Overall (0.47–1.93) *n* = 909	Q1 (<1.05) *n* = 228	Q2 (1.05–1.21) *n* = 229	Q3 (1.21–1.36) *n* = 226	Q4 (≥1.36) *n* = 226	*p*
Global function	90.2 ± 7.9	89 (12)	90 (10)	94 (9)	95 (6)	<.001*
Verbal memory	6.6 ± 3.0	5.6 ± 3.0	6.3 ± 2.8	7.0 ± 2.9	7.5 ± 2.9	<.001
Executive function	6.3 ± 4.2	8 (6)	6 (5)	5 (6)	4 (5)	<.001*
Psychomotor speed	76.5 ± 20.6	66.0 ± 19.1	72.0 ± 20.0	79.6 ± 18.9	88.6 ± 16.9	<.001
Perceptual speed	15.6 ± 5.4	13.3 ± 5.3	14.5 ± 5.2	16.4 ± 5.2	18.2 ± 4.6	<.001

Notes: *p* Values are from analysis of variance for comparison of mean values of continuous variables, unless otherwise noted. IQR = interquartile range.

*Kruskal–Wallis test of equal medians.

Table 3. Partial Correlation Coefficients* Between Cognitive Domains at Baseline

	Global Function	Verbal Memory	Executive Function	Psychomotor Speed	Perceptual Speed
Global function	—	.44	.54	.31	.36
Verbal memory	.44	—	.27	.21	.26
Executive function	.54	.27	—	.32	.39
Psychomotor speed	.31	.21	.32	—	.58
Perceptual speed	.36	.26	.39	.58	—

Notes: $p < .001$ for all.
*Adjusted for age, sex, race, education, and clinic site.

over 5 years. These data are consistent with a shared potentially cerebrovascular pathology underlying cognitive and gait alterations but do not suggest that specific executive cognitive deficits account for slowing of usual gait in aging.

Previous Health ABC analyses found relationships of poorer psychomotor speed and executive function with slower gait or accelerated gait speed decline (3,16), providing support to the hypothesis that compromised attention and executive function may be particularly important to explain gait slowing with age. However, in this analysis of participants who completed additional detailed cognitive assessments, we identified similar associations with rate of gait decline across several distinct cognitive tasks, suggesting potential influences of a broader range of cognitive processes to mobility decline in aging. This finding is consistent with a complex neural basis for gait control hypothesized to involve important roles of frontal–hippocampal circuits in spatial orientation and navigation, in addition to those of the prefrontal cortex and striatal connections in executive function and attention (32).

The association of memory with longitudinal gait decline, not reported in previous literature, suggests a potential value for a general view of cognitive function, not restricted to executive function and attention, in identifying older adults at greatest risk of mobility decline. The long-term storage task in this study, although dependent in part on frontal-associated attention and rehearsal, allows inference to medial temporal lobe function, known as critical to memory storage and retrieval and vulnerable to Alzheimer's disease pathology (15). In contrast, executive function and processing speed are generally associated with frontal lobe and related networks found preferentially sensitive to aging and hypertension (10,12). It is important to acknowledge that executive dysfunction may often precede memory decline in both normal aging and preclinical dementia, preventing a clear distinction between cognitive changes related to aging versus neurodegenerative disease (33). Rather, consistent with the frequent co-occurrence of cerebrovascular and neurodegenerative alterations in aging and dementia (34), our findings among this initially broad range of cognitive performers are likely to reflect influences of both age- and disease-related cognitive changes to gait declines in older adults.

Although the modest effects sizes of the cognitive measures may not be considered clinically meaningful gait speed change, the relevance of cognitive function in this analysis may be interpreted in relative terms: the estimated contribution of a 1 *SD* poorer cognitive performance to annual gait speed decline was equal to that of 1.5–2 years older age. From an etiological perspective, general cognitive deficits and associated gait declines may reflect a diffuse shared pathogenesis not explained by traditional vascular risk factors or chronic conditions.

Considered evidence of cerebral small-vessel injury, white matter hyperintensities on brain magnetic resonance imaging preferentially influence executive and motor functions (10,35), although associations with memory have been reported (36). These common cerebrovascular alterations in aging and hypertension (37,38) predict gait speed decline (39), disability (40), and dementia (41), suggesting that underlying microvascular disease may in part explain associations of cognitive and gait performance in older adults. Although brain magnetic resonance imaging data were not available for analysis, we identified body mass index, physical activity, cerebrovascular disease, diabetes, hypertension, and peripheral arterial disease as important covariates in cross-sectional analyses of each cognitive test and gait speed, consistent with shared vascular contributions to cognitive and mobility declines. Additional brain anatomical changes associated with aging and neurodegenerative disease, including cerebral atrophy (34) and loss of dopaminergic activity (42,43), have previously been associated with executive and memory deficits and warrant further investigation in relation to both cognitive and gait declines in older adults (44,45).

Table 4. Coefficients With *SE* in Parentheses From Multivariable Linear Regression Models of Baseline Gait Speed (m/s)

	Model 1		Model 2		Model 3	
Cognitive Measure (*SD*)	β (*SE*)	*p*	β (*SE*)	*p*	β (*SE*)	*p*
Global function (7.9)	.032 (0.008)	<.001	.037 (0.008)	<.001	.031 (0.008)	<.001
Verbal memory (3.0)	.027 (0.007)	<.001	.026 (0.007)	<.001	.021 (0.007)	.002
Executive function (4.2)	.030 (0.008)	<.001	.032 (0.008)	<.001	.021 (0.008)	.007
Psychomotor speed (20.6)	.056 (0.008)	<.001	.056 (0.007)	<.001	.048 (0.007)	<.001
Perceptual speed (5.4)	.036 (0.008)	<.001	.038 (0.007)	<.001	.029 (0.007)	<.001

Note: Model 1 adjusted for age, sex, race, education, and clinic site.
Model 2 additionally adjusted for body mass index, smoking status, and physical activity.
Model 3 additionally adjusted for depressive symptoms, coronary heart disease, cerebrovascular disease, diabetes mellitus, hypertension, and peripheral arterial disease.

Table 5. Calculated Contribution of a 1 *SD* Lower Cognitive Performance to Decline in Gait Speed (m/s) Per Year

	Model 1		Model 2		Model 3	
Cognitive Measure (*SD*)	Estimate (*SE*)	*p*	Estimate (*SE*)	*p*	Estimate (*SE*)	*p*
Global function (7.6)	0.003 (0.002)	.04	0.003 (0.002)	.03	0.003 (0.002)	.03
Verbal memory (3.0)	0.004 (0.001)	.01	0.004 (0.001)	.03	0.004 (0.001)	.03
Executive function (4.0)	0.003 (0.001)	.04	0.003 (0.001)	.04	0.003 (0.001)	.05
Psychomotor speed (20.4)	0.002 (0.002)	.29	0.002 (0.002)	.27	0.002 (0.002)	.32
Perceptual speed (5.3)	0.001 (0.001)	.57	0.001 (0.001)	.55	0.001 (0.001)	.60

Note: Model 1 adjusted for age, sex, race, education, clinic site, baseline gait speed, and interaction of each covariate with time.

Model 2 additionally adjusted for body mass index, smoking status, physical activity, and interaction of each covariate with time.

Model 3 additionally adjusted for depressive symptoms, coronary heart disease, cerebrovascular disease, diabetes mellitus, hypertension, peripheral arterial disease, and interaction of each covariate with time.

Consistent with our findings, poorer global and executive function in the full Health ABC cohort (16) and verbal fluency, involving memory, executive, and processing speed components, in the Three-City Study (17) were each associated with accelerated gait speed decline independent of clinical disease and risk factors. However, we did not observe associations of processing speed tasks with rate of gait speed decline, as identified for the Trail Making Test part A (46), a test of psychomotor speed, in the Three-City Study. The inconsistent findings may reflect the sensitivity of the Trail Making Test part A to subtle psychomotor deficits, previously found associated with microvascular alterations in aging (47). Alternatively, the lack of association in this cohort may reflect parallel declines in cognitive processing and gait speed, in contrast to the suggested contribution of initial executive and memory deficits to subsequent gait slowing. In fact, among initially well-functioning participants in the Women's Health and Aging Study, periods of marked declines in executive function and verbal memory were common throughout 9-year follow-up, whereas psychomotor speed declined linearly over time (33). We also observed continuous declines in average psychomotor speed and gait speed, in contrast to modest changes in other cognitive tasks. These data suggest that cognitive processing and gait speed might similarly reflect the integrity of multiple physiological systems (33), not limited to executive and memory-associated neural networks.

Several additional limitations should be considered. First, some components of the cognitive tests required motor skills (eg, drawing and copying figures), possibly obscuring the distinction between cognitive and physical function in cross-sectional analyses. Second, the potential influence of executive function on mobility may best be evaluated under dual task or otherwise attention-demanding conditions that represent unpredictable environments in daily life (5). In fact, among nondemented older adult participants in the InCHIANTI study, executive function was more strongly associated with walking speed during obstacle navigation relative to walking at usual pace (1). We similarly found that in cross-sectional models of cognitive performance and gait speed at fast pace, the association of executive function with this task condition was substantially strengthened, possibly reflecting a higher demand of attention in the fast relative to usual pace walk (48).

Finally, strengths of our study include the large community-dwelling population, detailed cognitive assessment found to appropriately categorize executive function, memory and processing speed factors, ability to account for several important confounders, and longitudinal assessment of gait speed, a reliable (49) and valid measure of physical function that predicts functional limitation (50,51) in older adults.

Conclusions

Several cognitive tasks were associated with gait speed cross-sectionally and predicted longitudinal gait decline in this initially well-functioning cohort. These findings are consistent with a diffuse shared pathogenesis of cognitive and gait alterations but do not suggest that specific executive cognitive deficits account for gait slowing during usual walking in aging. More work is needed to evaluate the potential role of vascular disease in dysfunction of both systems; magnetic resonance imaging studies of cerebral small-vessel disease may clarify the mechanisms underlying co-occurring declines in cognitive and gait performance. Further understanding of these pathways may provide targeted strategies to preserve function in both central facets of successful aging.

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