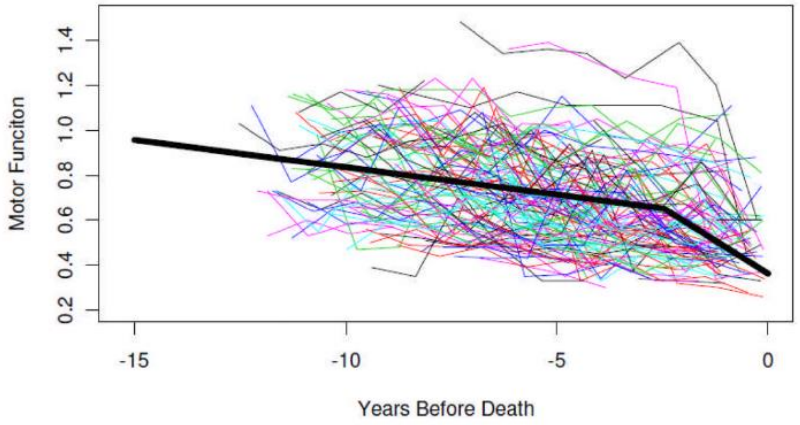
Dal declino cognitivo a quello motorio e dalla sarcopenia al declino cognitivo: un continuum bidirezionale

Giorgio Basile
Ricercatore Geriatria Università di Messina

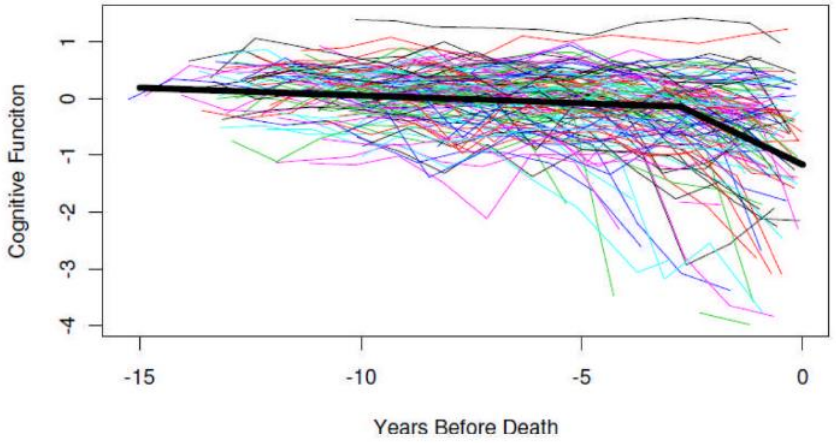
Terminal Decline in Motor Function

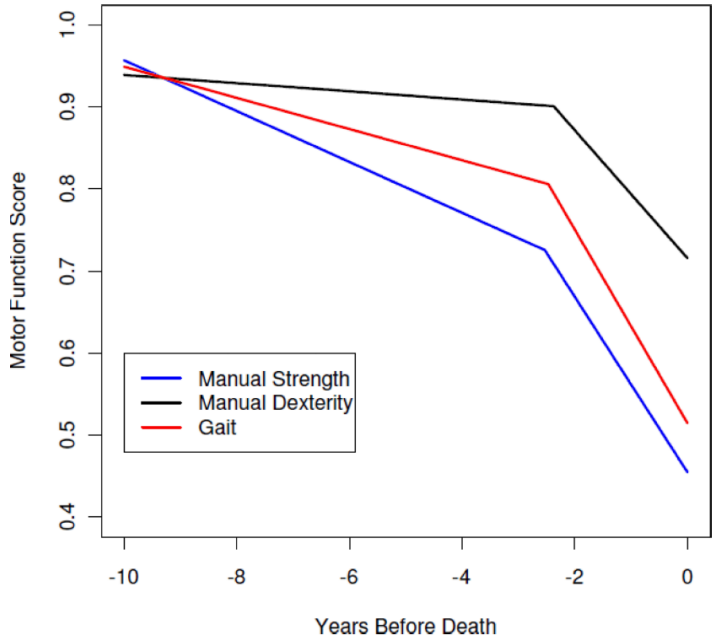
Robert S. Wilson, PhD, Eisuke Segawa, PhD, Aron S. Buchman, MD, Patricia A. Boyle, PhD, Loren P. Hizel, BA, and David A. Bennett, MD

Motor function



Cognitive function



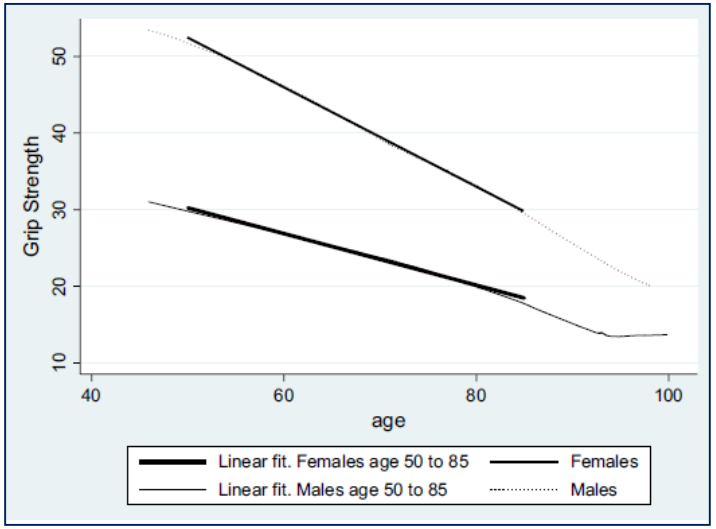


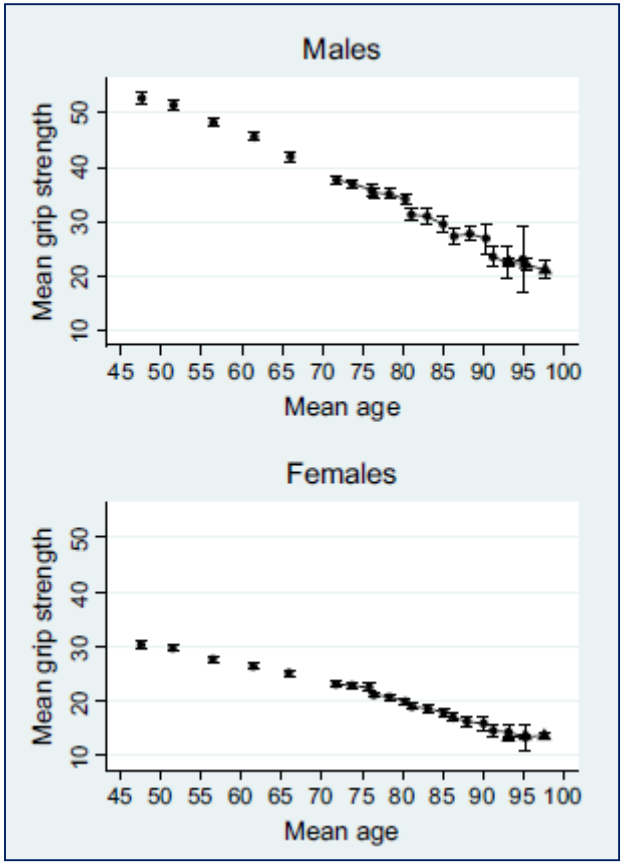
The study aim was to test the hypothesis that motor function undergoes accelerated decline proximate to death. As part of a longitudinal clinical-pathologic study, 124 older Catholic nuns, priests, and monks completed at least 7 annual clinical evaluations, died, and underwent brain autopsy and uniform neuropathologic examination. Each evaluation included administration of 11 motor tests and 19 cognitive tests from which global measures of motor and cognitive function were derived. The global motor measure (baseline mean = 0.82, SD=0.21) declined a mean 0.024-unit per year (95% confidence interval [CI]: −0.032, −0.016) until a mean of 2.46 years (95% CI: −2,870, −2.108) before death when rate of decline increased nearly 5-fold to −0.117-unit per year (95% CI: −0.140, −097). The global cognitive measure (baseline mean =0.07, SD =0.45) declined a mean of 0.027-unit per year (95% CI: −0.041, −0.014) until a mean of 2.76 years (95% CI: −3.157, −2.372) before death when rate of decline increased more than 13-fold to −0.371-unit per year (95% CI: −0.443, −0.306). Onset of terminal motor decline was highly correlated with onset of terminal cognitive decline (r=0.94, 95% CI: 0.81, 0.99), but rates of motor and cognitive change were not strongly correlated (preterminal r = 0.20, 95% CI: −0.05, 0.38; terminal r = 0.34, 95% CI: 0.03, 0.62). Higher level of plaques and tangles was associated with earlier onset of terminal decline in motor function, but no pathologic measures were associated with rate of preterminal or terminal motor decline. The results demonstrate that motor and cognitive function both undergo a period of accelerated decline in the last few years of life.

Psychol Aging. 2012 December ; 27(4): 998–1007.

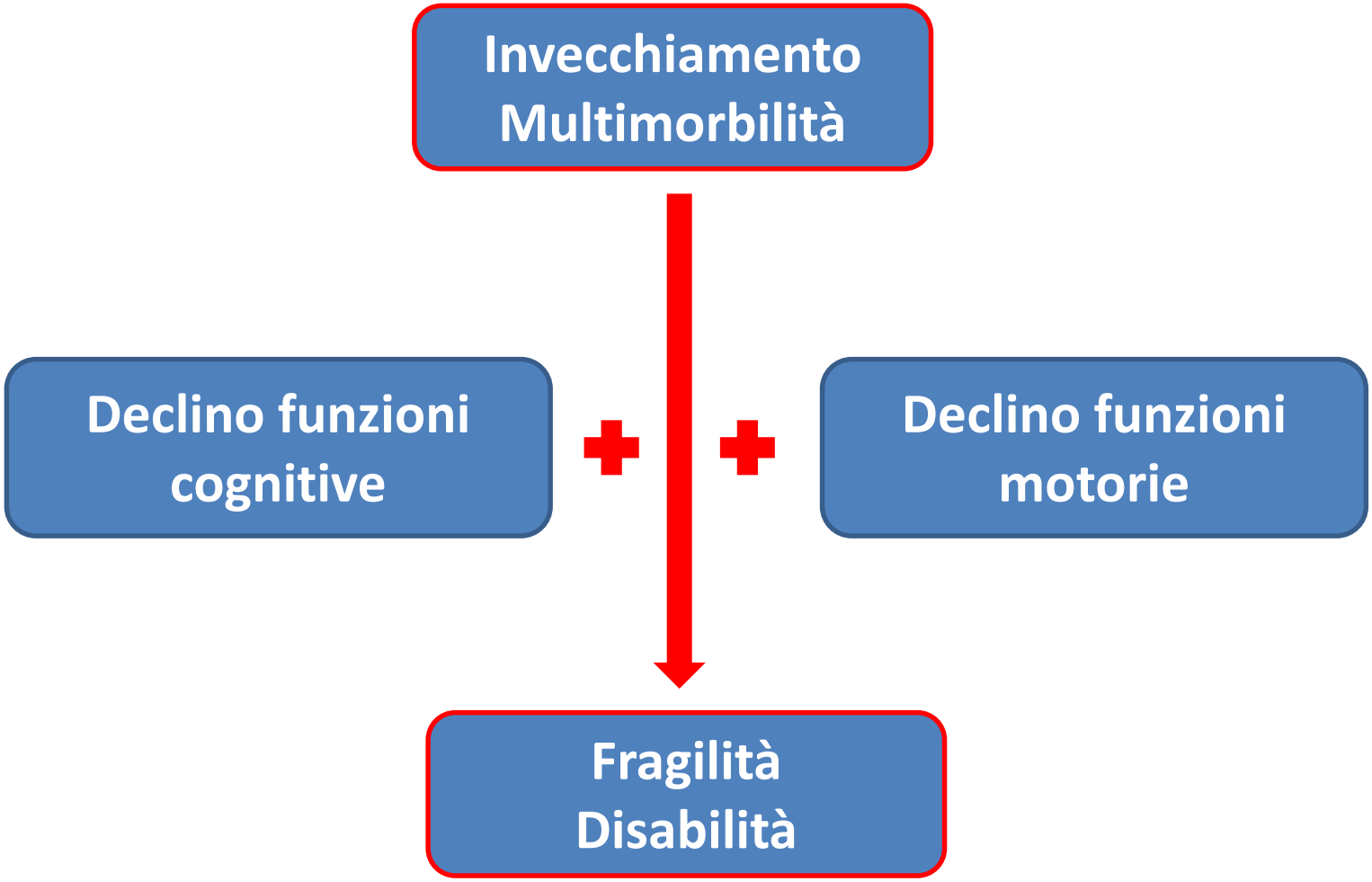
Age Trajectories of Grip Strength: Cross-Sectional and Longitudinal Data Among 8,342 Danes Aged 46 to 102

PURPOSE: The purpose is to study the age trajectory of hand-grip strength after the age of 45 years.
METHODS: In this study, we use data from three large nationwide population-based surveys of Danes aged 45 to 102 years with a total of 8342 participants with grip-strength measurements and up to 4 years of follow-up. Grip strength was measured by using a portable hand dynamometer.
RESULTS: Grip strength declines throughout life for both males and females, but among the oldest women, the longitudinal curve reaches a horizontal plateau. The course of the decline is estimated by using full information in the longitudinal data and is found to be almost linear in the age span of 50 to 85 years. In this age span, mean annual grip-strength loss is estimated to be 0.59 (0.02) (SE) kg for men and 0.31 (0.01) kg for women.
CONCLUSION: This study confirms the previously reported grip-strength decline with increasing age. Estimates were obtained by using full-information methods from large population-representative studies. Equations of expected grip strength, as well as tables with sex-, age-, and height-stratified reference data, provide an opportunity to include grip-strength measurement in clinical care in similar populations.
Ann Epidemiol 2006;16:554–562. © 2006 Elsevier Inc. All rights reserved.



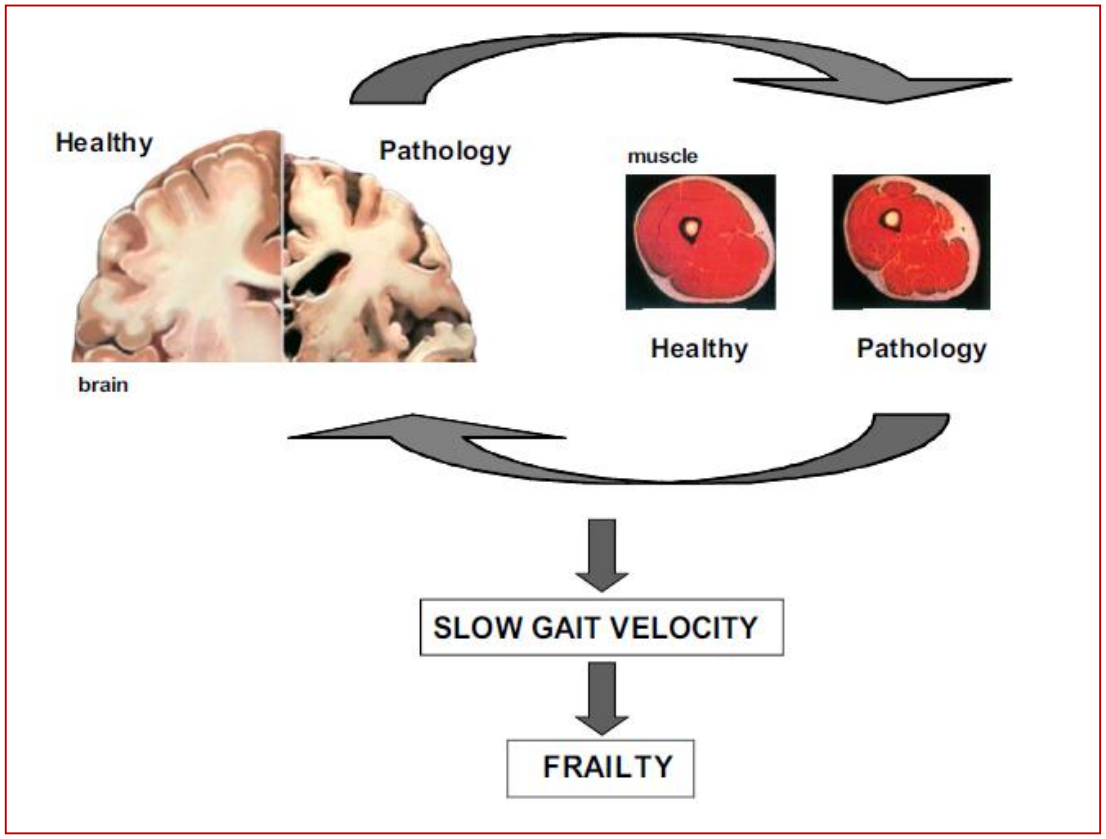


AEP Vol. 16, No. 7
July 2006: 554–562


Invecchiamento
Multimorbilità
Declino funzioni cognitive
+
+
Declino funzioni motorie
Fragilità
Disabilità

"Brain-muscle loop" in the fragility of older persons: from pathophysiology to new organizing models

Fulvio Lauretani[1,3] · Tiziana Meschi[1] · Andrea Ticinesi[1] · Marcello Maggio[2,3]



Aging Clin Exp Res 2017 Dec;29(6):1305-1311

Experimental Gerontology

Review

Measuring gait speed to better identify prodromal dementia

Giulia Grande[a,1], Federico Triolo[a,b,1], Arturo Nuara[b,c], Anna-Karin Welmer[a,d,e,f], Laura Fratiglioni[a,d], Davide L. Vetrano[a,g,*]

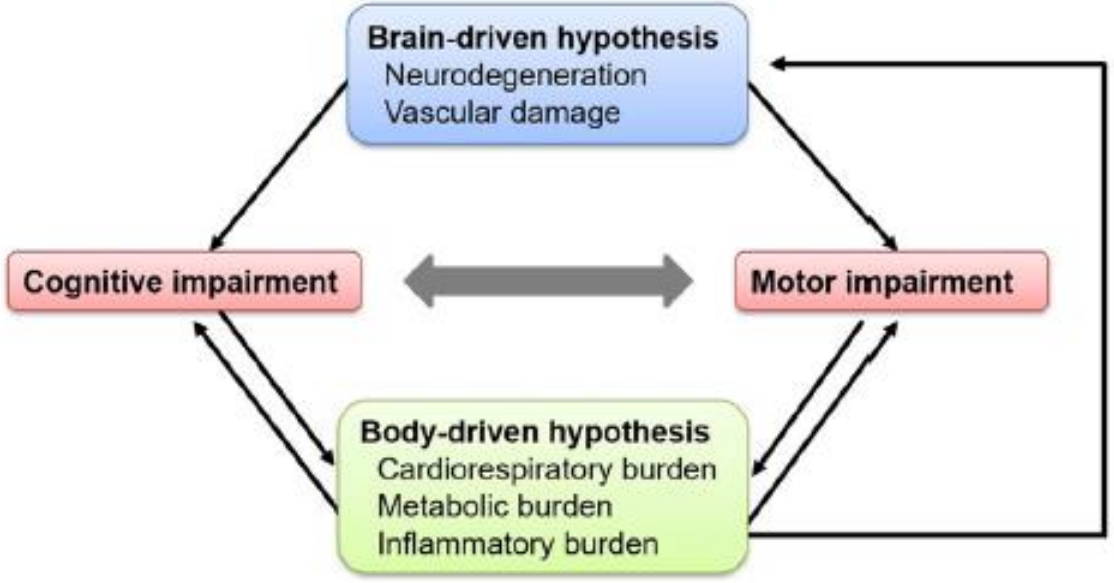
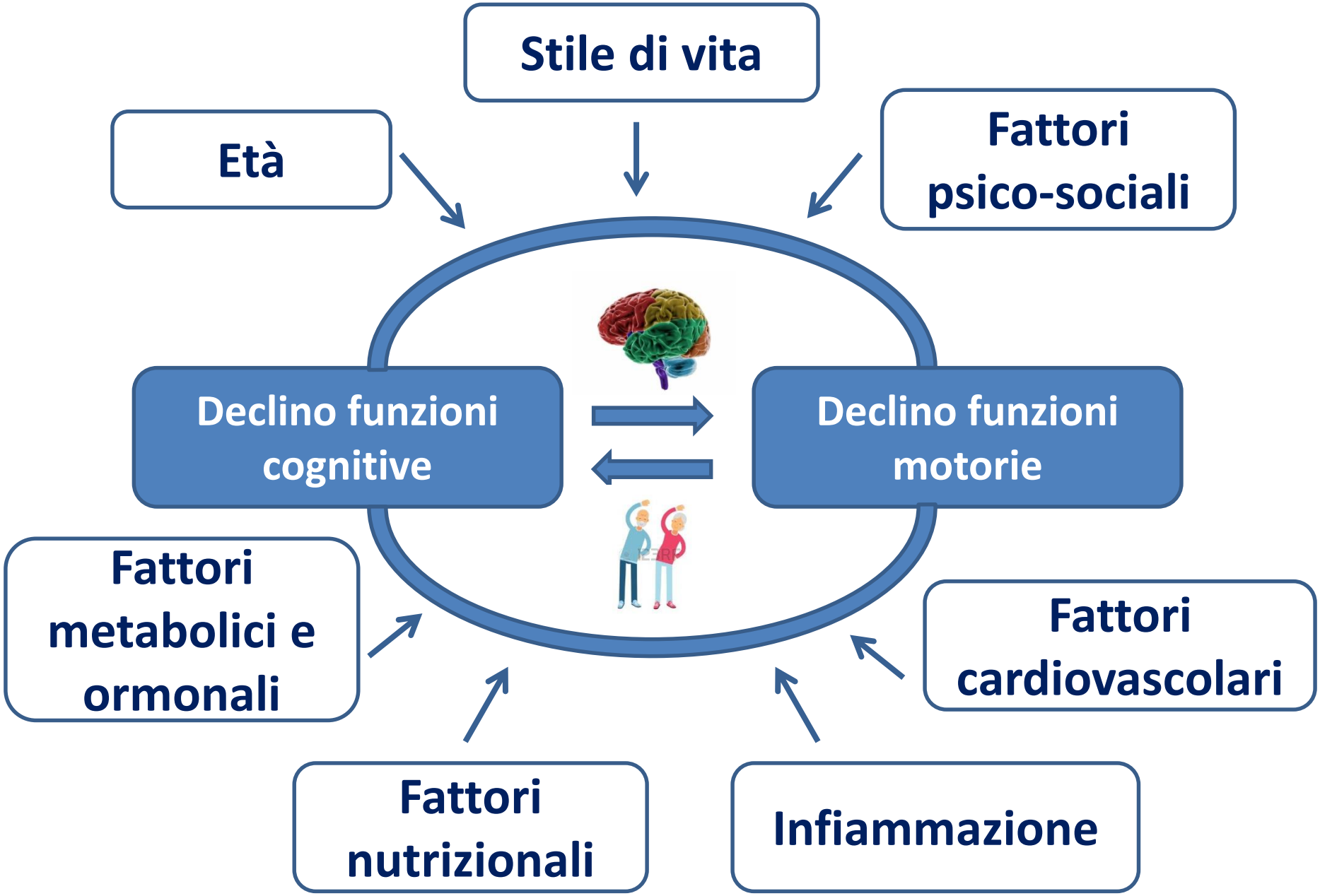


Fig. 4. Determinants and interplay of cognitive and motor impairment.


Stile di vita
Età
Fattori psico-sociali
Declino funzioni cognitive
Declino funzioni motorie
Fattori metabolici e ormonali
Fattori cardiovascolari
Fattori nutrizionali
Infiammazione

frontiers in Neurology

October 2018 | Volume 9 | Article 913

Cognitive Involvement in Balance, Gait and Dual-Tasking in Aging: A Focused Review From a Neuroscience of Aging Perspective

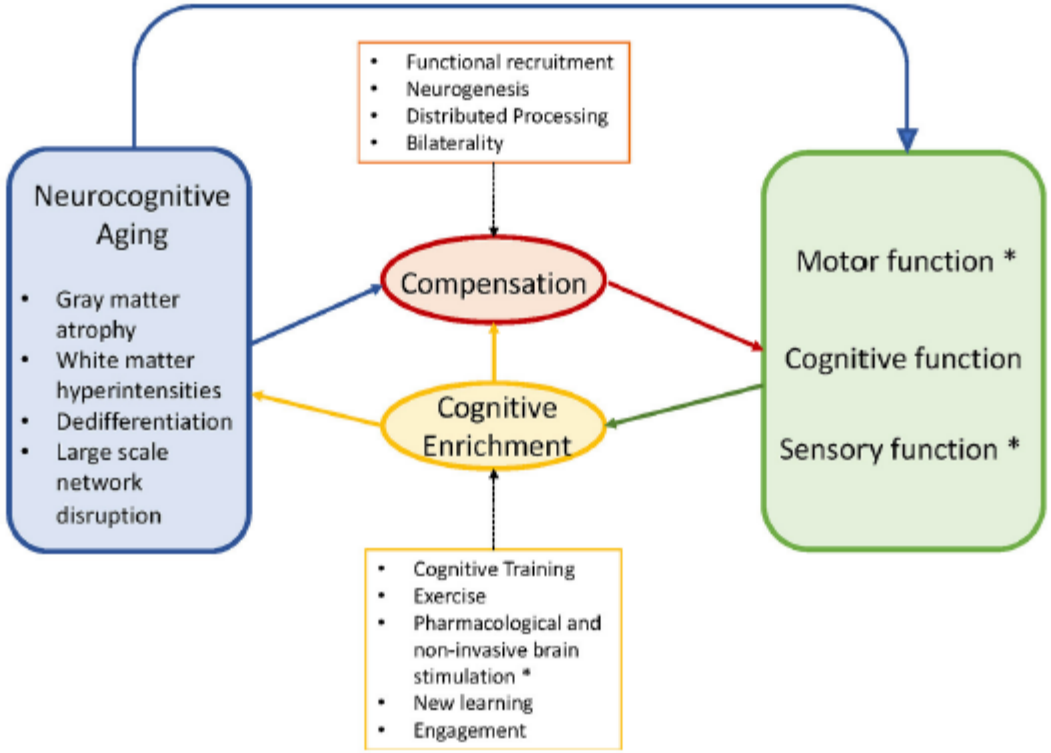


FIGURE 1 | Conceptual diagram adapted from Park and Reuter-Lorenz (11). Asterisks indicate our modifications and extensions, particularly the inclusion of motor and sensory functioning. The schematic shows the negative aspects of neurocognitive aging (blue) that trigger compensation via functional recruitment in older adults (red). These maladaptive and adaptive factors jointly contribute to observed motor, cognitive, sensory functions (green). Cognitive enrichment (yellow) can ameliorate aspects of brain aging and facilitate compensatory efficiency if there is neural overlap between the improved and targeted outcomes. Motor, cognitive, and sensory functions benefit from, and compete for, common capacity (e.g., prefrontal cortex), particularly during complex behaviors such as cognitive-motor dual-tasking. Copyright permission not required.

Journal of Cachexia, Sarcopenia and Muscle 2017; **8**: 557–566
Published online 29 March 2017 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12195

Muscle mass decline, arterial stiffness, white matter hyperintensity, and cognitive impairment: Japan Shimanami Health Promoting Program study

Background There is a close association between frailty and cognitive impairment. However, the underlying contribution of sarcopenia to the development of cognitive impairment is unclear. We investigated the possible association between muscle mass decline and cognitive impairment in a cross-sectional study of 1518 subjects aged 55 years or above. We also evaluated arterial stiffness and white matter hyperintensities (WMHs) as possible underlying mechanisms for this association.

Methods Two sarcopenic indices were measured: thigh muscle cross-sectional area (CSA; calculated by computed tomography) and skeletal muscle mass (bioelectric impedance). Muscle mass decline was defined as either the bottom 10% or 20% of participants for each sex. Cognitive function was assessed using the Touch Panel-type Dementia Assessment Scale, and brachial–ankle pulse wave velocity was measured as an index of arterial stiffness.

Results Both sarcopenic indices were modestly but significantly associated with brachial–ankle pulse wave velocity in male and female subjects. The presence of WMHs was significantly associated with low thigh muscle CSA in men and with low skeletal muscle mass in women. The Touch Panel-type Dementia Assessment Scale score was modestly but significantly and positively associated with thigh muscle CSA in men and skeletal muscle mass in women. Muscle mass decline in the bottom 10% of participants on both sarcopenic indices was significantly and independently related to cognitive impairment in women.

Conclusions Lower sarcopenic indices are significantly related to lower cognitive scores. Arterial stiffness and WMHs could account, at least in part, for this association.

Journal of Cachexia, Sarcopenia and Muscle 2017; **8**: 557–566
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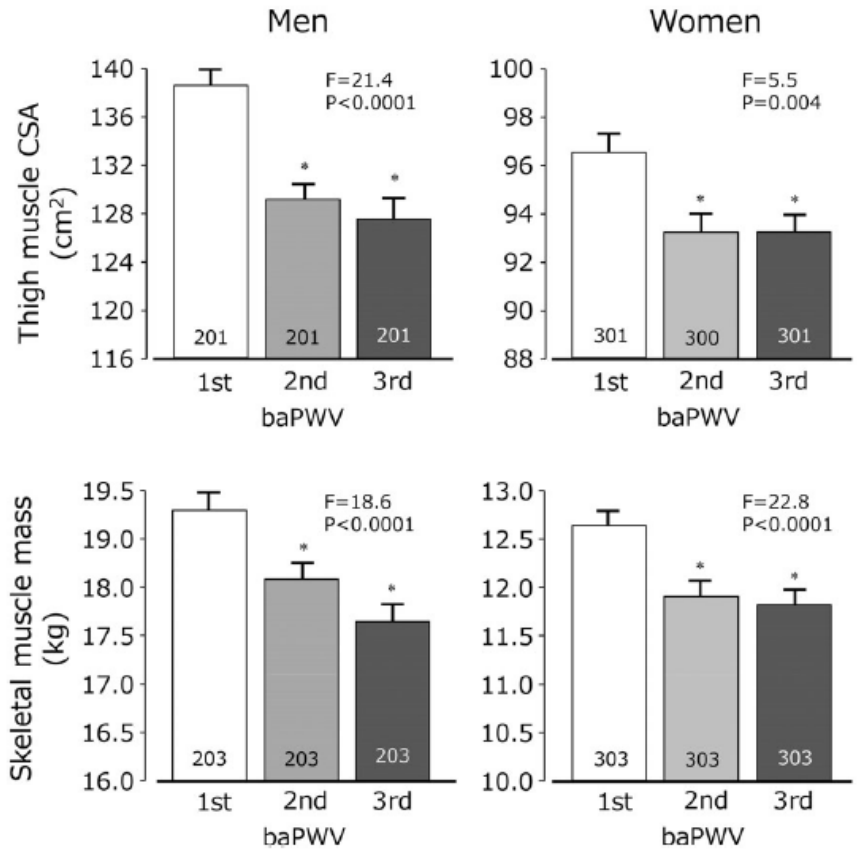
Figure 1 Tertiles of brachial-ankle pulse wave velocity (baWV) and thigh muscle cross sectional area (upper) and skeletal muscle mass [bottom]. For men and women, the lowest tertile of baPWV scores had the highest thigh muscle cross-sectional area (CSA) and skeletal muscle mass. Values are mean ± SEM. *$P < 0.05$ vs. first tertile.



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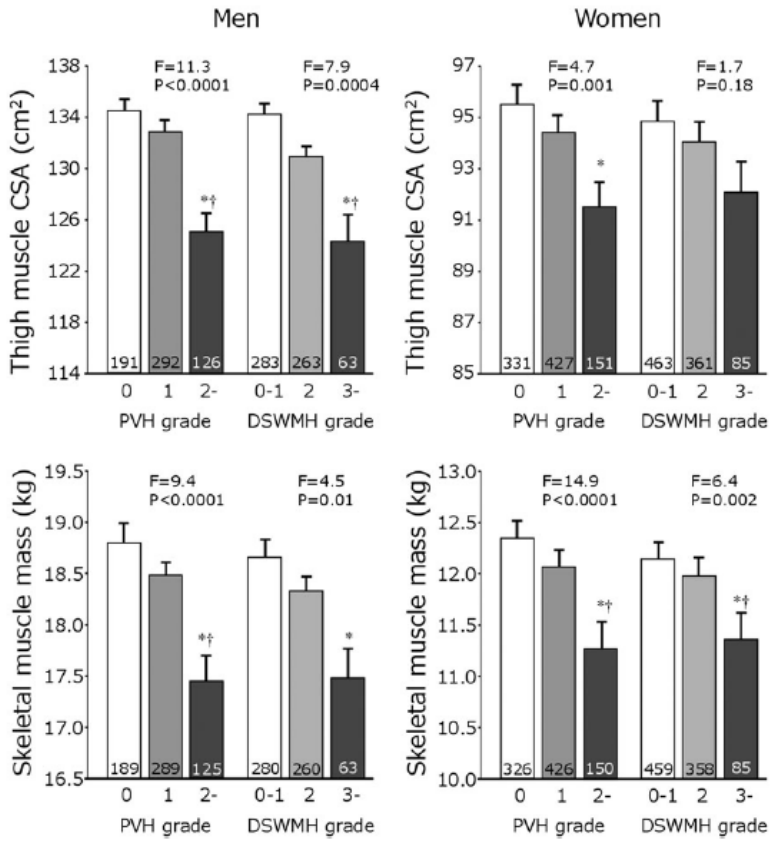
Muscle mass decline, arterial stiffness, white matter hyperintensity, and cognitive impairment: Japan Shimanami Health Promoting Program study

Figure 2 White matter hyperintensity and sarcopenic indices. Two indices for sarcopenia are shown, split by periventricular hyperintensity (PVH) and deep subcortical white matter hyperintensity (DSWMH). The numbers in the columns indicate the number of subjects. Values are mean ± SEM. *$P < 0.05$ vs. first tertile. †$P < 0.05$ vs. second tertile.



Exercise training increases size of hippocampus and improves memory

Kirk I. Erickson[a], Michelle W. Voss[b,c], Ruchika Shaurya Prakash[d], Chandramallika Basak[e], Amanda Szabo[f], Laura Chaddock[b,c], Jennifer S. Kim[b], Susie Heo[b,c], Heloisa Alves[b,c], Siobhan M. White[f], Thomas R. Wojcicki[f], Emily Mailey[f], Victoria J. Vieira[f], Stephen A. Martin[f], Brandt D. Pence[f], Jeffrey A. Woods[f], Edward McAuley[b,f], and Arthur F. Kramer[b,c,1]

The hippocampus shrinks in late adulthood, leading to impaired memory and increased risk for dementia. Hippocampal and medial temporal lobe volumes are larger in higher-fit adults, and physical activity training increases hippocampal perfusion, but the extent to which aerobic exercise training can modify hippocampal volume in late adulthood remains unknown. Here we show, in a randomized controlled trial with 120 older adults, that aerobic exercise training increases the size of the anterior hippocampus, leading to improvements in spatial memory. Exercise training increased hippocampal volume by 2%, effectively reversing age-related loss in volume by 1 to 2 y. We also demonstrate that increased hippocampal volume is associated with greater serum levels of BDNF, a mediator of neurogenesis in the dentate gyrus. Hippocampal volume declined in the control group, but higher preintervention fitness partially attenuated the decline, suggesting that fitness protects against volume loss. Caudate nucleus and thalamus volumes were unaffected by the intervention. These theoretically important findings indicate that aerobic exercise training is effective at reversing hippocampal volume loss in late adulthood, which is accompanied by improved memory function.

Exercise training increases size of hippocampus and improves memory

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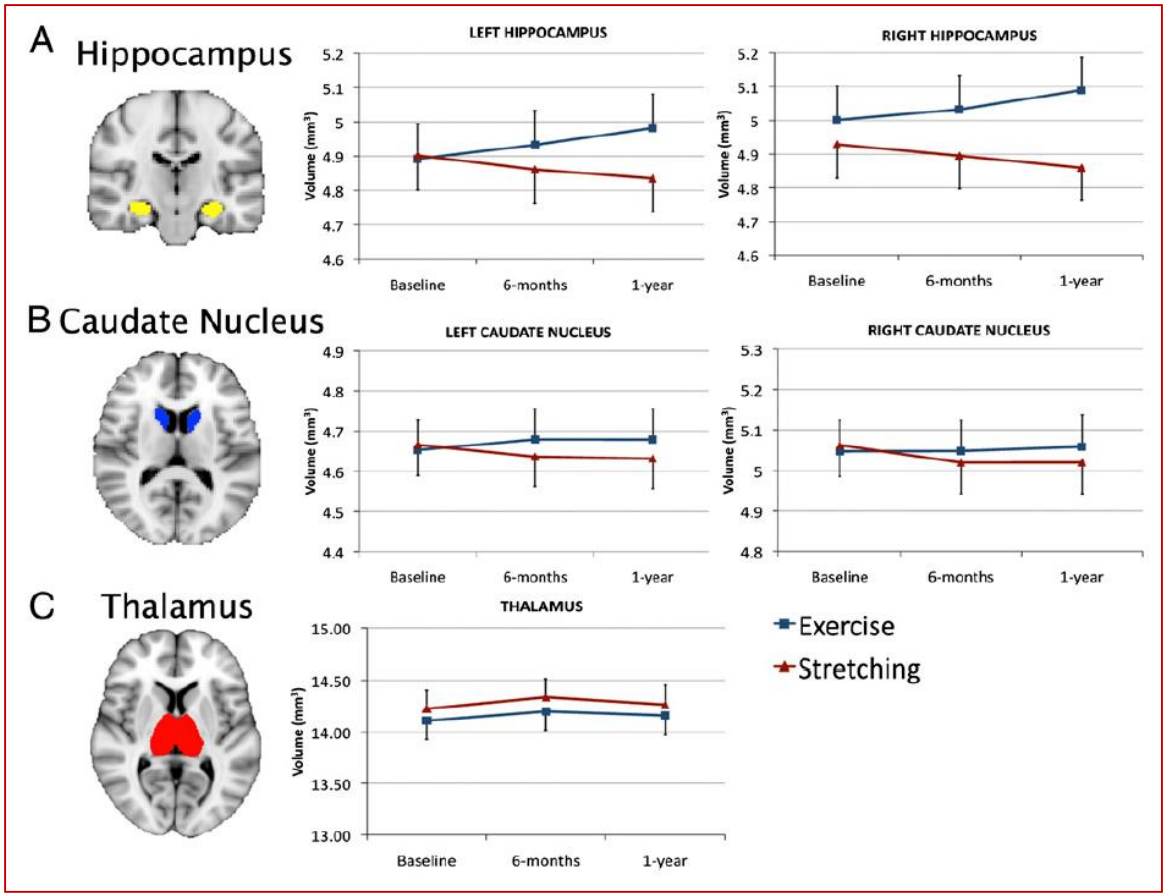
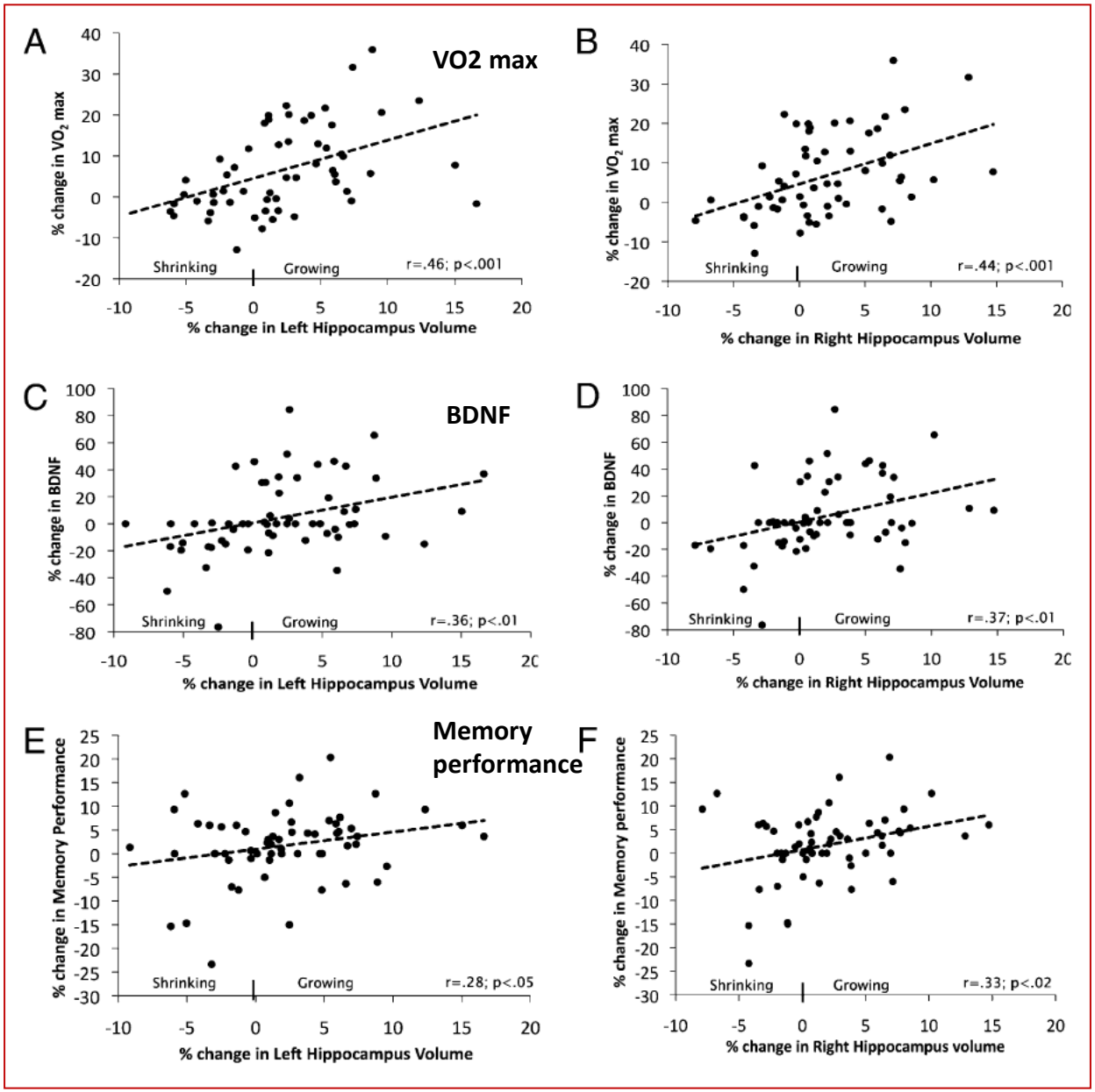



A
VO2 max
% change in VO2 max
% change in Left Hippocampus Volume
Shrinking
Growing
r=.46; p<.001
B
% change in VO2 max
% change in Right Hippocampus Volume
Shrinking
Growing
r=.44; p<.001
C
BDNF
% change in BDNF
% change in Left Hippocampus Volume
Shrinking
Growing
r=.36; p<.01
D
% change in BDNF
% change in Right Hippocampus Volume
Shrinking
Growing
r=.37; p<.01
E
Memory performance
% change in Memory Performance
% change in Left Hippocampus Volume
Shrinking
Growing
r=.28; p<.05
F
% change in Memory performance
% change in Right Hippocampus volume
Shrinking
Growing
r=.33; p<.02

Herold et al. European Review of Aging and Physical Activity (2019) 16:10
https://doi.org/10.1186/s11556-019-0217-2

European Review of
Aging and Physical Activity

REVIEW ARTICLE Open Access

Functional and/or structural brain changes in response to resistance exercises and resistance training lead to cognitive improvements – a systematic review

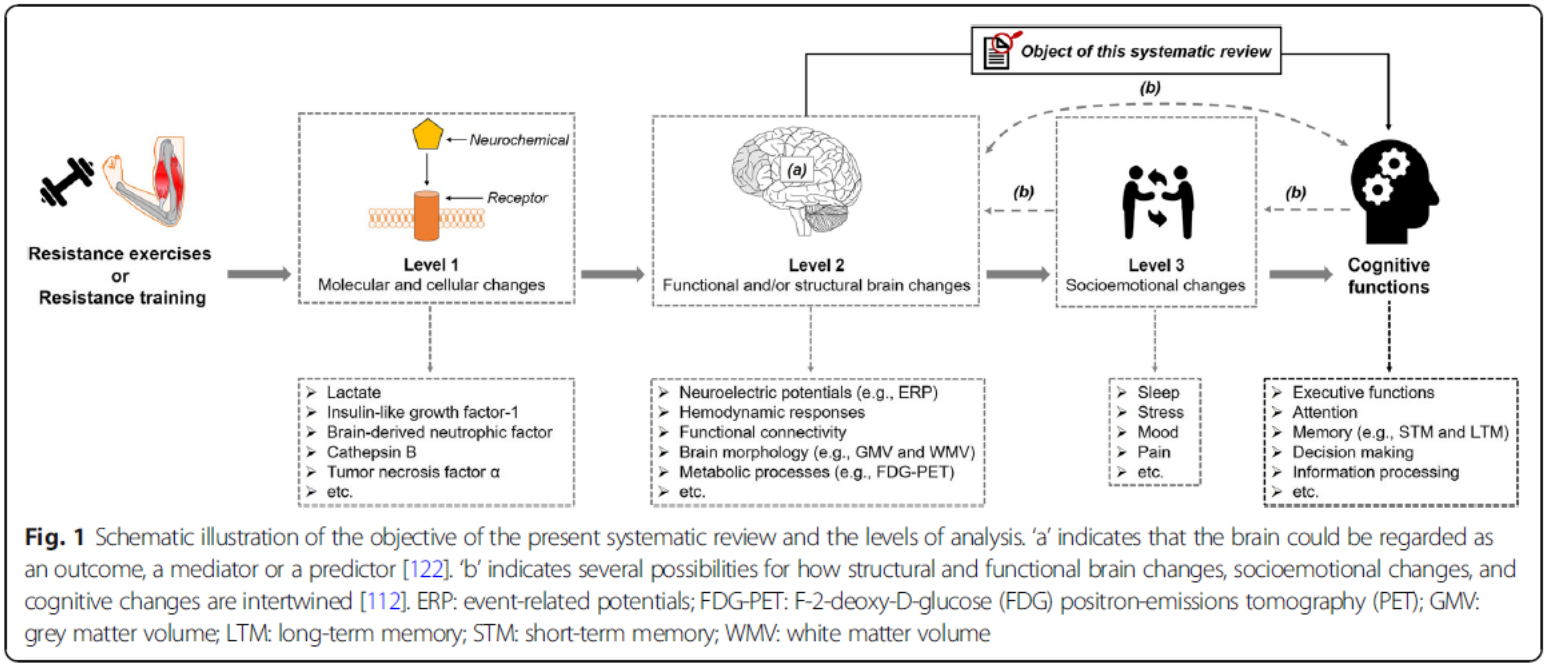
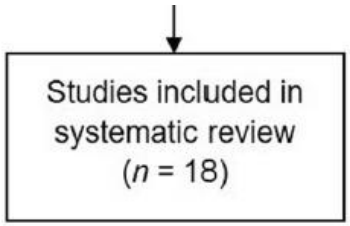


Fig. 1 Schematic illustration of the objective of the present systematic review and the levels of analysis. 'a' indicates that the brain could be regarded as an outcome, a mediator or a predictor [122]. 'b' indicates several possibilities for how structural and functional brain changes, socioemotional changes, and cognitive changes are intertwined [112]. ERP: event-related potentials; FDG-PET: F-2-deoxy-D-glucose (FDG) positron-emissions tomography (PET); GMV: grey matter volume; LTM: long-term memory; STM: short-term memory; WMV: white matter volume



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REVIEW ARTICLE Open Access

Functional and/or structural brain changes in response to resistance exercises and resistance training lead to cognitive improvements – a systematic review

Abstract

Background: During the aging process, physical capabilities (e.g., muscular strength) and cognitive functions (e.g., memory) gradually decrease. Regarding cognitive functions, substantial functional (e.g., compensatory brain activity) and structural changes (e.g., shrinking of the hippocampus) in the brain cause this decline. Notably, growing evidence points towards a relationship between cognition and measures of muscular strength and muscle mass. Based on this emerging evidence, resistance exercises and/or resistance training, which contributes to the preservation and augmentation of muscular strength and muscle mass, may trigger beneficial neurobiological processes and could be crucial for healthy aging that includes preservation of the brain and cognition. Compared with the multitude of studies that have investigated the influence of endurance exercises and/or endurance training on cognitive performance and brain structure, considerably less work has focused on the effects of resistance exercises and/or resistance training. While the available evidence regarding resistance exercise-induced changes in cognitive functions is pooled, the underlying neurobiological processes, such as functional and structural brain changes, have yet to be summarized. Hence, the purpose of this systematic review is to provide an overview of resistance exercise-induced functional and/or structural brain changes that are related to cognitive functions.

Methods and results: A systematic literature search was conducted by two independent researchers across six electronic databases; 5957 records were returned, of which 18 were considered relevant and were analyzed.

Short conclusion: Based on our analyses, resistance exercises and resistance training evoked substantial functional brain changes, especially in the frontal lobe, which were accompanied by improvements in executive functions. Furthermore, resistance training led to lower white matter atrophy and smaller white matter lesion volumes. However, based on the relatively small number of studies available, the findings should be interpreted cautiously. Hence, future studies are required to investigate the underlying neurobiological mechanisms and to verify whether the positive findings can be confirmed and transferred to other needy cohorts, such as older adults with dementia, sarcopenia and/or dynapenia.

Keywords: Cognition, Neuroplasticity, Strength exercises, Strength training, Physical activity

"Brain-muscle loop" in the fragility of older persons: from pathophysiology to new organizing models

Fulvio Lauretani[1,3] · Tiziana Meschi[1] · Andrea Ticinesi[1] · Marcello Maggio[2,3]

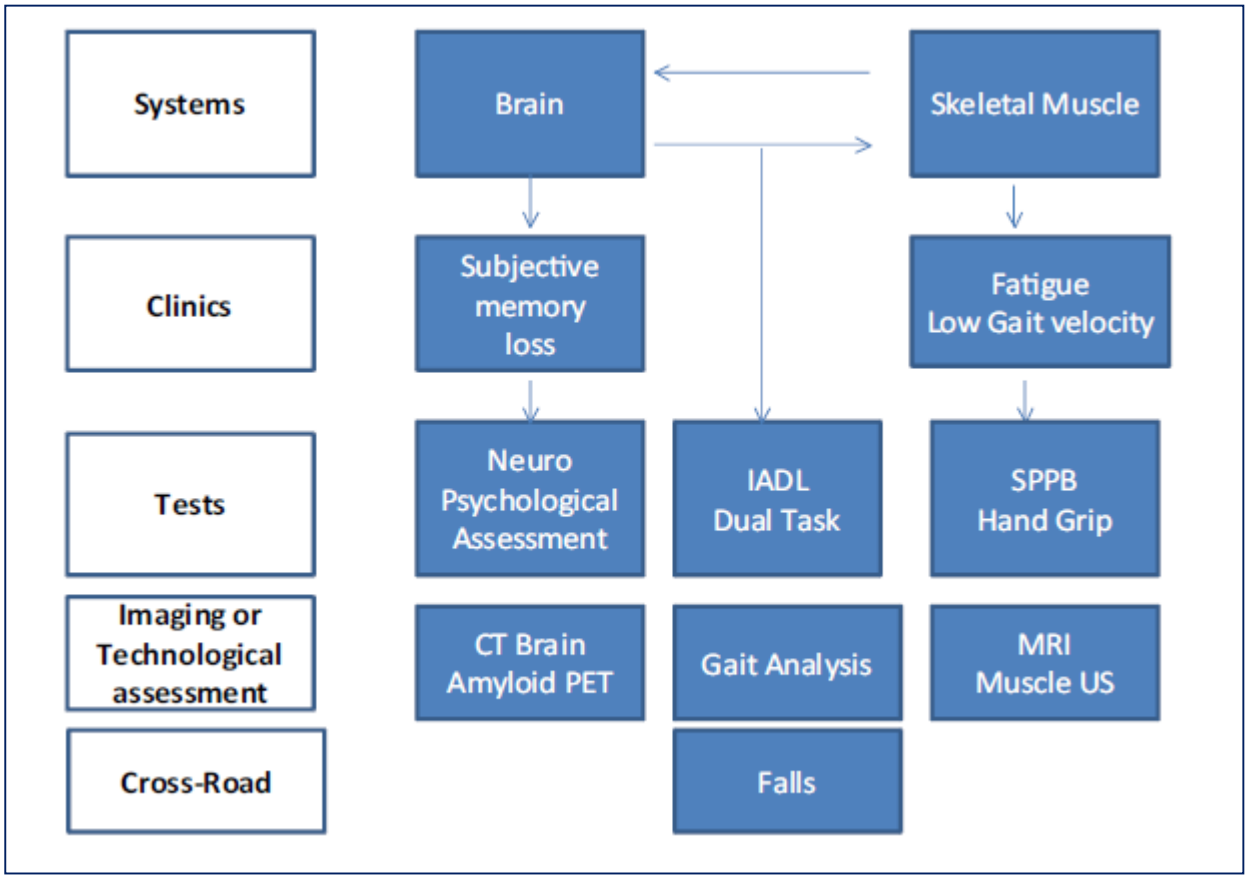


Fig. 2 The rationale of brain muscle loop in the clinical practice

Aging Clin Exp Res 2017 Dec;29(6):1305-1311

COGNITIVE FRAILTY: RATIONAL AND DEFINITION FROM AN (I.A.N.A./I.A.G.G.) INTERNATIONAL CONSENSUS GROUP

Possible biological, clinical, and imaging markers for identifying risk of physical disability and neurodegenerative disease

	Physical disability	Neurodegenerative disease
Biomarkers		
Inflammatory markers (e.g. CRP, IL-6)	+++	+++
Beta-amyloid protein (Aβ)	-	++
APOEε4 genotype	-	++
Anemia	++	++
Serum Albumin	++	++
Cholesterol	++	++
Vitamin D status	++	++
Clinical markers		
MMSE	++	+++
Executive tests	+	+++
ADAS-Cog	-	+++
CDR	++	+++
MoCA	++	+++
Gait speed	+++	++
Hand grip strength	+++	++
Weight loss	+++	++
Psychological marker: GDS	++	+++
Actigraphy	+++	+++
Imaging		
Dual energy X-ray absorptiometry scans (DEXA)	++	++
Cerebral Computed tomography (CT scans)	+	+++
Cerebral Magnetic resonance imaging (MRI)	+	+++
Functional magnetic resonance imaging (FMRI)	+	+++
Diffusion tensor imaging (DTI)	+	+++
Tractography	+	+++
Electrophysiological methods	+	+++
Cognitive evoked potentials	+	+++

The Journal of Nutrition, Health & Aging©
Volume 17, Number 9, 2013

Functional Decline in Cognitive Impairment – The Relationship between Physical and Cognitive Function

Table 1. Comparison of baseline characteristics between men and women

	Men (n = 2,000)	Women (n = 2,000)	p value
Age, years	72.3 ± 5.0	72.5 ± 5.3	0.26
Body weight, kg	62.4 ± 9.3	54.5 ± 8.4	<0.001
Body mass index	23.4 ± 3.1	23.9 ± 3.4	<0.001
Ex- or current smokers, %	63.8	9.5	<0.001
Years of education, %			<0.001
0 years	5.2	37.7	
1–6 years	55.2	45.2	
7–12 years	26.1	11.3	
>12 years	13.6	6.0	
PASE score	97.2 ± 50.2	85.3 ± 33.1	<0.001
CSI-D cognitive score ≥28.40, %	4.9	25.3	<0.001
MMSE score	26.9 ± 2.7	24.2 ± 3.9	<0.001
Whole-body muscle mass, kg	44.2 ± 5.4	33.83 ± 4.1	<0.001
ASM, kg	19.1 ± 2.6	13.81 ± 1.9	<0.001
Arthritis, %	24.3	21.8	0.067
Diabetes mellitus, %	14.7	14.3	0.753
Stroke, %	5.5	3.3	0.001
Heart disease, %	18.3	16.5	0.102

Values are expressed as means ± standard deviation or percentages. MMSE = Mini-Mental State Examination. Statistical comparison was made by unpaired Student t test for continuous variables and χ^2 test for categorical variables.

Table 2. Comparison between the cognitively impaired and non-impaired groups

	Impaired group	Nonimpaired group	p value
Age, years			
Men	76.43 ± 0.59	72.18 ± 0.11	<0.001
Women	74.64 ± 0.26	71.87 ± 0.13	<0.001
Whole-body muscle mass, kg			
Men	42.14 ± 0.56	44.34 ± 0.12	<0.001
Women	33.69 ± 0.19	33.88 ± 0.12	0.350
ASM, kg			
Men	18.12 ± 0.27	19.23 ± 0.06	<0.001
Women	13.73 ± 0.08	13.84 ± 0.05	0.264
Grip strength, kg			
Men	26.36 ± 0.71	31.46 ± 0.14	<0.001
Women	19.44 ± 0.19	20.52 ± 0.11	<0.001
Six-meter walk speed, m/s			
Men	0.89 ± 0.024	1.02 ± 0.004	<0.001
Women	0.85 ± 0.009	0.93 ± 0.005	<0.001
Chair stand test, s			
Men	13.99 ± 0.45	12.57 ± 0.088	<0.001
Women	14.45 ± 0.27	13.07 ± 0.12	<0.001
PASE score			
Men	84.2 ± 5.60	97.9 ± 1.14	0.008
Women	79.4 ± 1.39	87.3 ± 0.86	<0.001

Values are expressed as means ± standard error. Statistical comparison was made by unpaired Student t test.

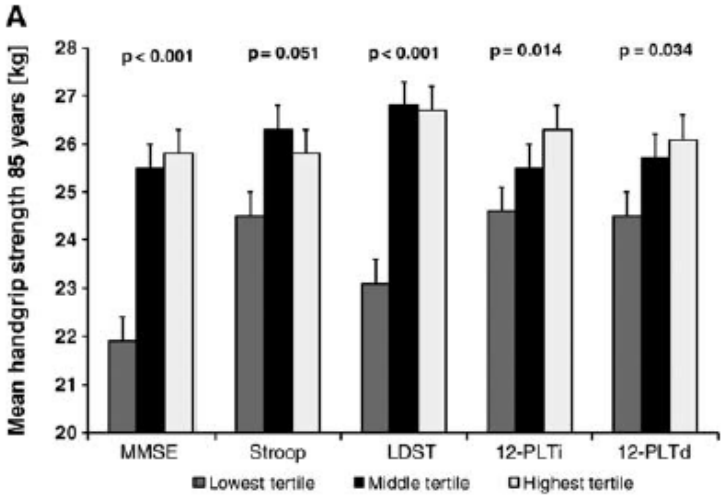
Neuroepidemiology 2008;31:167–173

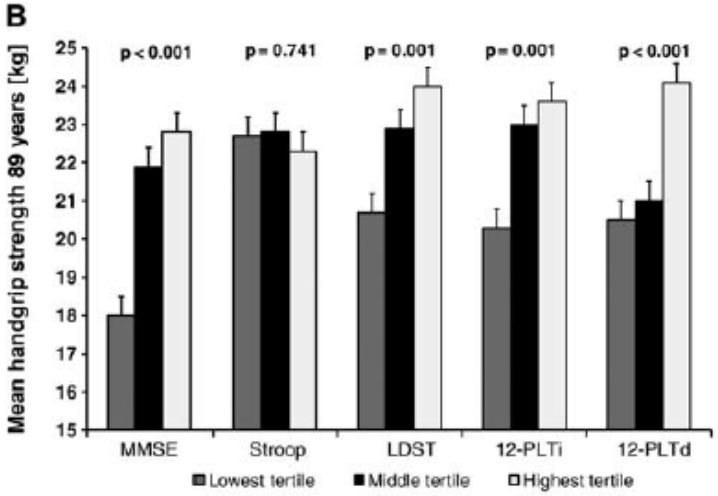
Temporal relationship between handgrip strength and cognitive performance in oldest old people

Background: cognitive decline and muscle weakness are prevalent health conditions in elderly people. We hypothesised that cognitive decline precedes muscle weakness.
Objective: to analyse the temporal relationship between cognitive performance and handgrip strength in oldest old people.
Design: prospective population-based 4-year follow-up study.
Subjects: a total of 555 subjects, all aged 85 years at baseline, were included into the study.
Methods: handgrip strength measured at age 85 and 89 years. Neuropsychological test battery to assess global cognitive performance, attention, processing speed and memory at baseline and repeated at age 89 years. Associations between handgrip strength and cognitive performance were analysed by repeated linear regression analysis adjusted for common confounders.
Results: at age 85 and 89 years, better cognitive performance was associated with higher handgrip strength (all, $P < 0.03$), except for attention. There was no longitudinal association between baseline handgrip strength and cognitive decline (all, $P > 0.10$), except for global cognitive performance ($P = 0.007$). Better cognitive performance at age 85 years was associated with slower decline in handgrip strength (all, $P < 0.01$) after adjustment for common confounders.
Conclusion: baseline cognitive performance was associated with decline in handgrip strength, whereas baseline handgrip strength was not associated with cognitive decline. Our results suggest that cognitive decline precedes the onset of muscle weakness in oldest old people.

Age and Ageing 2012; **41:** 506–512

Temporal relationship between handgrip strength and cognitive performance in oldest old people





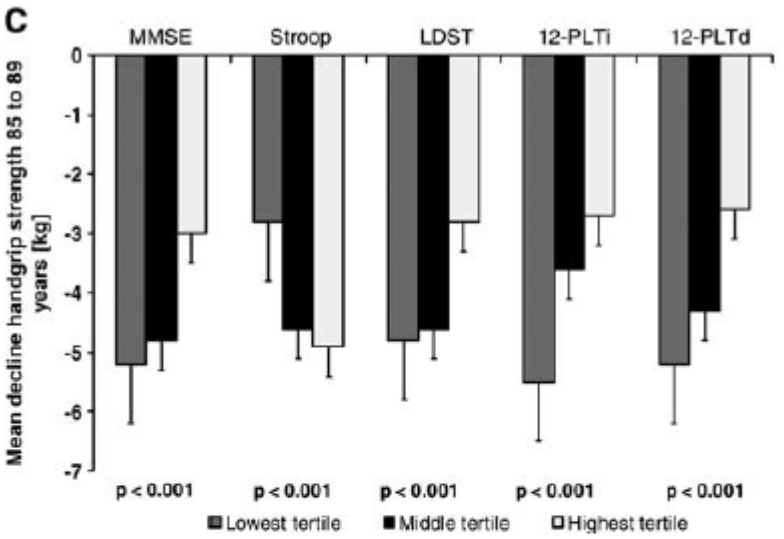


Figure 1. Mean handgrip strength at (*A*) age 85 and (*B*) 89 years in tertiles of baseline cognitive performance and (*C*) decline in handgrip strength from age 85 to 89 years in tertiles of baseline cognitive performance (MMSE, Stroop, Letter Digit Substitution Task (LDST) and 12-Picture Learning Test (12-PLT immediate (i) and delayed recall (d)). Error bars indicate standard error (SE). *P* value indicates *p* for trend of mean handgrip strength in tertiles of test scores (linear regression analysis adjusted for sex, height and weight). Take note that lower Stroop scores indicate better performance.

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Review

Handgrip strength as a means of monitoring progression of cognitive decline – A scoping review

Nora E. Fritz [a,b], Caitlin J. McCarthy [a], Diane E. Adamo [a,c,*]

[a] Physical Therapy Program, Eugene Applebaum College of Pharmacy and Health Sciences, Wayne State University, Detroit, MI, United States
[b] Department of Neurology, Wayne State University, Detroit, MI, United States
[c] Institute of Gerontology, Wayne State University, Detroit, MI, United States

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ABSTRACT

Cognitive decline in older adults contributes to reduced ability to perform daily tasks and continued disuse leads to muscle weakness and potentiates functional loss. Despite explicit links between the motor and cognitive systems, few health care providers assess motor function when addressing the needs of individuals with cognitive loss. Early and easy measurable biomarkers of cognitive decline have the potential to improve care for individuals with dementia and mild cognitive impairment. The aim of this study was to conduct a systematic search to determine the relationship among handgrip strength, as a measure of global muscle strength, and cognitive decline over time. Fifteen prospective, cohort, longitudinal studies of adults >60 years old who were healthy or at risk of cognitive decline at study onset were included in the review. Studies that investigated changes in cognition relative to baseline grip strength and, those that investigated changes in grip strength relative to cognitive function were revealed. Findings here support the use of handgrip strength as a way to monitor cognitive changes and show that reduced handgrip strength over time may serve as a predictor of cognitive loss with advancing age.

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The Trajectory of Gait Speed Preceding Mild Cognitive Impairment

Teresa Buracchio, MD; Hiroko H. Dodge, PhD; Diane Howieson, PhD; Dara Wasserman, BS; Jeffrey Kaye, MD

Objectives: To compare the trajectory of motor decline, as measured by gait speed and finger-tapping speed, between elderly people who developed mild cognitive impairment (MCI) and those who remained cognitively intact. We also sought to determine the approximate time at which the decline in motor function accelerated in persons who developed MCI.

Design: Longitudinal cohort study.

Participants: Participants were 204 healthy seniors (57.8% women) from the Oregon Brain Aging Study evaluated for up to 20 years using annual neurologic, neuropsychological, and motor examinations.

Main Outcome Measures: The pattern of motor decline with aging was compared using a mixed-effects model with an interaction term for age and a clinical diagnosis of MCI. The time before diagnosis of MCI, when the change in gait or finger-tapping speed accelerates, was assessed using a mixed-effects model with a change point for men and women, separately and combined, who developed MCI.

Results: The rates of change, with aging, in gait speed ($P<.001$) and finger-tapping speed in the dominant hand ($P=.003$) and nondominant hand ($P<.001$) were significantly different between participants who developed MCI (converters) and those who did not (nonconverters). Using a change point analysis for MCI converters, the decrease in gait speed accelerated by 0.023 m/s/y ($P<.001$), occurring 12.1 years before the onset of MCI. An acceleration in gait speed decline occurred earlier in men than women. For tapping speed, the change point occurred after the onset of MCI for both dominant and nondominant hands when men and women were combined.

Conclusions: Motor decline as indexed by gait speed accelerates up to 12 years before MCI. Longitudinal changes in motor function may be useful in the early detection of dementia during preclinical stages, when the utility of disease-modifying therapies would be greatest.

Arch Neurol. 2010;67(8):980-986

The Trajectory of Gait Speed Preceding Mild Cognitive Impairment

Teresa Buracchio, MD; Hiroko H. Dodge, PhD; Diane Howieson, PhD; Dara Wasserman, BS; Jeffrey Kaye, MD

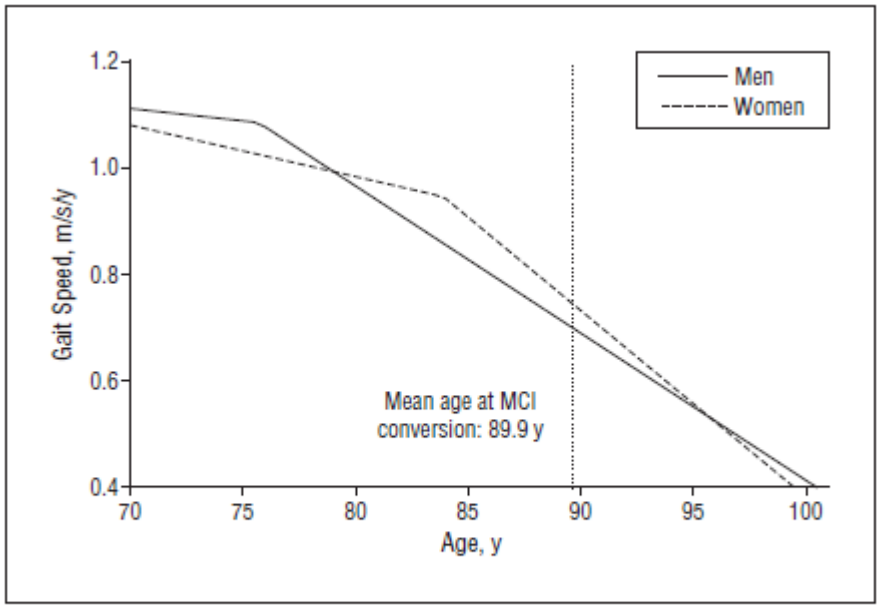


Figure 1. Change point example for gait speed in mild cognitive impairment (MCI) converters in relation to the mean age at conversion in men and women.

Arch Neurol. 2010;67(8):980-986

Experimental Gerontology

Review

Measuring gait speed to better identify prodromal dementia

Giulia Grande[a,1], Federico Triolo[a,b,1], Arturo Nuara[b,c], Anna-Karin Welmer[a,d,e,f], Laura Fratiglioni[a,d], Davide L. Vetrano[a,g,*]

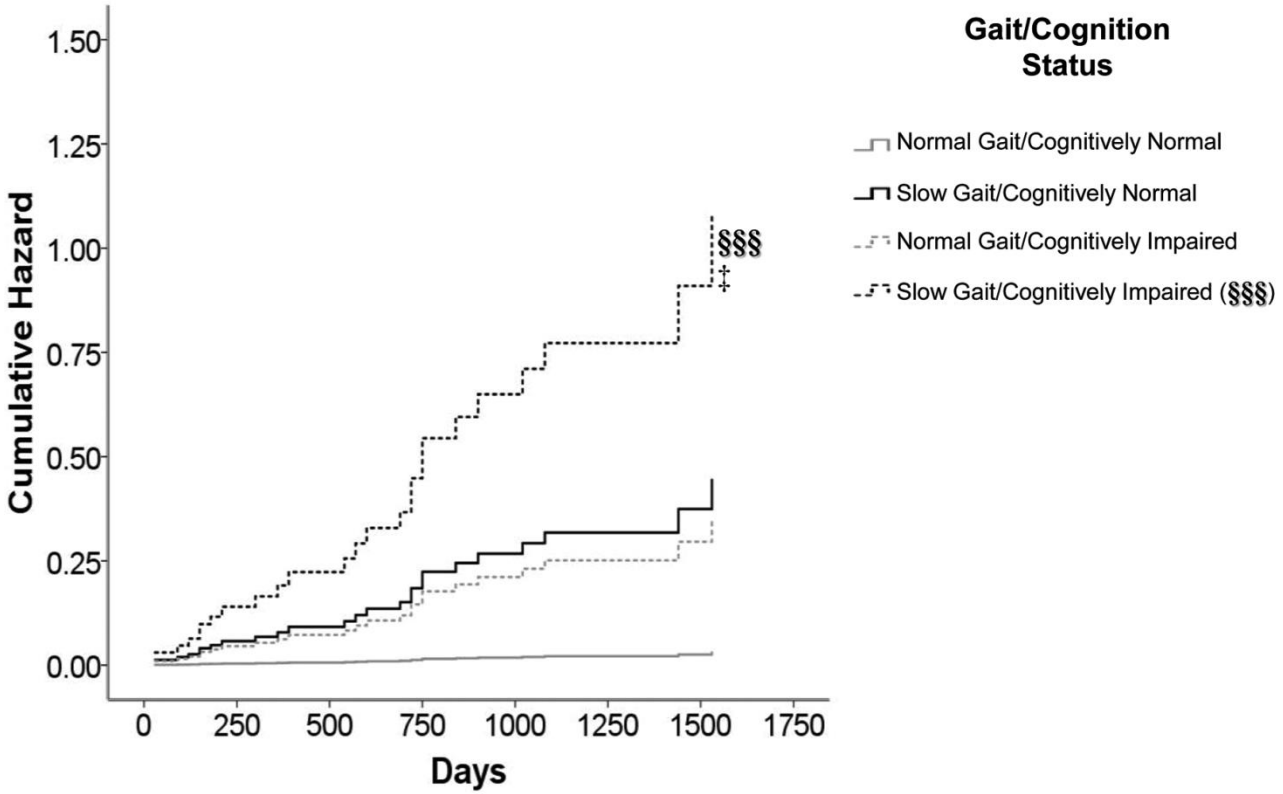
Main characteristics and findings of longitudinal studies investigating the prognostic role of gait speed for cognitive outcomes. Grey bands identify studies investigating the role of the interplay between baseline slow gait speed and cognitive impairment.

● = significant association; ● = not associated or non-significant association.

Study	N	Mean age years; (% female sex)	Outcomes *				
			Cognitive decline[a]	Cognitive impairment[b]	Dementia		
					All-cause	AD	Vascular/non-AD dementia
Atkinson et al, 2010	1793	70; (100)	●				
Auyeung et al, 2011	2737	72; (45)	●				
Best et al, 2016	2876	74; (52)	●				
Boyle et al, 2010	761	79; (76)		●			
Bullain et al, 2016	578	93; (70)			●		
Byun et al, 2018	91	67; (44)		●			
Camargo et al, 2016	2176	62; (54)			●	●	
Demnitz et al, 2017	387	70; (19)		●			
Deshpande et al; 2009	660	75; (54)	●	●			
Doi et al; 2018	3937	74; (53)			●		
Doi et al; 2019	4011	72; (54)			●		
Donoghue et al; 2018	1740	72; (52)	●				
Dumurgier et al, 2017	3663	73; (62)			●	●	●
Gale et al, 2014	2654	69; (56)	●				
Gillain et al, 2016	13	70-74; (50)				●	
Gray et al, 2013	2619	77; (60)			●	●	●
Hackett et al; 2018	3932	60+; (55)			●		
Hooghiemstra et al; 2017	309	70; (35)	●	●	●		
Hsu et al; 2017	249	86; (0)		●			
Inzitari et al; 2007	2776	73; (53)		●			
Krall et al; 2014	395	74; (100)	●				
Kuate-Tegueu et al, 2017	1265	74; (60)			●	●	●
Lipnicki et al, 2017	873	79; (56)		●	●		
MacDonald et al, 2017	121	85; (64)	●				
Marquis et al, 2002	108	83; (63)		●			
Mielke et al, 2013	1478	80; (51)	●				
Montero-Odasso et al; 2016	252	77; (63)			●		
Montero-Odasso et al; 2017	112	76; (49)			●		
Montero-Odasso et al; 2018	154	74; (54)			●		
Park et al, 2017	620	60+; (51)		●			
Savica et al; 2017	3426	74; (50)	●				
Sibbett et al; 2018	488	79; (57)			●		
Taniguchi et al; 2012	853	75; (59)		●			
Taylor et al; 2017	134	82; (59)	●				
Tian et al; 2017	412	71; (51)	●				
Verghese et al; 2013	**767**	70+; (61)			●		●
Verghese et al; 2014	4555	60-108; (-)		●	●		
Veronese et al; 2016	1249	72; (59)	●	●			
Welmer et al; 2014	2232	72; (63)			●		

Disentangling Cognitive-Frailty: Results From the Gait and Brain Study

Cumulative hazard ratio for progression to dementia stratified by



J Gerontol A Biol Sci Med Sci, 2016, Vol. 71, No. 11, 1476–1482

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doi:10.1093/gerona/glx003
Advance Access publication February 8, 2017

OXFORD

Research Article

Gait Speed and Grip Strength Reflect Cognitive Impairment and Are Modestly Related to Incident Cognitive Decline in Memory Clinic Patients With Subjective Cognitive Decline and Mild Cognitive Impairment: Findings From the 4C Study

Background: Prospective studies in the general population show that slow gait speed is associated with cognitive decline and clinical progression to dementia. However, longitudinal studies in memory clinic populations are mostly lacking. We aimed to study the association between gait speed and grip strength and cognitive functioning at baseline and cognitive decline over time in memory clinic patients with subjective cognitive decline and mild cognitive impairment.

Methods: We included 309 patients (age 70 ± 9 years, 108 [35%] women, Mini-Mental State Examination 27 ± 3 points). Baseline gait speed was assessed over 15 feet, grip strength with a hydraulic hand dynamometer. Cognitive functioning was assessed annually with a comprehensive test battery during 3 years.

Results: Age- and gender-adjusted linear mixed models showed that slower gait speed was related to worse baseline attention, memory, information processing speed, and verbal fluency. Longitudinally, gait speed was related to decline in information processing speed and executive functioning. Weaker grip strength was related to worse baseline information processing speed and executive functioning but there were no longitudinal associations. Cox proportional hazards models revealed no significant associations with clinical progression.

Conclusions: Our findings suggest that markers of physical performance are related to current cognitive status and modestly related to cognitive decline but are seemingly not useful as an early marker of incident clinical progression.

Table 5. Associations Between Gait Speed and Grip Strength and Clinical Progression

	Gait Speed		Grip Strength	
Models	HR (95% CI)		HR (95% CI)	
Model 1[a]	1.18 (0.84–1.65)		1.07 (0.77–1.48)	
Model 2	1.23 (0.86–1.77)		1.05 (0.76–1.46)	
	SCD (n = 141)	MCI (n = 168)	SCD (n = 141)	MCI (n = 168)
Model 1[a]	0.97 (0.64–1.45)	1.62 (0.93–2.81)	1.06 (0.60–1.90)	1.11 (0.74–1.67)
Model 2	1.01 (0.65–1.56)	1.80 (0.97–3.32)	1.13 (0.63–2.03)	1.06 (0.70–1.60)
	≤65 y (n = 112)	>65 y (n = 197)	≤65 y (n = 112)	>65 y (n = 197)
Model 1[b]	0.81 (0.46–1.44)	1.49 (1.00–2.23)	1.08 (0.60–1.92)	1.07 (0.73–1.57)
Model 2	0.85 (0.48–1.52)	1.84 (1.14–2.99)	1.05 (0.58–1.87)	1.09 (0.75–1.60)

Notes: HR = hazard ratio; MCI = mild cognitive impairment; SCD = subjective cognitive decline. Gait speed and grip strength were standardized with the SCD patients as reference group. Cox proportional hazard models. Analyses were stratified for baseline diagnosis (SCD/MCI) and for age (≤ 65/> 65 y).

[a]Model 1: HR adjusted for age, gender, education, and center. [b]Model 1: HR adjusted for gender, education, and center. Model 2: Additional adjustment for CIRS-G cardiac and vascular sub scores.

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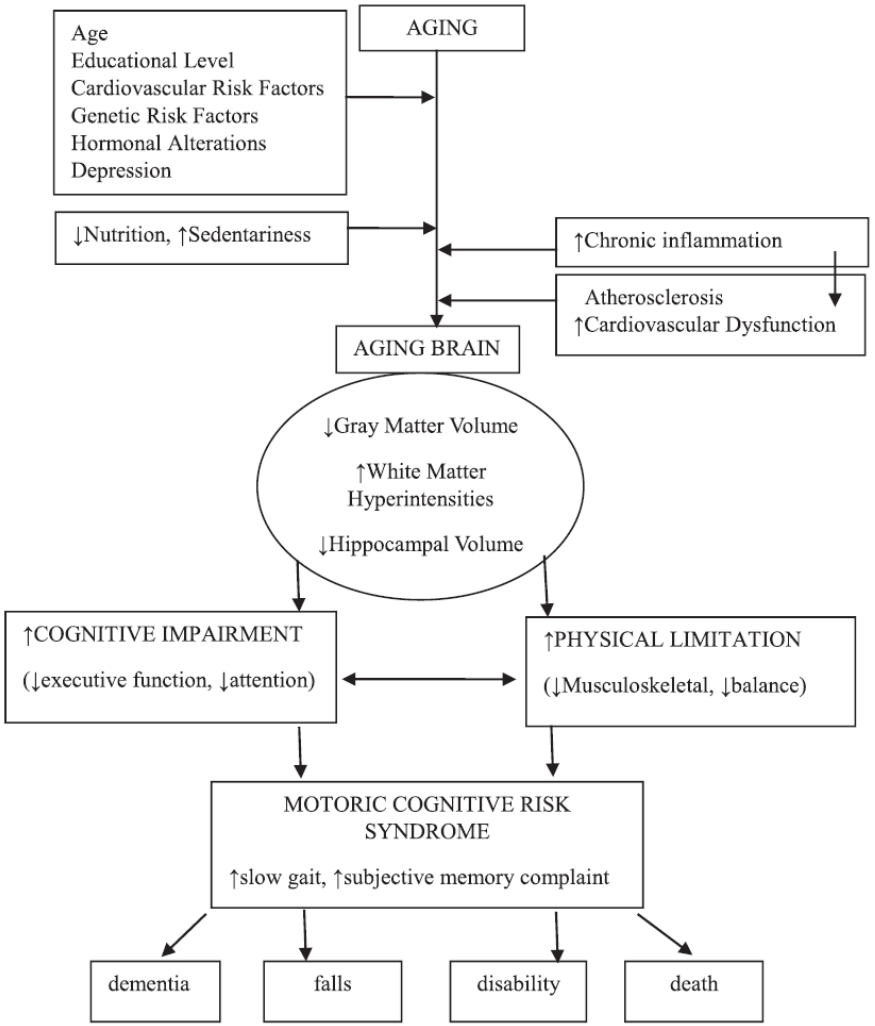
Motoric Cognitive Risk Syndrome: Predictor of Dementia and Age-Related Negative Outcomes

Jagadish K. Chhetri[1], Piu Chan[2], Bruno Vellas[1,3] and Matteo Cesari[1,3]*



Prevalence, incidence, and clinical impact of cognitive–motoric risk syndrome in Europe, USA, and Japan: facts and numbers update 2019

Marcello Maggio[1,2]* and Fulvio Lauretani[1,2]

Table 1 Prevalence, incidence, and clinical impact of the motoric–cognitive risk syndrome (MCRS) in Europe, USA, and Japan

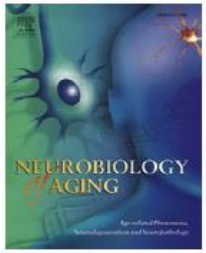
Country or/and name of the study	Prevalence of the MCRS	Incidence of the MCRS	Incident dementia	Mortality/disability risk
Europe, SHARE study/InCHIANTI study	8.0%, 319/3977	65.2 per 1000 person years in adults aged >60 years	2.74 [1.54–4.86], 0.001	1.50 [1.04–2.16], $P = 0.030$
USA, HRS study/EAS study	7.0%, 142/2025		3.27 [1.55–6.90], $P < 0.001$	1.87 [1.54–2.28], $P < 0.001$
Japan, NCGGSGS study	6.3%, 265/4235		2.49 [1.52–4.10], $P < 0.001$	1.69 [1.08–2.02], $P < 0.001$

EAS, Einstein Aging Study; HRS, Health and Retirement Study; NCGGSGS study, National Center for Geriatrics and Gerontology Study of Geriatric Syndrome; SHARE study: Survey of Health, Ageing and Retirement in Europe.

Table 2 Proposed 'Minimum-battery' of tests to identify older persons with cognitive–motoric risk syndrome

Mobility tests	Cognitive tests
Gait speed (usual pace)	MoCA
Dual task (speed)	Trail Making Test A and B

MoCA, Montreal Cognitive Assessment.



Review

Get a grip: individual variations in grip strength are a marker of brain health

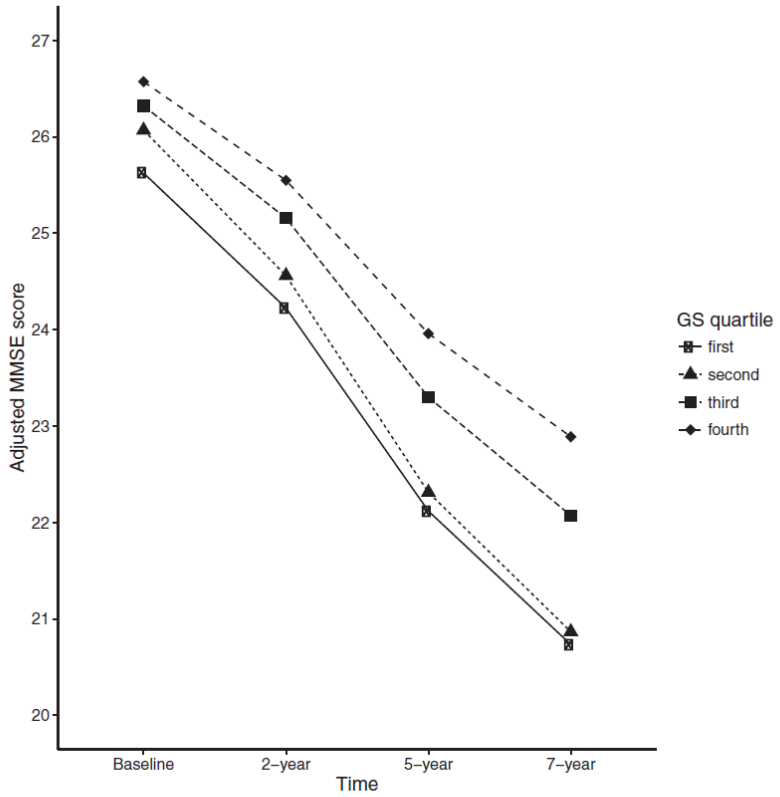


Fig. 10. Means of the distributions of MMSE scores obtained over a 7-year period, partitioned by handgrip strength quartiles at baseline in 2160 participants (1242 females; 918 males, with MMSE >21 at baseline and complete data on all covariates). The MMSE values are adjusted for age, gender, education, marital status, medical conditions, depression, near and distant vision impairment, and body mass index. This original plot by the author was generated using data extracted from Alfaro-Acha et al. (2006). It shows that maximum grip strength measured at the beginning of the assessment period predicts subsequent decreases in a multiple domain test of cognitive function. The extent of the decline is greatest for the group with the lowest grip strength on initial observation and smallest for the group with the highest grip strength on initial observation. Abbreviations: GS, grip strength; MMSE, mini mental state examination.

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OXFORD

Research Article

Association of Sarcopenia With Performance on Multiple Cognitive Domains: Results From the ELSA-Brasil Study

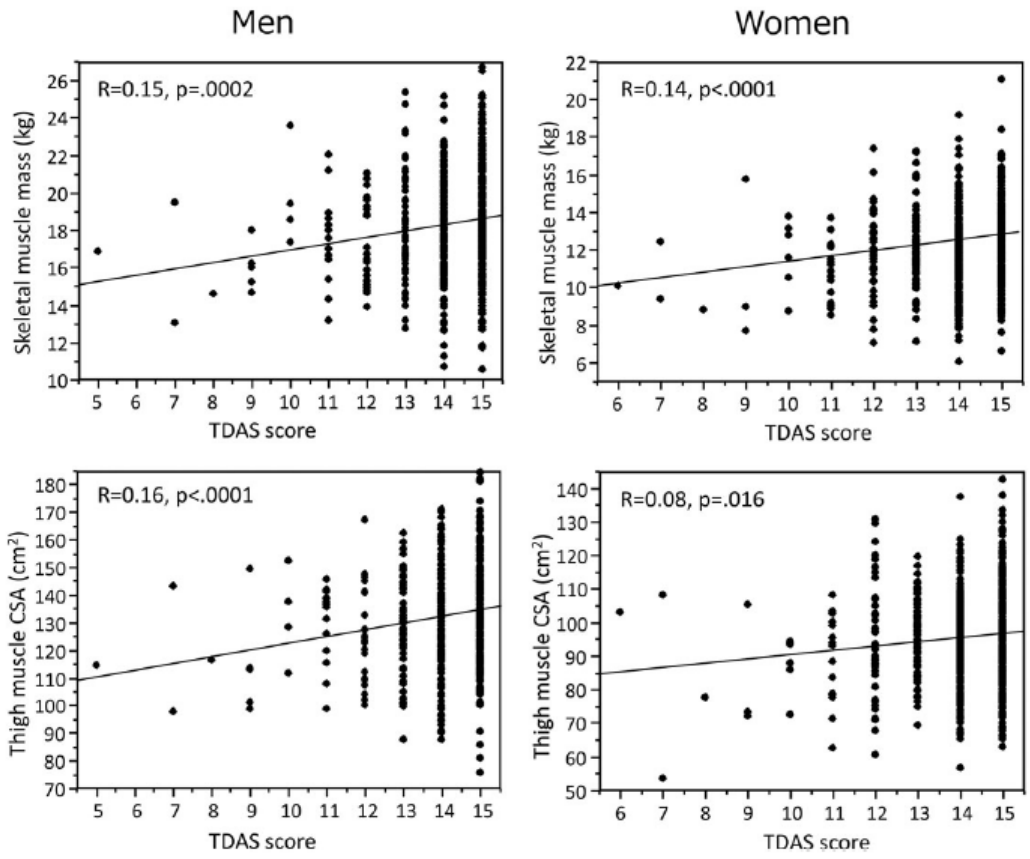
Claudia Szlejf, PhD,[1,2,*] Claudia K. Suemoto, PhD,[1,3] Paulo A. Lotufo, PhD,[1,4] and Isabela M. Benseñor, PhD[1,4]

	Without Sarcopenia (n = 4,946)	With Sarcopenia (n = 92)	p
ALM_{BMI}, median (IQR)	0.707 (0.582 to 0.859)	0.494 (0.437 to 0.669)	<.001[a]
Handgrip strength (kg), median (IQR)	30 (24 to 39)	15 (13 to 23)	<.001[a]
Delayed word recall test z-score	0.03 (–0.45 to 1.01)	0.03 (–0.94 to 0.52)	.122[a]
Verbal fluency test z-score	–0.11 (–0.77 to 0.76)	–0.55 (–1.21 to 0.32)	<.001
Trail making test z-score	0.30 (–0.21 to 0.60)	0.0 (–0.97 to 0.45)	<.001
Global composite cognitive z-score	0.12 (–0.57 to 0.72)	–0.29 (–1.06 to 0.42)	<.001
Age (y), mean ± SD	62.4 ± 5.7	68.0 ± 6.4	<.001[b]
Female, n (%)	2,648 (53.5)	56 (60.9)	.162[b]
Race, n (%)			.368[d]
Black	688 (13.9)	8 (8.7)	
Brown	1,186 (24.0)	23 (25.0)	
White	2,859 (57.8)	55 (59.8)	
Other	213 (4.3)	6 (6.5)	
Higher education, n (%)	2,882 (58.3)	43 (46.7)	.026[c]
Hypertension, n (%)	2,512 (50.8)	60 (65.2)	.006[c]
Diabetes mellitus, n (%)	1,378 (27.9)	40 (43.5)	.001[c]
Previous coronary artery disease, n (%)	120 (2.4)	3 (3.3)	.492[d]
Depression, n (%)	173 (3.5)	6 (6.5)	.143[d]
Obesity, n (%)	1,276 (25.8)	34 (37.0)	.016[c]

Journal of Cachexia, Sarcopenia and Muscle 2017; **8**: 557–566
Published online 29 March 2017 in Wiley Online Library (wileyonlinelibrary.com) **DOI:** 10.1002/jcsm.12195

Muscle mass decline, arterial stiffness, white matter hyperintensity, and cognitive impairment: Japan Shimanami Health Promoting Program study

Figure 3 Scatter plots showing relationship between touch panel-type dementia assessment scale (TDAS) score and thigh muscle cross-sectional area (CSA) (top) and skeletal muscle mass (bottom) in men and women.



Original Study

The Longitudinal Associations of Handgrip Strength and Cognitive Function in Aging Americans

Ryan McGrath PhD [a,*], Brenda M. Vincent MS [b], Kyle J. Hackney PhD [a], Sheria G. Robinson-Lane PhD, RN [c], Brian Downer PhD [d], Brian C. Clark PhD [e,f,g]

A B S T R A C T

Objectives: Factors that are responsible for age-related neurologic deterioration of noncognitive and cognitive processes may have a shared cause. We sought to examine the temporal, directional associations of handgrip strength and cognitive function in a national sample of aging Americans.
Design: Longitudinal panel.
Setting: Enhanced interviews that included physical, biological, and psychosocial measures were completed in person. Core interviews were often conducted over the telephone.
Participants: The analytic sample included 14,775 Americans aged at least 50 years who participated in at least 2 waves of the 2006-2016 waves of the Health and Retirement Study.
Measures: Handgrip strength was measured with a hand-held dynamometer. Participants were considered cognitively intact, mildly impaired, or severely impaired according to the Telephone Interview of Cognitive Status questionnaire. Separate lagged general estimating equations analyzed the directional associations of handgrip strength and cognitive function.
Results: The overall time to follow-up was 2.1 ± 0.4 years. Every 5 kg higher handgrip strength was associated with 0.97 [95% confidence interval (CI) 0.93, 0.99] lower odds for both future cognitive impairment and worse cognitive impairment. Those who were not weak had 0.54 (CI 0.43, 0.69) lower odds for future cognitive impairment and 0.57 (CI 0.46, 0.72) lower odds for future worse cognitive impairment. Conversely, any ($\beta = -1.09$; CI −1.54, −0.64), mild ($\beta = -0.85$; CI −1.34, −0.36), and severe cognitive impairment ($\beta = -2.34$; CI −3.25, −1.42) predicted decreased handgrip strength. Further, the presence of any, mild, and severe cognitive impairment was associated with 1.82 (CI 1.48, 2.24), 1.65 (CI 1.31, 2.08), and 2.53 (CI 1.74, 3.67) greater odds for future weakness, respectively.
Conclusions/Implications: Strength capacity and cognitive function may parallel each other, whereby losses of functioning in 1 factor may forecast losses of functioning in the other. Handgrip strength could be used for assessing cognitive status in aging Americans and strength capacity should be monitored in those with cognitive impairment.

Nov. 2019

Original Study

The Longitudinal Associations of Handgrip Strength and Cognitive Function in Aging Americans

Ryan McGrath PhD [a,*], Brenda M. Vincent MS [b], Kyle J. Hackney PhD [a], Sheria G. Robinson-Lane PhD, RN [c], Brian Downer PhD [d], Brian C. Clark PhD [e,f,g]

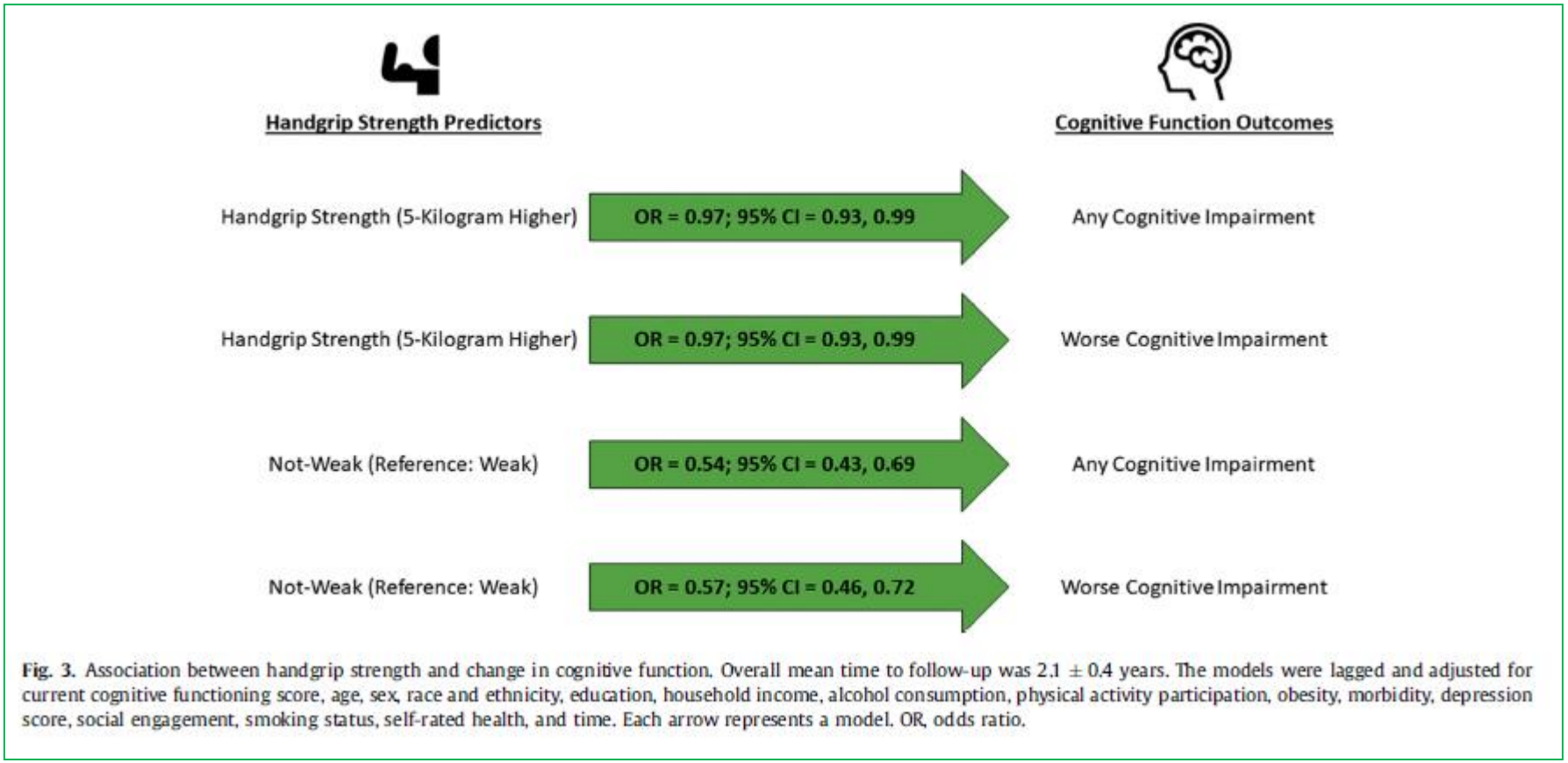


Fig. 3. Association between handgrip strength and change in cognitive function. Overall mean time to follow-up was 2.1 ± 0.4 years. The models were lagged and adjusted for current cognitive functioning score, age, sex, race and ethnicity, education, household income, alcohol consumption, physical activity participation, obesity, morbidity, depression score, social engagement, smoking status, self-rated health, and time. Each arrow represents a model. OR, odds ratio.

Nov. 2019

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Original Study

The Longitudinal Associations of Handgrip Strength and Cognitive Function in Aging Americans

Ryan McGrath PhD [a,*], Brenda M. Vincent MS [b], Kyle J. Hackney PhD [a], Sheria G. Robinson-Lane PhD, RN [c], Brian Downer PhD [d], Brian C. Clark PhD [e,f,g]

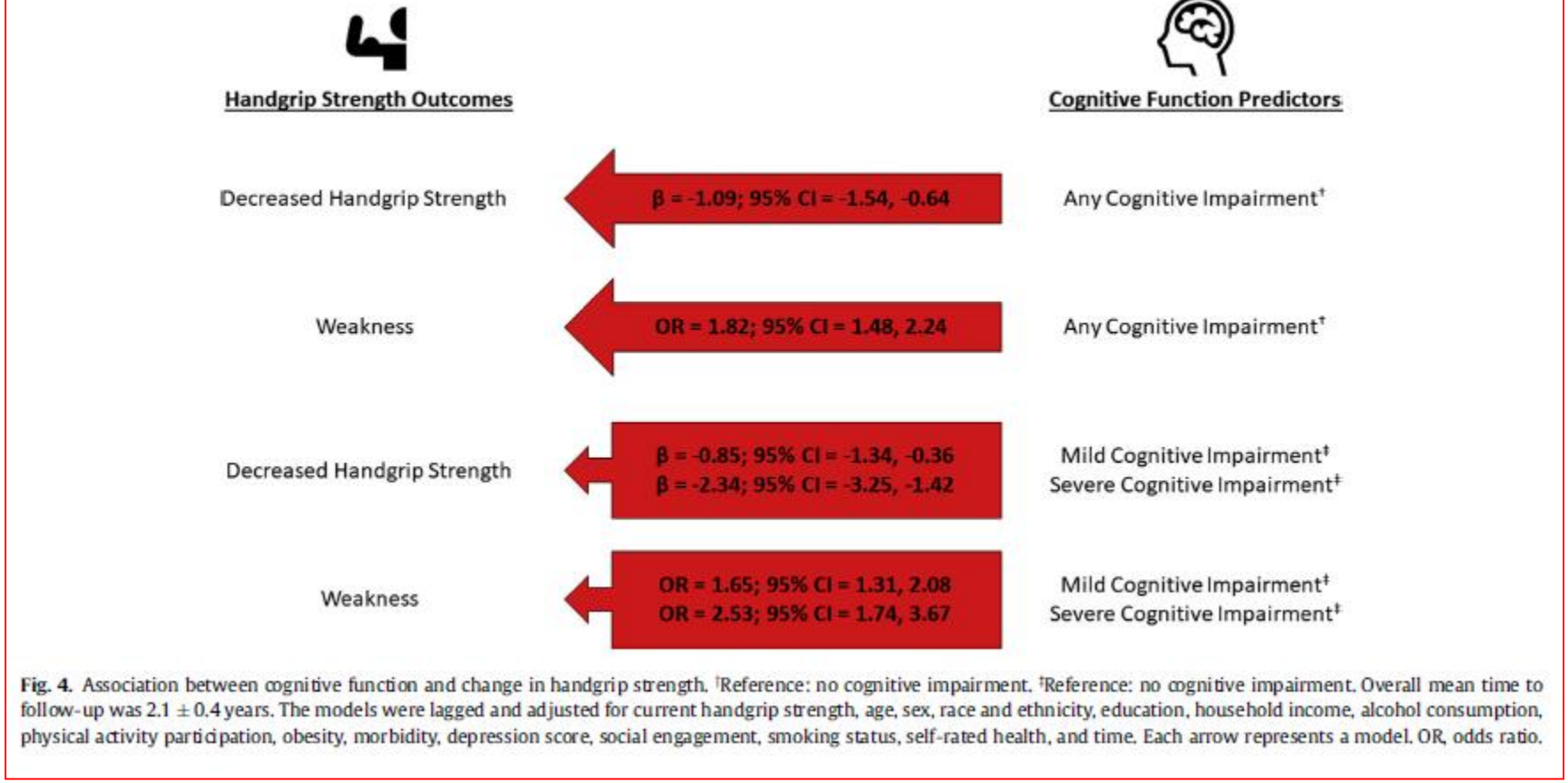

Fig. 4. Association between cognitive function and change in handgrip strength. †Reference: no cognitive impairment. ‡Reference: no cognitive impairment. Overall mean time to follow-up was 2.1 ± 0.4 years. The models were lagged and adjusted for current handgrip strength, age, sex, race and ethnicity, education, household income, alcohol consumption, physical activity participation, obesity, morbidity, depression score, social engagement, smoking status, self-rated health, and time. Each arrow represents a model. OR, odds ratio.

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Evaluation of the directional relationship between handgrip strength and cognitive function: the Korean Longitudinal Study of Ageing

Background: recent studies suggest that handgrip strength is linked with cognitive impairment at older ages. However, it remains unclear as to whether muscular strength influences subsequent cognitive performance, or whether lower levels of cognitive function increase the likelihood of muscle strength decline.

Objective: to investigate the directional relationship between handgrip strength and cognitive impairment using longitudinal data among older adults.

Methods: repeated measures of handgrip strength and cognitive function were collected in a sample of 5,995 participants of the Korean Longitudinal Study of Aging (KLoSA) over a period of 8 years. Time-lagged general estimating equations, while accounting for correlation among repeated measures, was used to assess the temporal effect of handgrip strength on cognitive impairment and vice versa with adjustment for other confounding factors.

Results: after adjustment, greater handgrip strength was related to subsequent reduction in the risk of cognitive impairment, such that participants in the highest quartile presented approximately 50% decrease in their risk of cognitive impairment [adjusted odds ratio (OR) = 0.499 (95% CI 0.422 to 0.589] compared to the lowest quartile after controlling for potential confounding factors. Conversely, cognitive impairment was a significant predictor of reduced muscular strength [β regression coefficient −0.804, 95% CI, −1.168 to −0.439, for participants with dementia compared with those with normal cognitive function].

Conclusions: in conclusion, a significant bi-directional relationship was found between muscular strength and cognitive function, suggesting that these may have shared common pathways that are worthy of being further elucidated in future studies.

Weakness and cognitive impairment are independently and jointly associated with functional decline in aging Americans

Ryan McGrath[1] · Brenda M. Vincent[2] · Kyle J. Hackney[1] · Soham Al Snih[3] · James Graham[4] · Laura Thomas[5] · Diane K. Ehlers[6] · Brian C. Clark[7,8,9]

Abstract

Background Discovering how certain health factors contribute to functional declines may help to promote successful aging.

Aims To determine the independent and joint associations of handgrip strength (HGS) and cognitive function with instrumental activities of daily living (IADL) and activities of daily living (ADL) disability decline in aging Americans.

Methods Data from 18,391 adults aged 50 years and over who participated in at least one wave of the 2006–2014 waves of the Health and Retirement Study were analyzed. A hand-held dynamometer assessed HGS and cognitive functioning was examined with a modified version of the Telephone Interview of Cognitive Status. IADL and ADL abilities were self-reported. Participants were stratified into four distinct groups based on their HGS and cognitive function status. Separate covariate-adjusted multilevel models were conducted for the analyses.

Results Participants who were weak, had a cognitive impairment, and had both weakness and a cognitive impairment had 1.70 (95% confidence interval (CI) 1.57–1.84), 1.97 (CI 1.74–2.23), and 3.13 (CI 2.73–3.59) greater odds for IADL disability decline, respectively, and 2.26 (CI 2.03–2.51), 1.26 (CI 1.05–1.51), and 4.48 (CI 3.72–5.39) greater odds for ADL disability decline, respectively.

Discussion HGS and cognitive functioning were independently and jointly associated with IADL and ADL disability declines. Individuals with both weakness and cognitive impairment demonstrated substantially higher odds for functional decline than those with either risk factor alone.

Conclusions Including measures of both HGS and cognitive functioning in routine geriatric assessments may help to identify those at greatest risk for declining functional capacity.

Aging Clin Exp Res. 2019 Sep 13

Weakness and cognitive impairment are independently and jointly associated with functional decline in aging Americans

Ryan McGrath[1] · Brenda M. Vincent[2] · Kyle J. Hackney[1] · Soham Al Snih[3] · James Graham[4] · Laura Thomas[5] · Diane K. Ehlers[6] · Brian C. Clark[7,8,9]

Table 2 Independent and joint associations of handgrip strength and cognitive function on instrumental activities of daily living disability decline

	Odds ratio	95% confidence interval
Weakness only[a]	1.70	1.57, 1.84
Cognitive impairment only[a]	1.97	1.74, 2.23
Both weakness and cognitive impairment[a]	3.13	2.73, 3.59
Age	1.07	1.06, 1.07
Body mass index	1.00	1.00, 1.01
Morbidity[b]	1.21	1.18, 1.25
Depression score	1.19	1.18, 1.21
Time since entry	0.92	0.91, 0.93
Black race (reference: white race)	1.82	1.65, 2.02
Male (reference: not-male)	0.59	0.54, 0.65
Current smoker (reference: non-smoker)	1.34	1.21, 1.49
Previous smoker (reference: non-smoker)	0.94	0.86, 1.03
Self-rated health (reference: excellent)		
Very good	0.99	0.88, 1.13
Good	1.46	1.28, 1.67
Fair	2.69	2.34, 3.09
Poor	4.51	3.83, 5.31

Table 3 Independent and joint associations of handgrip strength and cognitive function on activities of daily living disability decline

	Odds ratio	95% confidence interval
Weakness only[a]	2.26	2.03, 2.51
Cognitive impairment only[a]	1.26	1.05, 1.51
Both weakness and cognitive impairment[a]	4.48	3.72, 5.39
Age	1.04	1.03, 1.04
Body mass index	1.06	1.05, 1.07
Morbidity[b]	1.50	1.44, 1.56
Depression score	1.32	1.29, 1.34
Time since entry	0.95	0.94, 0.97
Black race (reference: white race)	1.04	0.91, 1.19
Male (reference: not-male)	0.91	0.82, 1.01
Current smoker (reference: non-smoker)	1.19	1.03, 1.37
Previous smoker (reference: non-smoker)	1.07	0.96, 1.19
Self-rated health (reference: excellent)		
Very good	1.21	0.99, 1.47
Good	2.39	1.97, 2.91
Fair	6.35	5.18, 7.79
Poor	20.64	16.31, 26.11

Aging Clin Exp Res. 2019 Sep 13

Disability trajectories and mortality in older adults with different cognitive and physical profiles

Giulia Grande[1] · Davide L. Vetrano[1,3,4] · Laura Fratiglioni[1,2] · Anna Marseglia[1] · Nicola Vanacore[5] · Erika Jonsson Laukka[1] · Anna-Karin Welmer[1] · Debora Rizzuto[1]

Abstract

Background Cognitive and physical deficits independently raise the risk for negative events in older adults. Less is known about whether their co-occurrence constitutes a distinct risk profile. This study quantifies the association between cognitive impairment, no dementia (CIND), slow walking speed (WS) and their combination and disability and mortality.

Methods We examined 2546 dementia-free people aged ≥ 60 years, part of the Swedish National study on Aging and Care in Kungsholmen (SNAC-K) up to 12 years. The following four profiles were created: (1) healthy profile; (2) isolated CIND (scoring 1.5 SD below age-specific means on at least one cognitive domain); (3) isolated slow WS (< 0.8 m/s); (4) CIND+ slow WS. Disability was defined as the sum of impaired activities of daily living and trajectories of disability were derived from mixed-effect linear regression models. Piecewise proportional hazard models were used to estimate mortality rate [hazard ratios (HRs)]. Population attributable risks of death were calculated.

Results Participants with both CIND and slow WS had the worst prognosis, especially in the short-term period. They experienced the steepest increase in disability and five times the mortality rate (HR 5.1; 95% CI 3.5–7.4) of participants free from these conditions. Similar but attenuated results were observed for longer follow-ups. Co-occurring CIND and slow WS accounted for 30% of short-term deaths.

Conclusions Co-occurring cognitive and physical limitations constitute a distinct risk profile in older people, and account for a large proportion of short-term deaths. Assessing cognitive and physical function could enable early identification of people at high risk for adverse events.

Aging Clinical and Experimental Research **2019**

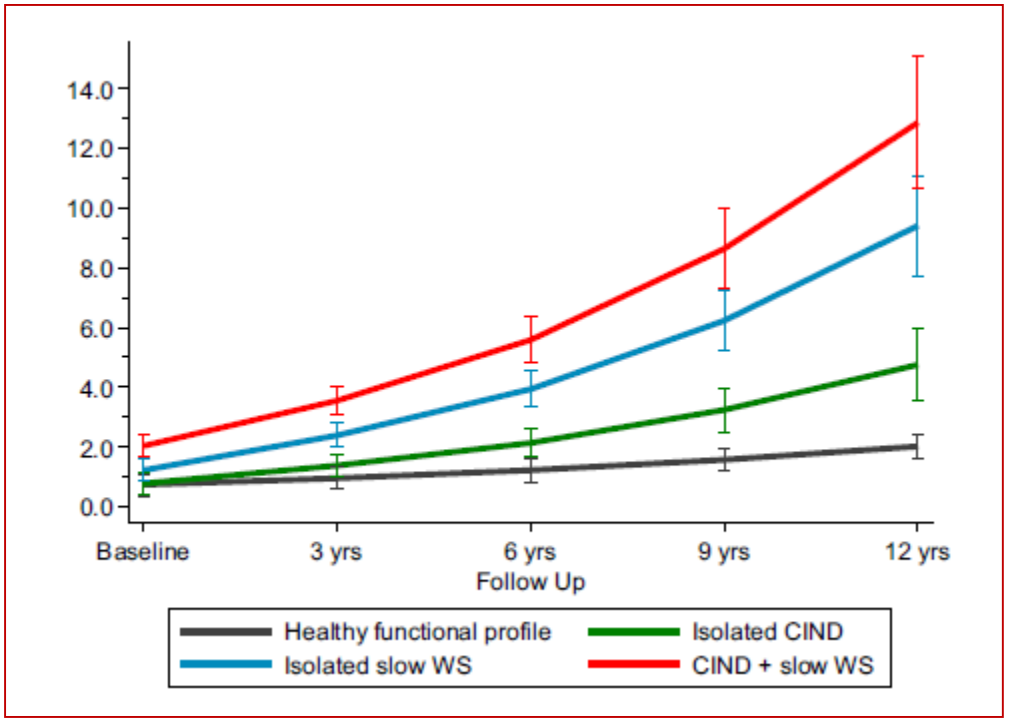


Fig. 3 Trajectories with 95% confidence intervals of disability over 12 years of follow-up by functional profiles. CIND, Cognitive impairment, no dementia; slow WS: walking speed < 0.8 m/s. Trajectories were derived from multilevel mixed-effect linear regression models adjusted for age, sex, education, socioeconomic position, physical inactivity, time to death, cardio- and cerebrovascular diseases, hypertension, depression and mood disorders, solid neoplasms, chronic obstructive pulmonary diseases, and malnutrition. Healthy functional profile is intended as participants without CIND and with a walking speed ≥ 0.8 m/s

Aging Clinical and Experimental Research **2019**

Journal of
Clinical Medicine

MDPI

Article

Prognosis and Interplay of Cognitive Impairment and Sarcopenia in Older Adults Discharged from Acute Care Hospitals

Elisa Zengarini [1], Robertina Giacconi [2], Lucia Mancinelli [3], Giovanni Renato Riccardi [4], Daniele Castellani [5,*], Davide Liborio Vetrano [6,7], Graziano Onder [7], Stefano Volpato [8], Carmelinda Ruggiero [9], Paolo Fabbietti [10], Antonio Cherubini [1], Francesco Guarasci [10], Andrea Corsonello [10] and Fabrizia Lattanzio [11]

Abstract: Sarcopenia and cognitive impairment are associated with an increased risk of negative outcomes, but their prognostic interplay has not been investigated so far. We aimed to investigate the prognostic interaction of sarcopenia and cognitive impairment concerning 12-month mortality among older patients discharged from acute care wards in Italy. Our series consisted of 624 patients (age = 80.1 ± 7.0 years, 56.1% women) enrolled in a prospective observational study. Sarcopenia was defined following the European Working Group on Sarcopenia in Older People (EWGSOP) criteria. Cognitive impairment was defined as age- and education-adjusted Mini-Mental State Examination (MMSE) score < 24 or recorded diagnosis of dementia. The study outcome was all-cause mortality during 12-month follow-up. The combination of sarcopenia and cognitive ability was tested against participants with intact cognitive ability and without sarcopenia. Overall, 159 patients (25.5%) were identified as having sarcopenia, and 323 (51.8%) were cognitively impaired. During the follow-up, 79 patients (12.7%) died. After adjusting for potential confounders, the combination of sarcopenia and cognitive impairment has been found associated with increased mortality (HR = 2.12, 95% CI = 1.05–4.13). Such association was also confirmed after excluding patients with dementia (HR = 2.13, 95% CI = 1.06–4.17), underweight (HR = 2.18, 95% CI = 1.03–3.91), high comorbidity burden (HR = 2.63, 95% CI = 1.09–6.32), and severe disability (HR = 2.88, 95% CI = 1.10–5.73). The co-occurrence of sarcopenia and cognitive impairment may predict 1-year mortality in older patients discharged from acute care hospitals.

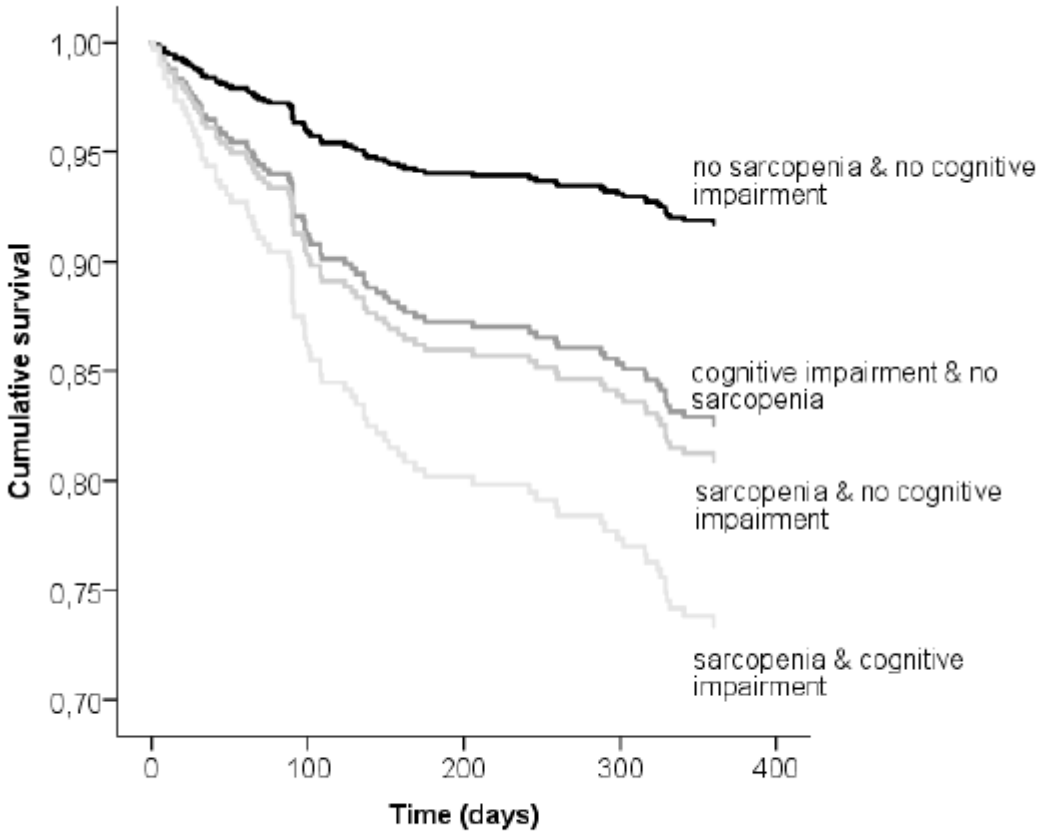
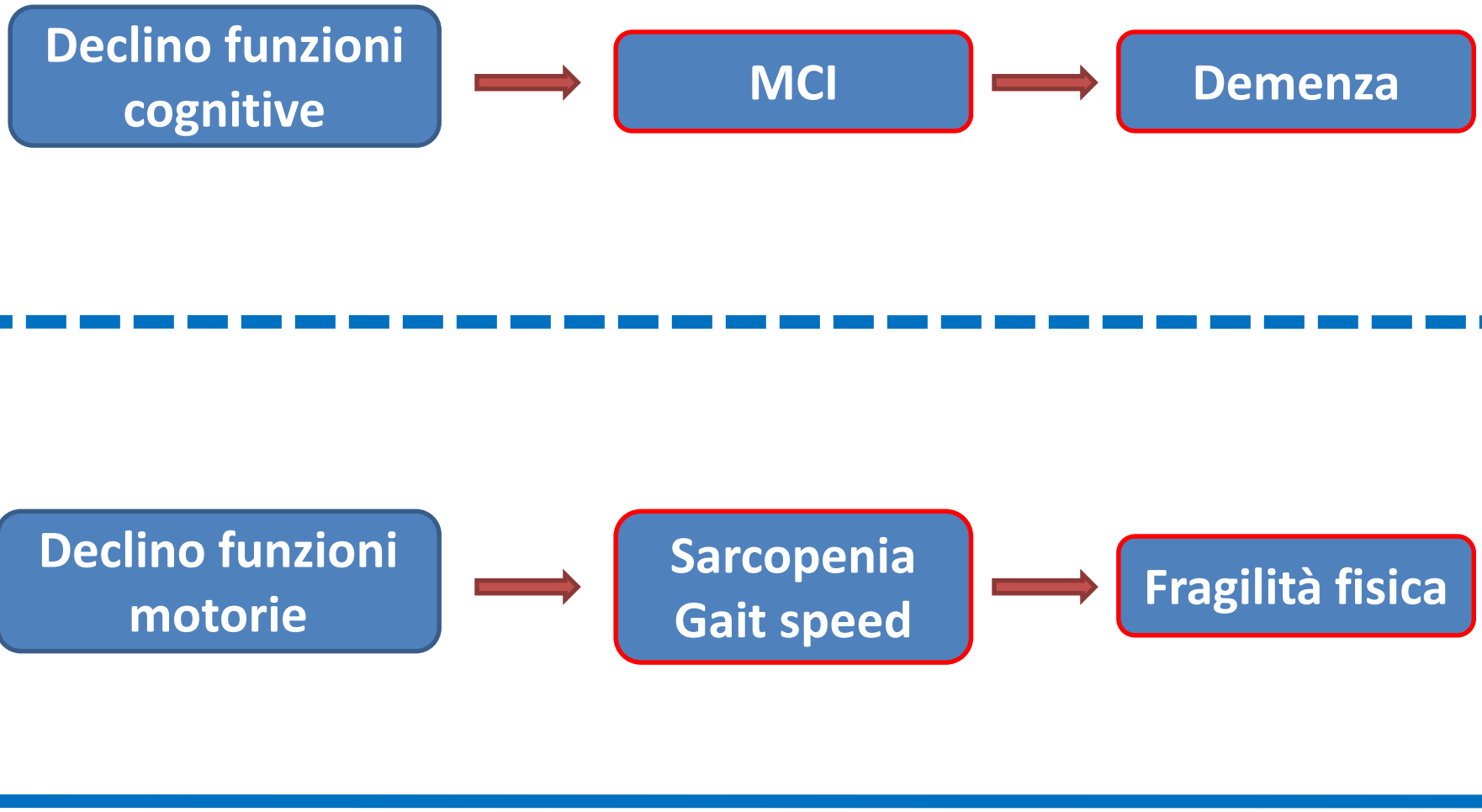


Figure 1. Kaplan–Meier curves showing survival according to the presence of cognitive impairment, sarcopenia or combined sarcopenia and cognitive impairment.


Declino funzioni cognitive
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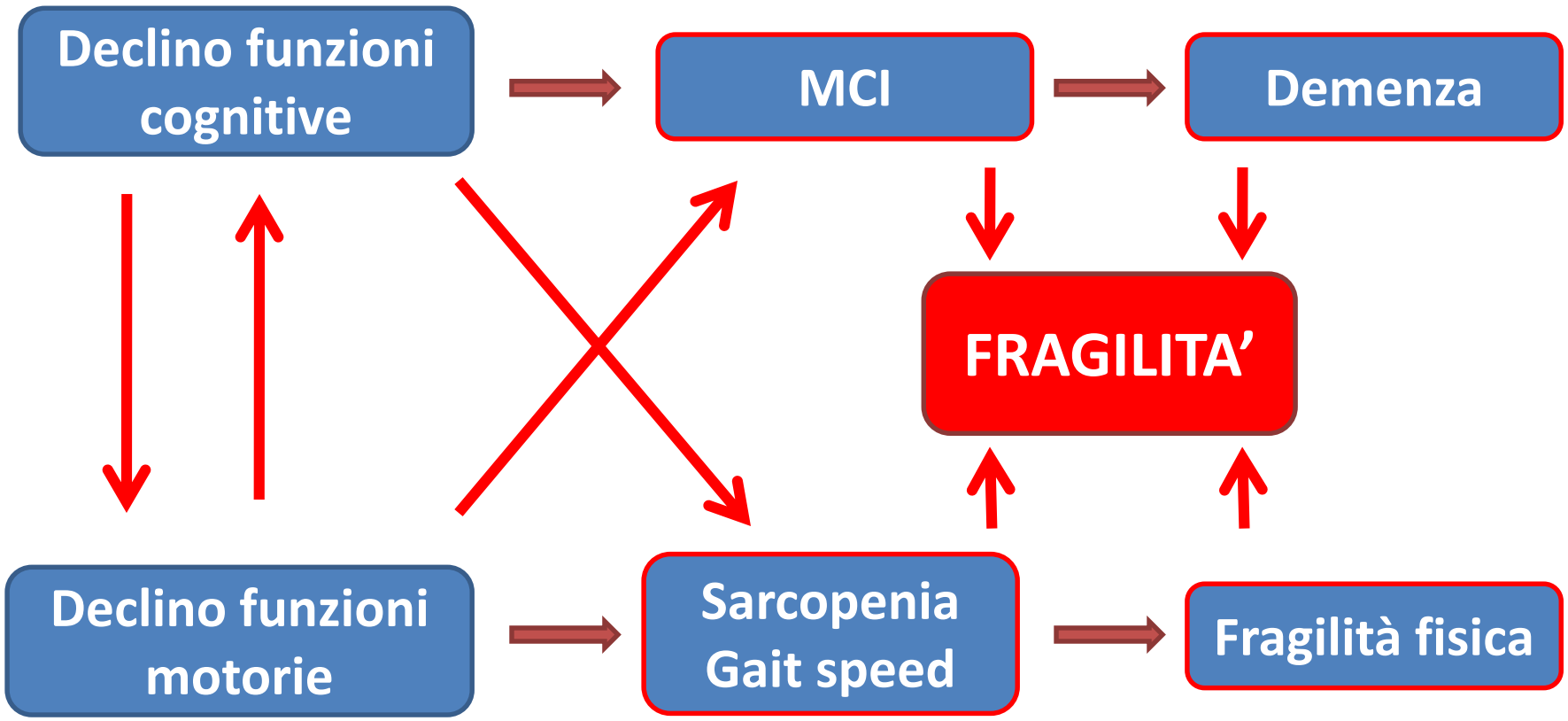
Demenza
Declino funzioni motorie
Sarcopenia
Gait speed
Fragilità fisica


Declino funzioni cognitive
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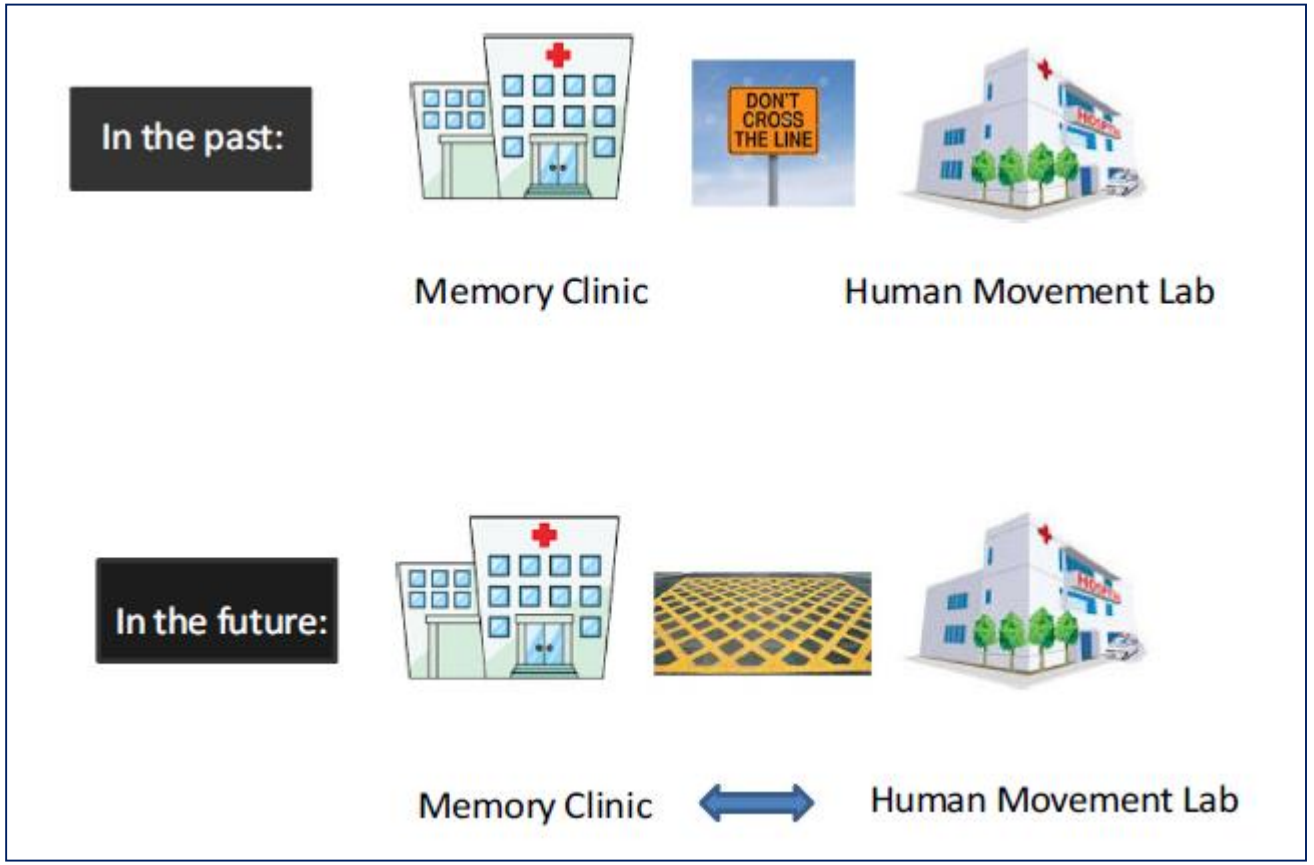
Declino funzioni motorie
Sarcopenia Gait speed
Fragilità fisica

POINT OF VIEW

"Brain-muscle loop" in the fragility of older persons: from pathophysiology to new organizing models

Fulvio Lauretani[1,3] · Tiziana Meschi[1] · Andrea Ticinesi[1] · Marcello Maggio[2,3]

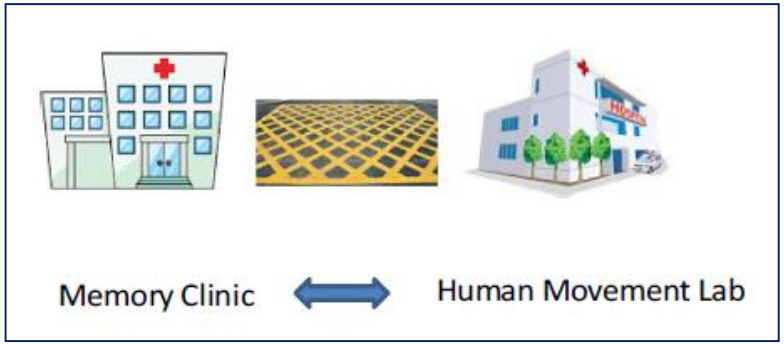


Aging Clin Exp Res 2017 Dec; 29(6):1305-1311

MMG

Ambulatorio di Geriatria

- Valutazione Multidimensionale
  - ...
  - MMSE/MoCA
  - Handgrip
  - gait speed (4 m)
  - ...
- Fragilità
- Traiettoria individuale
- Prognosi



Grazie per l'attenzione!