

NIH Public Access

Author Manuscript

Arch Intern Med. Author manuscript; available in PMC 2010 June 22.

Published in final edited form as:

Arch Intern Med. 2009 June 22; 169(12): 1139–1146. doi:10.1001/archinternmed.2009.135.

Association between Late-Life Social Activity and Motor Decline in Older Adults

Aron S. Buchman, MDa,b, **Patricia A. Boyle, PhD**a,c, **Robert S. Wilson, PhD**a,b,c, **Debra A. Fleischman, PhD**a,b,c, **Sue Leurgans, PhD**a,b, and **David A. Bennett, MD**a,b

^a Rush Alzheimer's Disease Center, Rush University Medical Center; Armour Academic Facility, Suite #1038; 600 South Paulina Street, Chicago, Illinois 60612, USA

b Department of Neurological Sciences, Rush University Medical Center

c Department of Behavioral Science, Rush University Medical Center

Abstract

Background—Loss of motor function is a common consequence of aging, but little is known about factors that predict idiopathic motor decline.

Methods—We studied 906 persons without dementia, history of stroke or Parkinson's disease participating in the Rush Memory and Aging Project. At baseline, they rated their frequency of participation in common social activities. Outcome was annual change in global motor function, based on nine measures of muscle strength and nine motor performances.

Results—Mean social activity score at baseline was 2.6 (SD=0.58), with higher scores indicating more frequent participation in social activities. In a generalized estimating equation model, controlling for age, sex and education, motor function declined by about 0.05 unit/year [Estimate, 0.016; 95%CI (-0.057, -0.041); p=0.017]. Each 1-point decrease in social activity was associated with about a 33% more rapid rate of decline in motor function [Estimate, 0.016; 95%CI (0.003, (0.029) ; p=0.017)]. This amount of annual motor decline was associated with a more than 40% increased risk of death (Hazard Ratio: 1.44; 95%CI: 1.30, 1.60) and 65% increased risk of incident Katz disability (Hazard Ratio: 1.65; 95%CI: 1.48, 1.83). The association of social activity with change in motor function did not vary along demographic lines and was unchanged after controlling for potential confounders including late-life physical and cognitive activity, disability, global cognition, depressive symptoms, body composition and chronic medical conditions [Estimate, 0.025; 95%CI (0.005, 0.045); p=0.010].

Corresponding Author: Aron S. Buchman, MD, Phone: (312) 942-8974, Fax (312) 563-4605; Aron_S_Buchman@rush.edu. *Conflict of Interests:* The authors have no conflicts of interest to report.

Financial Disclosures: This work was supported by National Institute on Aging grants R01AG17917 (Buchman, Wilson, Leurgans, Bennett), R01AG24480 (Buchman, Leurgans, Bennett), K23 AG23040 (Boyle), the Illinois Department of Public Health, and the Robert C. Borwell Endowment Fund.

Authors Contributions: Dr. Buchman had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. He was involved with study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. Dr. Boyle analyzed and interpreted the data and critically revised the manuscript for important intellectual content. Dr. Wilson was involved with study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. Dr. Fleischman analyzed and interpreted the data and critically revised the manuscript for important intellectual content. Dr. Leurgans provided statistical analyses and interpretation of data and drafted the manuscript. Dr. Bennett was involved with study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content.

Sponsor's Role: The organizations funding this study had no role in the design or conduct of the study; in the collection, management, analysis, or interpretation of the data; or in the preparation, review, or approval of the manuscript.

Conclusion—Less frequent participation in social activities is associated with a more rapid rate of motor decline in old age.

Introduction

Idiopathic decline in motor function is a familiar consequence of aging with older persons displaying a wide spectrum of loss of motor abilities ranging from mild decreased muscle strength and bulk and reduced speed and dexterity to overt motor impairment with concomitant disability. The motor deficits observed in older persons have been subsumed under several terms including sarcopenia,¹ physical frailty 2 and parkinsonian signs³ and are widely known to be related to adverse health outcomes including death, $4, 5$ disability, $6, 7$ and dementia.^{8,} ⁹ Although risk factors for common diseases known to cause motor dysfunction such as stroke are recognized, few risk factors for idiopathic motor decline have been identified.

Studies by our group and others have identified physical activity as a factor associated with the rate of declining motor function in community-dwelling elders.10-13 However, accumulating evidence suggests that a much broader range of leisure activities including latelife social activity are associated with health benefits such as longevity, 14 risk of dementia and rate of cognitive decline.^{15, 16} In animal studies, a broad array of activities including social, physical and cognitive activities are associated with a slower rate of functional decline.¹⁷ However, we are unaware of studies which have examined the extent to which late-life social activity is related to the rate of decline of motor performances in old age. We used data from more than 900 older participants in the Rush Memory and Aging Project, who underwent annual detailed examinations for up to 11 years ,¹⁸ to test the hypothesis that the frequency of participation in late-life social activity is related to the rate of motor decline.

Methods

Participants

Participants were recruited from about 40 retirement facilities and subsidized housing facilities, as well as from church groups and social service agencies in northeastern Illinois. All participants signed an informed consent agreeing to annual clinical evaluation. The study was in accordance with the latest version of the Declaration of Helsinki and was approved by our institutional review board. The clinical evaluation was uniform and included a medical history, complete neurological examination, and assessment of cognitive and motor function. Followup evaluations, were performed annually by examiners blinded to previously collected data.¹⁸

At the time of these analyses, 1194 participants had enrolled and completed a baseline evaluation. Eligibility for these analyses required the absence of clinical dementia, stroke or Parkinson's disease at the baseline evaluation, a valid assessment of social, physical and cognitive activities and motor assessment at baseline as well as at least one follow-up motor evaluation in order to assess change in motor function. We excluded 71 persons who met criteria for dementia at baseline, 114 with stroke, 15 with Parkinson's disease, and 1 with both, 41 persons who had completed a baseline evaluation but died before their first follow-up examination or had not been in the study long enough for follow-up evaluation, and 47 persons with incomplete data, leaving 906 participants for these analyses. The project began in 1997 and follow-up data through September of 2008 were analyzed. Because of the rolling admission and mortality, the length of follow-up and number of examinations varies across participants. Of the 906 persons included in these analyses, 195 died (21.5%) during the course of followup [mean 4.5 years (SD= 2.44 years)] There was missing data from 279 of 4747 examinations (5.8%) during the course of follow-up.

Clinical Diagnoses

Clinical diagnoses were made using a multi-step process, as previously described.18 Cognitive function testing included 19 performance tests were summarized into a composite measure of global cognition as described previously.¹⁸ Participants were then evaluated in person by an experienced neurologist or geriatrician who diagnosed dementia, stroke, Parkinson's disease, and other common neurologic conditions affecting cognitive or physical function. Criteria for dementia followed the joint working group of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association.19 Diagnosis of stroke was made as outlined for the Trial of ORG 10172 in Acute Stroke Treatment (TOAST). 20 The diagnosis of Parkinson's disease was made according to the clinical criteria recommended by the Core Assessment Program for Intracerebral Transplantation (CAPIT). ²¹

Assessment of Motor Function

Grip and pinch strength were measured bilaterally using the Jamar hydraulic dynamometers (Lafayette Instruments, Lafayette, IN). Hand-held dynamometry (Lafayette Manual Muscle Test System, Model 01163, Lafayette, IN) was used to assess muscle strength in arm abduction, arm flexion, arm extension, hip flexion, knee extension, plantar flexion, and ankle dorsiflexion bilaterally. Time and number of steps to walk 8 feet and turn 360° were measured. Time to stand on each leg and then on their toes for 10 seconds. We counted the number of steps off line when walking an 8 foot line in a heel to toe manner. We also measured the number of pegs that could be placed (Purdue pegboard) in 30 seconds and the rate of index finger tapping for 10 seconds (Western Psychological Services, Los Angeles, CA) bilaterally. Composite measures have been used effectively in other longitudinal studies of cognitive and motor function.^{8, 22, 23} A composite measure of global motor function was constructed by converting the raw score from each of the 18 motor measures to z scores using the mean and standard deviation from all participants at baseline (Table 1) and averaging z scores of all of the motor tests together as previously described.^{12, 24}

Assessment of Social Activity

We used a previously established composite measure of late-life social activity in these analyses.25, 26 Frequency of participation in social activity was assessed with a previously established scale based on 6 items about activities involving social interaction [1) go to restaurants, sporting events or teletract, or play bingo, 2) go on day trips or overnight trips, 3) do unpaid community/volunteer work, 4) visit relatives or friends houses, 5) participate in groups, such as senior center, Knights of Columbus, Rosary Society or something similar, 6. attend church or religious services]. Each activity was rated on a 5-point scale, with 1 indicating participation in the activity once a year or less; 2, several times a year; 3, several times a month; 4, several times a week; and 5, every day or almost every day. Responses on each item were averaged to yield the composite measure used in analyses as previously described.²⁶

Assessment of Other Covariates

Gender was recorded at the baseline interview. Age in years was computed from self-reported date of birth, and date of the baseline clinical examination was that at which the strength measures were first collected. Education (reported highest grade or years of education) was obtained at the time of the baseline cognitive testing. Weight and height were measured and recorded at each visit by a trained technician blinded to previously collected data. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared.

As done in previous studies, physical activity was assessed using questions adapted from the 1985 National Health Interview Survey. Participants were asked if they had engaged in any of

the activities within the past two weeks (e.g., walking for exercise, gardening or yardwork) and, if so, the number of occasions and average minutes per occasion. Minutes spent engaged in each activity were summed and expressed as hours of activity/week.¹²

Frequency of participation in cognitively stimulating activities was quantified with a previously established scale.²⁷ People rated how often they had participated in each of 7 cognitive activities (e.g., reading a newspaper) in the past year on a 5-point scale and the mean score for the seven activities was used in these analyses.

Disability was assessed at baseline with the 6-item Katz scale, ²⁸ 3- item Rosow-Breslau scale, 29 and eight items which assessed Instrumental activities of daily living (IADL) adapted from the Duke Older Americans Resources and Services project. ³⁰

Depressive symptoms were assessed with a 10-item version of the Center for Epidemiologic Studies Depression (CES-D) scale.³¹ Persons were asked whether they had experienced each of 10 symptoms in the past week, and the score was the number of symptoms reported.³²

As in previous studies, the sum of the number of vascular risk factors (i.e. the sum of hypertension, diabetes mellitus, and smoking), and vascular diseases (i.e., myocardial infarction, congestive heart failure, and claudication) were used in these analyses.³³ Joint pain was based on participant report.

Statistical Analyses

We examined the bivariate associations of late-life social activity and global motor function with age, education and other covariates. Then we divided the participants into two groups: high and low frequency of participation in social activity at baseline based on the median value and compared their demographic and covariate measures at baseline. We used generalized estimating equation models³⁴ to assess the relation of social activity with baseline level of global motor function and its annual rate of change. The core model included terms for time in years since baseline as well as terms for social activity at baseline which was centered at its mean and a term for its interaction with time since baseline. The term for time indicates the average rate of change in global motor function for a typical participant with a social activity score of 2.6; the term for social activity indicates the average difference in motor function at baseline associated with a 1- point change in social activity score; and the interaction of social activity with time indicates the effect of a 1-point change in social activity score on the annual rate of change in global motor function. To control for the effect of demographic variables, these and all subsequent models included terms for age, sex, and education and their interaction with time. In subsequent models, we added terms for the interactions of age, sex, and education with social activity. Next we examined several potential confounders of the association of social activity with motor function. Because of sex differences in level of and rate of decline in motor function, we also examined three way interactions of sex \times social activity \times motor function. To determine the clinical significance of the amount of change in global motor function, we constructed Cox proportional hazards models examining adverse health consequences of change in motor function and estimated the hazard ratios associated with a given unit of change. These models controlled for age, sex, education, and baseline global motor function. For these analyses we used ordinary least squares regression to estimate the annual rate of change in global motor function for each person. Finally, in exploratory analyses, we examined whether individual social activities were associated with rate of global motor decline. Models were examined graphically and analytically and assumptions were judged to be adequately met. *Apriori* level of statistical significance was 0.05. Using a mixed-model crude estimate of power, we estimate that a sample size of 900 persons with a follow-up pattern and distribution of social activity similar to that seen would have 80% power to detect a

coefficient of 0.0082 for the coefficient measuring the effect of social activity on motor function.³⁵ Programming was done in SAS version 9.1.3 (SAS Institute Inc, Cary, NC).³⁶

Results

Baseline Global Motor Function

There were 906 persons in these analyses with a mean follow-up of 4.9 years (SD, 2.21; range 2, 11 years), Baseline motor function ranged from -2.1 to 2.1 (mean, -0.02; SD, 0.57). Global motor function was inversely related to age ($r = -0.45$. p<0.001), positively associated with education ($r=0.19$, $p<0.001$), and men had higher levels of global motor function (mean, 0.25; SD, 0.58) than women (mean, -0.11 ; SD, 0.54) [t [904] = -8.69 , p < 0.001]. As expected, global motor function was associated with other activity measures, disability, cognition, depressive symptoms, vascular diseases and joint pain (Table 2).

Social Activity and Change in Global Motor Function

Baseline social activity scores were approximately normally distributed (mean, 2.6; SD, 0.58; skewness, -0.18). Scores ranged from 1.00 to 4.17 with higher values indicating more frequent participation in social activity. Social activity was inversely related to age $(r = -0.17, p < 0.001)$, positively associated with education $(r = 0.14, p < 0.001)$, and women had higher levels of social activity (mean, 2.6; SD, 0.57) than men (mean, 2.5; SD, 0.61), [t [904] = 2.23, p=0.026]. Social activity was associated with global motor function, activity measures, disability, cognition, and depressive symptoms (Table 2).

Participants who reported low social activity at baseline were older, more likely to be male, less educated, reported less frequent participation in physical and cognitive activities, reported more disability, had lower cognitive function, and were more likely to have lower BMI and diabetes than those who reported high social activity (Table 3).

We used a generalized estimating equation model to test the hypothesis that more frequent participation in social activity is associated with a slower rate of decline in global motor function. On average, global motor function declined at a rate of about 0.05 unit/year (Time, Table 4). Baseline frequency of participation in social activity was associated with both baseline level of global motor function (Social Activity, Table 4) and the rate of change in global motor function (Social Activity*Time, Table 4). That is, for each point below the mean social activity score at baseline, the average rate of decline in global motor function was 33% more rapid (Time, Table 4). Since age was also related to the rate of global motor decline, we can compare the amount of global motor decline associated with increased age with the amount of motor decline associated with social activity. For each additional year of age, global motor function declined an additional 0.003 standard units (Age*Time, Table 4). In contrast, each point decease in social activity, global motor function declined an additional 0.016 standard unit (Social Activity*Time, Table 4). Thus, in terms of declining motor function, a 1-point decrease on the social activity scale was equivalent to being about 5 years older at baseline.

The association of social activity with motor decline did not vary along demographic lines (results not shown). In a sensitivity analysis, we excluded participants who were unable to ambulate at baseline and the association was unchanged [Estimate, 0.017; 95% CI (-0.004, 0.030), $p<0.010$].

To illustrate the findings with a common measure, we used a similar model to examine the relationship between social activity and the rate of change in walking speed. In the average participant, walking speed at baseline was about 65cm/s and declined at about 2cm/sec/year. In contrast gait speed in a person with high social activity (score= 3.3 , $90th$ percentile) declined

by about 1.5cm/sec/year versus 2.6cm/sec/year for a participant with low social activity $\text{(score=1.8, 10}^{\text{th}} \text{ percentile)}.$

Social Activity, Other Covariates and the Rate of Change in Global Motor Function

Next we examined a number of covariates which might affect the association of social activity with change in motor function. None of these additional analyses altered the estimate of the association (Table 5). First, we adjusted for cognitive and physical activity (Table 5, Model 1). Next, we added terms for baseline disability using the Katz, Rosow-Breslau and IADL scales (Table 5, Model 2). We next adjusted for baseline global cognition and depressive symptoms (Table 5, Models $3 \& 4$). Then we examined a number of health-related covariates including body composition, vascular risk factors, vascular disease burden and joint pain (Table 5, Model 5). Finally all of the above covariates were included in a single model and social activity remained associated with the rate of motor decline (Table 5, Model 6).

Clinical Significance of Change in Global Motor Function

To determine the clinical significance of the amount of change in global motor function associated with social activity identified in the analyses above, we constructed Cox proportional hazards models examining the association of change in motor function with death and disability and subsequently estimated the hazard ratios associated with a change of 0.16 unit/year, i.e., the amount of change in global motor function associated with a 1-point decrease on the social activity scale. From these models (data not shown), we calculated that a mean annual change in motor function of 0.16 unit/year (Table 4, Social Activity \times Time) is associated with a more than 40% increased risk of death (Hazard Ratio: 1.44; 95% CI: 1.30, 1.60); 65% increased risk of incident Katz disability (Hazard Ratio: 1.65; 95% CI: 1.48, 1.83) and 34% increased risk of incident Rosow Breslau disability (Hazard Ratio: 1.34; 95% CI: 1.18, 1.52).

Components of Social Activity and Change in Global Motor Function

In a series of exploratory analyses, we examined the relation of each social activity index to rate of global motor decline. Three of the six activities were related to motor decline: unpaid volunteer or community work [Estimate 0.006; 95%CI (0.001, 0.021), p=0.027]; visiting friends or relatives [Estimate, 0.012; 95%CI (0.005, 0.019), p<0.001] and attending church or religious services [Estimate, 0.011 95%CI (0.0007, 0.0018), p=0.027].

Comment

In a cohort of more than 900 older persons free of dementia, stroke or Parkinson's disease at baseline, we found that a lower frequency of participation in social activity was associated with a more rapid rate of motor decline. The effect size was equivalent to about 5 years of age; an amount of change associated with more than 40% increased risk of death and more than 65% increased risk of developing disability. Moreover, the association of social activity was robust to a wide range of potential confounding variables and remained unchanged after controlling for disability and excluding persons unable to ambulate at baseline reducing the potential for reverse causality. These findings expand upon the accumulating literature showing that participation in a broad spectrum of late-life activities are associated with positive health outcomes in old age and suggest that more frequent participation in social activity may be protective against motor decline in older persons.

It is widely recognized that increased levels of physical activity are associated with a slower rate of motor decline and a reduced risk of other adverse health outcomes.¹⁰⁻¹³ However, emerging data suggest that physical activity is only one component of an active and healthy lifestyle.16 For example, increased cognitive and social activities in the elderly are associated

with increased survival and a decreased risk of dementia.³⁷⁻⁴¹ In addition, a number of studies have reported a link between social activity and disability or functional status.^{25, 42} The current study extends these previous studies by showing that late-life participation in social activity is related to the rate of change in motor function based on objective quantitative measures. Further, the association persisted even after controlling for the frequency of participation in physical and cognitive activities. These findings may be particularly relevant for intervention strategies designed for older adults, for whom participation in physical activities may be constrained because of underlying health problems. Furthermore, these results have important translational implications because they suggest that public health interventions using a broader range of leisure activities might increase the efficacy of efforts to decrease the burden of agerelated motor decline.

The basis for the association between social activity and motor decline is uncertain. Emerging evidence suggests that efficient goal-directed movement requires the orchestration and integration of a wide range of sensory, motor and cognitive functions^{43, 44} Human social interaction is complex and social behavior is generated in the brain through interconnected brain structures which process different elements of sociocognitive and socioaffective information which are eventually integrated and translated into action.45 Thus, both successful social and motor behavior depend on the structural and functional integrity of neural systems that integrate the varied inputs needed for planning and execution of behavior. For example, mirror neurons are thought to play important roles not only for generating movement but also for a wide range of activities essential for social interaction including self-awareness, empathy and language.^{46, 47} Recent work with mirror neurons suggest that social and motor behavior may be linked not only at the neural-system levels but also at the level of single neurons.^{46,} 47 Moreover, mirror neurons discharge not only when a particular motor act is being performed but also when we observe the same movement being done by others. Although the functional and structural links between social and motor behavior do not explain how higher levels of social activity que related to motor decline, it is noteworthy that physical activity in humans is thought to contribute to improved motor function by increasing neuronal plasticity and protecting against ischemic or neurotoxic damage.⁴⁸⁻⁵⁰ Animal studies suggest that physical activity may be associated with improved function through changes in brain plasticity.¹⁷

Our study has some limitations. Most importantly, inferences regarding causality must be drawn with great caution from observational studies. While the findings were robust to potential confounding variables and sensitivity analyses, the potential for reverse causality cannot be excluded. Further, it is possible that residual confounding from an unmeasured latent variable is related to both social activity and motor decline. Other limitations include the selected nature of the cohort, the self-report chronic diseases, in addition to self-report social, physical and cognitive activities. The combination of diaries and devices which provide quantitative measures of activity such as actigraphy would provide more accurate information about the duration of activity and energy expenditure. Death as informative censoring is also problematic in studies of aging.

However, several factors increase confidence in our findings. Perhaps most importantly, the study enjoys high follow-up participation reducing bias due to attrition. In addition, social activity was assessed among persons without dementia based on a detailed clinical evaluation and motor function was evaluated as part of a uniform clinical evaluation and incorporated many widely accepted and reliable strength and motor performance measures; strength testing was done in all four extremities, and motor performances were tested in both the arms and legs. The aggregation of multiple measures of motor function into a composite measure yields a more stable measure of motor function and increases statistical power to identify associations. In addition, a relatively large number of older persons representative of the general population

were studied, so that there was adequate statistical power to identify the associations of interest while controlling for several potentially confounding demographic variables.

Decline in motor function is a common condition with adverse health outcomes including death, disability, and the development of other conditions. Thus, it is increasingly being recognized as a major public health problem. Yet little is known about risk factors for motor decline which could translate into potential public health or clinical interventions. These data raise the possibility that social engagement can slow motor decline and possibly delay adverse health outcomes from such decline. Further work is needed to ensure that this is a causal relationship. First, the findings will need replication in other cohorts. Second, intervention studies may be needed. In fact, demonstration projects are already underway that may inform on the potential value of interventions. For example, in a novel translational study, a randomized trial of participation in Experience Corps is underway in Baltimore. 51 Participants are randomized to volunteering in elementary schools which serves as a rich source of cognitive, physical, and social engagement, vs. being on a wait list. Second, preclinical animal studies potentially could be used to determine whether different types of activities work through a common biologic mechanism. Finally, additional knowledge of the biology, in particular the neurobiology, of motor decline is needed. In this study, we excluded clinical stroke and Parkinson's disease. However subclinical manifestations of these or other conditions, in addition to non-neurologic conditions are responsible for motor decline. Very little is known about biology of motor decline. Such information would allow for much more refined hypotheses regarding the mechanisms underlying the association which will be important for the design and execution of potential interventions.

Acknowledgments

We thank all the participants in the Rush Memory and Aging Project. We also thank staff employed at the Rush Alzheimer's Disease Center including Traci Colvin, RN and Tracey Nowakowski, BA for project coordination; Barbara Eubeler, Mary Futrell, Karen Lowe Graham, MA and Pamela Smith, MA for participant recruitment; Wenqing Fan, MS and Liping Gu, MS for statistical programming; John Gibbons, MS and Greg Klein, BS for data management.

Aron S. Buchman, MD had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References

- 1. Baumgartner RN, Koehler KM, Gallagher D, et al. Epidemiology of sarcopenia among the elderly in New Mexico. Am J Epidemiol Apr 15;1998 147(8):755–763. [PubMed: 9554417]
- 2. Fried LP, Tangen CM, Walston J, et al. Frailty in older adults: evidence for a phenotype. J Gerontol A Biol Sci Med Sci Mar;2001 56(3):M146–156. [PubMed: 11253156]
- 3. Louis ED, Schupf N, Manly J, Marder K, Tang MX, Mayeux R. Association between mild parkinsonian signs and mild cognitive impairment in a community. Neurology Apr 12;2005 64(7):1157–1161. [PubMed: 15824340]
- 4. Bennett DA, Beckett LA, Murray AM, et al. Prevalence of parkinsonian signs and associated mortality in a community populaiton of older people. New England Journal of Medicine 1996;334(2):71–76. [PubMed: 8531961]
- 5. Buchman AS, Wilson RS, Bienias JL, Bennett DA. Change in frailty and risk of death in older persons. Exp Aging Res Jan-Mar;2009 35(1):61–82. [PubMed: 19173102]
- 6. Delmonico MJ, Harris TB, Lee JS, et al. Alternative Definitions of Sarcopenia, Lower Extremity Performance, and Functional Impairment with Aging in Older Men and Women. Journal of the American Geriatrics Society 2007;55(5):769–774. [PubMed: 17493199]05/121
- 7. Louis ED, Schupf N, Marder K, Tang MX. Functional correlates of mild parkinsonian signs in the community-dwelling elderly: poor balance and inability to ambulate independently. Mov Disord Mar; 2006 21(3):411–416. [PubMed: 16211605]
- 8. Louis ED, Tang MX, Schupf N, Mayeux R. Functional correlates and prevalence of mild parkinsonian signs in a community population of older people. Arch Neurol Feb;2005 62(2):297–302. [PubMed: 15710859]
- 9. Buchman AS, Boyle PA, Wilson RS, Tang Y, Bennett DA. Frailty is Associated With Incident Alzheimer's Disease and Cognitive Decline in the Elderly. Psychosomatic Medicine 2007;69(5):483– 489. [PubMed: 17556640]6/1
- 10. Visser M, Pluijm SM, Stel VS, Bosscher RJ, Deeg DJ. Physical activity as a determinant of change in mobility performance: the Longitudinal Aging Study Amsterdam. J Am Geriatr Soc Nov;2002 50 (11):1774–1781. [PubMed: 12410894]
- 11. Brach JS, FitzGerald S, Newman AB, et al. Physical activity and functional status in communitydwelling older women: a 14-year prospective study. Arch Intern Med Nov 24;2003 163(21):2565– 2571. [PubMed: 14638556]
- 12. Buchman AS, Boyle PA, Wilson RS, Bienias JL, Bennett DA. Physical Activity and Motor Decline in Older Persons. Muscle Nerve 2007;35:354–362. [PubMed: 17143881]
- 13. Warburton DE, Nicol CW, Bredin SS. Health benefits of physical activity: the evidence. Canadian Medical Association Journal Mar 14;2006 174(6):801–809. [PubMed: 16534088]
- 14. Agahi N, Parker MG. Leisure Activities and Mortality: Does Gender Matter? J Aging Health Oct; 2008 20(7):855–871. [PubMed: 18815413]
- 15. Fratiglioni L, Paillard-Borg S, Winblad B. An active and socially integrated lifestyle in late life might protect against dementia. The Lancet Neurology 2004;3(6):343–353.
- 16. Menec VH. The Relation Between Everyday Activities and Successful Aging: A 6-Year Longitudinal Study. Journals of Gerontology Series B: Psychological Sciences and Social Sciences 2003;58 (2):S74–82.
- 17. Hillman CH, Erickson KI, Kramer AF. Be smart, exercise your heart: exercise effects on brain and cognition. Nat Rev Neurosci 2008;9(1):58–65. [PubMed: 18094706]
- 18. Bennett DA, Schneider JA, Buchman AS, Mendes de Leon C, Bienias JL, Wilson RS. The Rush Memory and Aging Project: study design and baseline characteristics of the study cohort. Neuroepidemiology 2005;25(4):163–175. [PubMed: 16103727]
- 19. McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology Jul;1984 34(7):939– 944. [PubMed: 6610841]
- 20. Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 1993;24(1):35–41. [PubMed: 7678184]
- 21. Langston JW, Widner H, Goetz CG, et al. Core assessment program for intracerebral transplantations (CAPIT). Mov Disord 1992;7(1):2–13. [PubMed: 1557062]
- 22. Petersen RC, Thomas RG, Grundman M, et al. Vitamin E and donepezil for the treatment of mild cognitive impairment. N Engl J Med Jun 9;2005 352(23):2379–2388. [PubMed: 15829527]
- 23. Onder G, Penninx BW, Lapuerta P, et al. Change in physical performance over time in older women: the Women's Health and Aging Study. J Gerontol A Biol Sci Med Sci May;2002 57(5):M289–293. [PubMed: 11983722]
- 24. Buchman AS, Wilson RS, Boyle PA, Bienias JL, Bennett DA. Motor Function and Mortality in Older Persons. Journal of the American Geriatrics Society 2007;55:11–19. [PubMed: 17233680]
- 25. Mendes de Leon CF, Glass TA, Berkman LF. Social Engagement and Disability in a Community Population of Older Adults: The New Haven EPESE. American Journal of Epidemiology 2003;157 (7):633–642. [PubMed: 12672683]
- 26. Wilson RS, Krueger KR, Arnold SE, et al. Loneliness and Risk of Alzheimer Disease. Archives of General Psychiatry 2007;64(2):234–240. [PubMed: 17283291]
- 27. Wilson RS, Barnes LL, Krueger KR, Hoganson G, Bienias JL, Bennett DA. Early and late life cognitive activity and cognitive systems in old age. J Int Neuropsychol Soc Jul;2005 11(4):400–407. [PubMed: 16209420]
- 28. Katz S, Akpom CA. A measure of primary sociobiological functions. Int J Health Serv 1976;6(3): 493–508. [PubMed: 133997]

- 29. Rosow I, Breslau N. A Guttman health scale for the aged. J Gerontol Oct;1966 21(4):556–559. [PubMed: 5918309]
- 30. Lawton MP, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. Gerontologist Autumn;1969 9(3):179–186. [PubMed: 5349366]
- 31. Kohout FJ, Berkman LF, Evans DA, Cornoni-Huntley J. Two shorter forms of the CES-D (Center for Epidemiological Studies Depression) depression symptoms index. J Aging Health 1993;5(2): 179–193. [PubMed: 10125443]
- 32. Wilson RS, Schneider JA, Boyle PA, Arnold SE, Tang Y, Bennett DA. Chronic distress and incidence of mild cognitive impairment. Neurology 2007;68(24):2085–2092. [PubMed: 17562829]
- 33. Boyle PA, Wilson RS, Aggarwal NT, et al. Parkinsonian signs in subjects with mild cognitive impairment. Neurology Dec 27;2005 65(12):1901–1906. [PubMed: 16380610]
- 34. Zeger SL, Liang KY, Albert PS. Models for longitudinal data: a generalized estimating equation approach. Biometrics Dec;1988 44(4):1049–1060. [PubMed: 3233245]
- 35. Fitzmaurice, G.; Laird, NM.; Ware, JH., editors. Applied Longitudinal Analysis. New Jersey: John Wiley & Sons; 2004.
- 36. SAS/STAT® Software for Unix, Version (9.18) [computer program]. Version. Cary, NC: SAS Institute Inc.; 20022003.
- 37. Bassuk SS, Glass TA, Berkman LF. Social Disengagement and Incident Cognitive Decline in Community-Dwelling Elderly Persons. Ann Intern Med 1999;131(3):165–173. [PubMed: 10428732]
- 38. Wang HX, Karp A, Winblad B, Fratiglioni L. Late-Life Engagement in Social and Leisure Activities Is Associated with a Decreased Risk of Dementia: A Longitudinal Study from the Kungsholmen Project. American Journal of Epidemiology 2002;155(12):1081–1087. [PubMed: 12048221]
- 39. Wilson RS, Mendes De Leon CF, Barnes LL, et al. Participation in cognitively stimulating activities and risk of incident Alzheimer disease. Journal American Medical Association Feb 13;2002 287(6): 742–748.
- 40. Barnes BJ, Tuong CM, Mellen NM. Functional Imaging Reveals Respiratory Network Activity During Hypoxic and Opioid Challenge in the Neonate Rat Tilted Sagittal Slab Preparation. Journal of Neurophysiology 2007;97(3):2283–2292. [PubMed: 17215506]3/1
- 41. Jacobs JM, Hammerman-Rozenberg R, Cohen A, Stessman J. Reading Daily Predicts Reduced Mortality Among Men From a Cohort of Community-Dwelling 70-Year-Olds. Journals of Gerontology Series B: Psychological Sciences and Social Sciences 2008;63(2):S73–80.
- 42. Everard KM, Lach HW, Fisher EB, Baum MC. Relationship of Activity and Social Support to the Functional Health of Older Adults. Journals of Gerontology Series B: Psychological Sciences and Social Sciences 2000;55(4):S208–212.
- 43. Lehericy S, Bardinet E, Tremblay L, et al. Motor control in basal ganglia circuits using fMRI and brain atlas approaches. Cerebral Cortex 2006;16(2):149–161. [PubMed: 15858164]
- 44. Rizzolatti, G.; Sinigaglia, C. Mirrors in the Brain: How Our Minds Share Actions, Emotions, and Experience. New York: Oxford University Press; 2008.
- 45. Adolphs R. The Social Brain: Neural Basis of Social Knowledge. Annual Review of Psychology 2009;60(1):693–716.
- 46. Rizzolatti G, Fabbri-Destro M. The mirror system and its role in social cognition. Current Opinion in Neurobiology 2008;18(2):179–184. [PubMed: 18706501]
- 47. Iacoboni M. Imitation, Empathy, and Mirror Neurons. Annual Review of Psychology 2009;60(1): 653–670.
- 48. Kramer AF, Erickson KI, Colcombe SJ. Exercise, cognition, and the aging brain. J Appl Physiol Oct; 2006 101(4):1237–1242. [PubMed: 16778001]
- 49. Vaynman S, Gomez-Pinilla F. License to run: exercise impacts functional plasticity in the intact and injured central nervous system by using neurotrophins. Neurorehabil Neural Repair Dec;2005 19(4): 283–295. [PubMed: 16263961]
- 50. Dishman RK, Berthoud HR, Booth FW, et al. Neurobiology of exercise. Obesity Mar;2006 14(3): 345–356. [PubMed: 16648603]

51. Fried LP, Carlson MC, Freedman M, et al. A social model for health promotion for an aging population: Initial evidence on the Experience Corps model. Journal Urban Health 2004;81(1):64– 78.

*** The composite global motor measure was constructed by converting the raw score from each of the 18 motor measures to z scores using the mean and standard deviation from all participants at baseline. The values in the tables are approximations to the underlying measures that correspond to average performance at baseline, a "0" z score and 1 SD better than average, a z score of "1".

*** Pearson correlation coefficients

ˆ p<0.01;

§ p<0.001

Table 3 Demographics of the Cohort at Baseline***

+ =p<0.05,

ˆ =p<0.01,

§ =p<0.001

*** The cohort was divided based on the median baseline social activity score. Above the median value is "High Activity" and below the median value was "Low Activity". Mean (SD, range), except when the standard deviation exceeded the mean; in that case, summary statistics are the median, 25th and 75th quartiles. For variables with a value of 0 for the median, the percentage of the non-zero participants is provided, along with the 25th and 75th percentiles of the distribution of the non-zero values (Disability, Depressive Symptoms, Vascular Risk Factors and Diseases).

MMSE: Mini-Mental State Examination (range: 18-30), a higher score indicates a higher level of cognition. Social Activity: Self-reported frequency of participation in six social activities a higher score indicates more frequent participation. **Physical Activity**: Self-reported frequency of participation in 5 physical activities (hours/week), a higher score indicates more frequent participation. **Cognitive Activity**: Self reported frequency of participation in 7 cognitive activities, a higher score indicates more frequent participation. **Katz Disability**: 6 item measure of basic activities of daily living, a higher score indicates greater disability. **Rosow-Breslau Disability**: 3 item measure of mobility disability, a higher score indicates greater disability. **IADL Disability**: 8 item measure of instrumental activities of daily living, a higher score indicates greater disability. **Global Cognition**: Composite measure of cognition based on performances on 19 cognitive tests, a higher score indicates a higher level of cognition. **Depressive Symptoms**: Modified 10 item CESD scale, a higher score indicates greater depressive symptomatology. **BMI**: Body mass index: weight in kilograms divided by height in meters squared. **Vascular Risk Factors**: sum of smoking, diabetes, and hypertension self-reported. **Vascular Diseases**: sum of myocardial infarction, congestive heart failure, claudication and stroke self-reported. **Joint Pain**: single self-report item indicating whether or not joint pain is present.

*** Derived from generalized estimating equation which show the results for a 1-unit change in the social activity scale at baseline. Units of comparison: Time in years since baseline,; Age and education in years.

Table 5 Late-Life Social Activity, Other Covariates and the Rate of Change in Motor Function ***

 $=\approx 0.01$

§=p<0.001

*** **Estimate (95% Confidence Interval)** for each of the terms for the different covariates and their interaction with Time added to the core model shown in table 4. Derived from generalized estimating equations each model also included terms for Lag, Age, Sex, Education and their interaction with Time.