

Linking Perceived Control, Physical Activity, and Biological Health to Memory Change

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Perceived control plays an important role for remaining cognitively fit across adulthood and old age. However, much less is known about the role of perceived control over and above common correlates of cognition, and possible factors that underlie such control–cognition associations. Our study examined whether perceived control was predictive of individual differences in subsequent 4-year changes in episodic memory, and explored the mediating role of physical activity and indicators of physical fitness, cardiovascular, and metabolic health for control–memory associations. To do so, we used longitudinal data from the nationwide Health and Retirement Study (HRS; $N = 4,177$; ages 30 to 97 years; 59% women). Our results show that perceiving more control over one’s life predicted less memory declines, and this protective effect was similar in midlife and old age. We additionally observed that higher levels and maintenance of physical activity over 2 years, better pulmonary function, lower systolic blood pressure (SPB), lower hemoglobin A_{1c}, and higher high-density lipoprotein cholesterol (HDL–C) also predicted less memory declines. Mediation analyses revealed that levels of, and 2-year changes in, physical activity, as well as levels of pulmonary function and hemoglobin A_{1c} and HDL–C, each uniquely mediated control–memory change associations. Our findings illustrate that perceived control, physical activity, and indicators of physical fitness and cardiovascular and metabolic health moderate changes in memory, and add to the literature on antecedents of cognitive aging by conjointly targeting perceived control and some of its mediating factors. We discuss possible pathways underlying the role of control for memory change and consider future routes of inquiry to further our understanding of control–cognition associations in adulthood and old age.

Keywords: sense of control, cognitive aging, health and retirement study, adulthood and old age, behavioral and biological health

Perceived control is a general-purpose belief system associated with cognitive, mental, and physical health across adulthood and old age (Bandura, 1997; Lachman, 2006; Rodin, 1986; Skaff, 2007; Uchino, 2006). For example, perceived control has repeatedly been found to be associated with shallower rates of decline in cognitive abilities, including memory, processing speed, and executive functioning among middle-aged and older adults (Agrigoroaei & Lachman, 2011; Bielak et al., 2007; Caplan & Schooler, 2003; Lachman, 1986; Windsor & Anstey, 2008). However, much less is known regarding the role of perceived control over and above common correlates of cognition, such as behavioral and biological health, and factors underlying control–cognition associations. Conceptual models of control outline that physical activ-

ity and biological health factors may underlie why perceived control is protective against cognitive declines (Lachman, Neupert, & Agrigoroaei, 2011; Rodin, 1986; Uchino, 2006). For example, perceiving control relates to engaging in health-promoting behaviors and exhibiting better biological health profiles (Infurna & Gerstorf, in press; Lachman & Firth, 2004; White, Wójcicki, & McAuley, 2012), which have downstream effects on cognitive functioning. Our aim in this study is to use data from the nationwide Health and Retirement Study (HRS) to empirically test conceptual models of control by conjointly examining behavior (levels of and 2-year change in physical activity) and biological health (physical fitness, cardiovascular, and metabolic systems) factors that are linked to cognition and possibly underlie control–cognition associations. We will additionally examine whether control–cognition associations and the mediators of such associations differ by chronological age.

Associations Between Perceived Control and Cognition

Perceptions of control refer to individuals’ beliefs about their capability to exert influence over and shape one’s life circumstances (Pearlin & Schooler, 1978; Skinner, 1996). Cognitive functioning can be broadly viewed as people’s capability to learn, comprehend, make decisions, and adapt to changes in the face of obstacles, and is often considered a key outcome of successful aging (Baltes, Lindenberger, & Staudinger, 2006; Rowe & Kahn, 1987). Previous studies have shown that perceived control is

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The Health and Retirement Study (HRS) and HRS 2006 Biomarker data was supported by a cooperative agreement (Grant U01 AG09740) between the NIA and the University of Michigan. The content is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies.

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associated with cognitive functioning at one point in time and protective of declines over time. For example, cross-sectional results focusing on midlife in a U.S. study showed that individuals who reported higher levels of control were more likely to exhibit better memory, executive functioning, and processing speed (Agrigoroaei & Lachman, 2011). Longitudinal evidence has corroborated these cross-sectional findings, suggesting that perceiving more control over one's life circumstances is related to subsequently experiencing shallower cognitive declines. For example, longitudinal findings from the PATH Through Life Project revealed that perceiving control over one's life was linked to more positive 4-year changes in memory, processing speed and verbal intelligence in cohorts of persons in young adulthood, midlife, and old age (Windsor & Anstey, 2008). Over a 20-year follow-up, Caplan and Schooler (2003) observed that people in young adulthood and midlife who reported fewer feelings of fatalism were more likely to report fewer cognitive difficulties in midlife and old age.

Behavioral and Biological Health Correlates of Cognition

Factors that represent behavioral and biological health are associated with cognition in adulthood and old age. The behavior factor targeted in this study is *physical activity*, which refers to one's involvement with, and engagement in, vigorous (e.g., running or gym workout), moderate (e.g., gardening or walking), and mild (e.g., vacuuming) activities (Levine, Eberhardt, & Jensen, 1999; McAuley, 1993). Accumulated empirical evidence shows that individuals who are physically active are more likely to perform better on tests of cognitive functioning and report better physical health (Anstey & Christensen, 2000; Bielak, Christensen, & Windsor, 2012; Penedo & Dahn, 2005; Small, Dixon, McArdle, & Grimm, 2012; Yaffe et al., 2009). These findings map onto the vascular hypothesis of cognitive aging (Bielak, 2010; Casserly & Topol, 2004; de la Torre, 2004; Fratiglioni, Paillard-Borg, & Winblad, 2004; Spiro & Brady, 2008), which suggests that physical activity affects cognitive functioning through increasing cerebral blood flow and maintaining the structure and function of the brain via keeping neural processing and synaptic organization efficient, adaptive, and plastic (Colcombe et al., 2004; Colcombe & Kramer, 2003; Kramer, Bherer, Colcombe, Dong, & Greenough, 2004; Stern, 2002).

Biological systems that we target in our report include physical fitness and cardiovascular and metabolic health. *Physical fitness* represents one's state of general muscle strength, musculoskeletal capacity, subclinical disease, and general vitality (Anstey, 2012; Anstey, Luszcz, Giles, & Andrews, 2001; Rantanen et al., 1999), and is, among other things, often indexed with grip strength and forced expiratory volume. Limitations in physical fitness represent typical proxies for the presence of chronic disease, lack of resistance to external stressors, and cumulative biological burdens (MacDonald, DeCarlo, & Dixon, 2011; McEwen, 1998; Rantanen et al., 2003; Wahlin, MacDonald, de Frias, Nilsson, & Dixon, 2006). Reduced forced expiratory volume is indicative of weakened pulmonary functioning, which decreases blood flow to the brain, leads to brain atrophy, and increases white matter hyperintensities, which, in turn, undermines cognitive abilities (MacDonald et al., 2011; Sachdev et al., 2006). Grip strength refers to one's

musculoskeletal capacity and is an indicator of one's physical fitness, and the absence or presence of constraining health conditions, which can also compromise cognitive functioning (Anstey, 2012).

Cardiovascular and metabolic systems represent a constellation of interrelated factors that characterize one's overall vascular and metabolic functioning across various organs, including the heart, kidney, and liver (Grundy et al., 2005). Poor management of one's cardiovascular and metabolic health is associated with increased risk for declines in cognitive and physical functioning, disease incidence, and mortality (Anstey & Christensen, 2000; Barter & Rye, 1996; Després et al., 2008; Khaw & Wareham, 2006; Kumari, Brunner, & Fuhrer, 2000; Verhaegen, Borchelt, & Smith, 2003). For example, elevated levels of systolic blood pressure (SBP) and pulse rate (PR) are linked to an increased likelihood of exhibiting poorer levels and steeper rates of decline in cognitive functioning (Bender & Raz, 2012; Brady, Spiro, & Graziano, 2005; Qiu, Winblad, & Fratiglioni, 2005; Thorvaldsson et al., 2012). Elevated blood glucose is associated with oxidative stress, hardening of arterial walls, and advanced glycation end-product level, which contributes to accelerated cognitive decline in old age (Yaffe et al., 2011). Furthermore, low levels of high-density lipoprotein cholesterol (HDL-C) can lead to atherosclerosis or the hardening of arterial walls and plaque accumulation, which obstructs or decreases blood flow and results in deficits in overall cognitive functioning (Stampfer, 2006). Taken together, factors that represent behavioral functioning and biological health are associated with cognition in adulthood and old age, and our goal is to examine whether they show unique predictive effects for changes in memory when examined in a conjoint manner.

Behavioral and Biological Health Mediators of Control–Cognition Associations

Lachman's integrative model of perceived control (2006; Lachman et al., 2011; Soederberg Miller & Lachman, 1999) outlines that behavioral, motivational, affective, and biological health factors are possible mediators that link control beliefs to aging-related outcomes such as cognitive functioning. For example, people who report stronger control beliefs are more likely to have higher motivation to develop and use effective strategies to maintain cognitive functioning (Hertzog, McGuire, & Lineweaver, 1998; Lachman et al., 2011). Consistent with the model, empirical evidence suggests that more perceived control is directly associated with engagement in physical activity and better physical fitness and with lower cardiometabolic risk (Infurna & Gerstorf, *in press*; Roepke & Grant, 2011; White et al., 2012), which have downstream effects on cognition (Lachman & Firth, 2004; Spiro & Brady, 2011; Stampfer, 2006). Up to this point, however, empirical studies have typically only focused on one of these underlying factors and primarily used data obtained at one point in time. The next steps to provide a more complete picture of control–cognition associations is to simultaneously test these underlying factors within the context of longitudinally linking control to cognition. Conjointly examining physical activity and biological health factors that underlie such associations will provide researchers with initial insights into possible pathways of how perceived control is associated with cognitive functioning. Furthermore, examining whether change in a mediator such as physical activity affects

control–cognition associations promises to shed light onto how mediation processes unfold over time, and will put us in a better position to disentangle the unique effects that level of activity and change in activity have. For example, persons who report higher levels of perceived control are more likely to view their health as controllable, resulting in acknowledging the importance of and consistently participating in physical activity over time rather than only sporadically, which could result in additional protective effects on maintaining memory. Ultimately, this route of inquiry will further our knowledge of systems to target in tailored prevention and intervention programs so as to protect against declines in cognitive functioning (for discussion, see [Spiro & Brady, 2011](#)).

More specifically, in an additional exploratory step, we examine whether behavioral and biological health factors may underlie associations between perceived control and 4-year changes in memory. First, sense of control is important in the context of physical activity because people who report higher levels of control are more likely to view their health as controllable, thereby partaking in the necessary health-promoting behaviors such as strenuous exercise ([Bandura, 2004](#); [Lachman et al., 1997](#); [White et al., 2012](#)). Second, focusing on the role of physical fitness, empirical evidence suggests that people who perceive more control over their life were found to be in better physical health ([Gerstorf, Röcke, & Lachman, 2011](#); [Infurna, Gerstorf, & Zarit, 2011](#)). Third, perceived control is associated with better cardiovascular and metabolic functioning because people high in perceived control are more likely to initiate and persist with health-promoting behaviors, and to implement the available motivational and coping strategies needed to downregulate negative emotional states ([Diehl & Hay, 2010](#); [Hay & Diehl, 2010](#); [Lachman, 2006](#)). For example, perceived control is a psychological resource individuals can draw upon to help downregulate sustained periods of negative emotional states, such as depressive symptoms and negative affect, which can lead to deficiencies in one's cardiovascular and metabolic functioning ([Danner, Snowdon, & Friesen, 2001](#); [Rodin, 1986](#); [Seligman, 1975](#)).

The Role of Age in Moderating Control–Cognition Associations

Theoretical accounts and conceptual models of control have long discussed that associations between control and aging-related outcomes (e.g., cognitive functioning and health) may be stronger in old age compared with young adulthood and midlife (see [Lachman, 2006](#); [Rodin, 1986](#)). It is possible that health-related activities prompted by control beliefs may have a larger impact later in life when the overall functional and health system has become more vulnerable and fragile ([Rodin, 1986](#)). This falls in line with previous research examining control–health associations showing that perceived control is protective against health declines in older ages but not in younger ages ([Caplan & Schooler, 2003](#); [Infurna et al., 2011](#); [Lachman & Agrigoroaei, 2010](#)). However, perceived control can be expected to be equally likely to have health implications in younger ages due to the importance of fostering one's perceptions of control to set people on a more positive health trajectory ([Infurna, Ram, & Gerstorf, in press](#)). The empirical evidence thus far is very inconclusive about the direction in which control–cognition associations differ across adulthood and old age. Recent research focusing on midlife in a U.S. study

did not find evidence to suggest that associations of perceived control with reasoning, executive functioning, and episodic memory were stronger in older ages ([Agrigoroaei & Lachman, 2011](#)). In contrast, [Caplan and Schooler \(2003\)](#) found that associations between fatalism and cognitive difficulties were stronger in midlife compared with old age. Finally, research from the PATH Through Life Project suggests that associations between perceived control and processing speed were stronger in the older age group ([Windsor & Anstey, 2008](#)).

In the current study, we will also examine whether underlying factors (i.e., behavior and biological health) of control–cognition associations differ between midlife and old age. One line of reasoning would suggest that associations between perceived control and mediators of cognitive change may be stronger in younger ages because the effects of mediators may take longer to develop and accumulate ([Infurna et al., 2011, in press](#)). In older ages, losses in the health domain may have accumulated over time, become more salient, and thereby exert their effects more proximally on cognition. A second line of reasoning argues that possible pathways that link perceived control to cognition may differ across adulthood and old age. In old age, strategy use may be a possible pathway linking perceived control to cognition. For example, in an adult-life-span sample, [Lachman and Andreoletti \(2006\)](#) found that older adults with higher levels of control were more likely to utilize strategies such as word clustering for recall performance. In sum, our goal here is to disentangle the age-differential nature of control–cognition associations.

The Present Study

Our objective in this study is to extend insights into psychological, behavioral, and biological health factors that are linked to change in memory, and possible factors that underlie control–memory associations in adulthood and old age. Although research has long shown that psychological, behavioral, and biological health factors are associated with better cognitive functioning, less is known about their conjoint effects and factors that underlie control–cognition associations. We use data from the HRS, which involves a nationally representative population of middle-aged and older adults and has obtained data on multiple behavior and biological health factors that allows for conjointly examining factors conceptualized to be linked to changes in cognition. In a first step, we examine whether perceived control is associated with 4-year memory change. We hypothesize that more perceived control is associated with less-steep memory declines. In a second step, we examine whether behavior (levels of and 2-year change in physical activity) and biological health (physical fitness: grip strength, forced expiratory volume; cardiovascular: SBP, pulse rate (PR); and metabolic: hemoglobin A_{1c}, HDL–C) factors predict 4-year memory change. In an additional step, we examine whether behavior and biological health factors underlie control–memory change associations. We hypothesize that the level of and change in physical activity and factors representing each of the biological health factors included will mediate control–memory associations. We note that our presented mediation analyses should be considered as an exploratory step, given the present design, because of nonrandom assignment and cross-sectional data on the mediators of interest. Mediation analyses stem from prevention and intervention research, in which participants are typically randomized into

a treatment and control group to ensure similarities in the groups prior to the initiation of the treatment exposure (for discussion, see MacKinnon, Fairchild, & Fritz, 2007). In our study, the treatment or independent predictor (perceived control) was not randomized, with likely confounds that we did not account for that share common associations with behavioral and biological health factors and cognition. An example of this approach in the control literature comes from Reich and Zautra (1990), who randomized participants into a control-enhancing intervention or not, and found that participants in the intervention group experienced increases in internal beliefs of control over time and that this was associated with improvements in mental health. We consider conditions for longitudinal mediation in the Discussion section, including possible scenarios for longitudinal mediation and bidirectional associations among the constructs of interest. Third, we will explore whether control-memory change associations and the mediators underlying such associations differ by chronological age. It is largely an open question as to whether, and in which direction, an age-differential pattern will emerge.

Method

Participants and Procedure

The HRS is a nationally representative probability sample of households in the contiguous United States of noninstitutionalized adults aged 50 years and older (McArdle, Fisher, & Kadlec, 2007; Soldo, Hurd, Rodgers, & Wallace, 1997). The measures assessed cover a wide range of economic, sociological, psychological, mental, and physical health information. Over the 20-year history of the HRS, over 30,000 participants have been assessed. In 2006, the HRS introduced a new component to the study design—an enhanced face-to-face interview for half of the eligible households, where participants were given a leave-behind psychosocial questionnaire (for details, see Clarke, Fisher, House, Smith, & Weir, 2008) and the opportunity to provide physical and biomarker

measurements (for details, see Crimmins et al., 2008, 2009). In 2006, 22,422 participants were eligible to participate in HRS, and from this sample, half of the households were randomly selected to partake in the enhanced face-to-face interview: 9,570 individuals qualified for the enhanced face-to-face interview and 8,567 provided data for either the psychosocial questionnaire or physical and biomarker measurements. Our reduced sample is largely driven by missing data on some of the biomarker measurements measures (e.g., HDL-C, $n = 5,041$; hemoglobin A_{1c}, $n = 6,507$).

In the present study, we used data from 4,177 participants who (a) participated in the 2006 enhanced face-to-face interview, and (b) provided data on all of our measures of interest. Table 1 shows descriptive statistics for the measures of interest. Participants were, on average, 67 years of age ($SD = 10.43$) and attained 13 years of education ($SD = 3.01$); 59% were women, 86% were White, 11% were African American, 3% were other, and 71% were married or partnered. Relative to those who participated in the enhanced face-to-face interview, but were not included in our analyses because of missing data ($n = 4,420$), our participants reported more control ($M = 4.82$, $SD = 0.93$ vs. $M = 4.67$, $SD = 1.01$; $F[1, 7,622] = 43.90$, $p < .05$), performed better on the episodic memory test ($M = 10.05$, $SD = 3.33$ vs. $M = 9.28$, $SD = 3.52$; $F[1, 8,326] = 106.73$, $p < .05$), were younger ($M = 66.75$, $SD = 10.43$ vs. $M = 68.12$, $SD = 11.16$; $F[1, 8,595] = 34.95$, $p < .05$), were more highly educated ($M = 12.77$, $SD = 3.01$ vs. $M = 12.25$, $SD = 3.32$; $F[1, 8,582] = 57.15$, $p < .05$), were more likely to be White (86% vs. 79%; $\chi^2[1, 8,595] = 72.94$, $p < .05$), were married (71% vs. 64%; $\chi^2[1, 8,595] = 46.87$, $p < .05$), were physically active ($M = 2.97$, $SD = 0.88$ vs. $M = 2.71$, $SD = 0.98$; $F[1, 8,594] = 164.73$, $p < .05$), were higher in grip strength ($M = 31.22$, $SD = 10.80$ vs. $M = 30.55$, $SD = 11.47$; $F[1, 7,403] = 6.57$, $p < .05$), were higher in forced expiratory volume ($M = 343.16$, $SD = 127.23$ vs. $M = 318.18$, $SD = 132.25$; $F[1, 7,426] = 66.95$, $p < .05$), and exhibited lower hemoglobin A_{1c} ($M = 5.80$, $SD = 0.82$ vs. $M = 5.90$, $SD = 0.94$; $F[1, 6,505] = 20.35$, $p < .05$), but did not differ in gender, HDL-C, SBP, PR, and waist circumference

Table 1
Means, Standard Deviations, and Intercorrelations for Measures Used in the Present Study

| Construct | <i>M</i> | <i>SD</i> | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|--|----------|-----------|-------|------|------|------|------|------|------|------|------|-------|------|------|------|
| 1. Perceived control | 4.82 | 0.93 | — | | | | | | | | | | | | |
| 2. Age | 66.75 | 10.43 | -.12 | — | | | | | | | | | | | |
| 3. Gender (1 = women) | 0.59 | 0.49 | -.04 | -.05 | — | | | | | | | | | | |
| 4. Education | 12.77 | 3.01 | .20 | -.15 | -.05 | — | | | | | | | | | |
| 5. Physical activity: 2006 | 2.97 | 0.88 | .23 | -.17 | -.06 | .21 | — | | | | | | | | |
| 6. Physical activity: 2008 | 2.90 | 0.92 | .22 | -.22 | -.02 | .21 | .56 | — | | | | | | | |
| 7. Grip strength | 31.22 | 10.80 | .15 | -.30 | -.72 | .14 | .19 | .17 | — | | | | | | |
| 8. Forced expiratory volume | 343.16 | 127.23 | .21 | -.31 | -.52 | .26 | .24 | .24 | .64 | — | | | | | |
| 9. Systolic blood pressure | 131.43 | 20.39 | -.002 | .23 | -.11 | -.13 | -.06 | -.08 | .06 | -.04 | — | | | | |
| 10. Pulse rate | 70.43 | 11.16 | -.05 | -.14 | .07 | -.05 | -.05 | -.05 | -.03 | -.08 | -.10 | — | | | |
| 11. Hemoglobin A _{1c} | 5.80 | 0.82 | -.08 | .09 | .005 | -.12 | -.13 | -.15 | -.07 | -.09 | .12 | .10 | — | | |
| 12. High-density lipoprotein cholesterol | 57.41 | 14.38 | .07 | -.07 | .36 | .10 | .11 | .11 | -.24 | -.13 | -.07 | -.005 | -.12 | — | |
| 13. Waist circumference | 39.62 | 5.95 | -.04 | -.01 | -.26 | -.09 | -.19 | -.18 | .24 | .13 | .15 | .07 | .20 | -.34 | — |
| Outcome | | | | | | | | | | | | | | | |
| Memory: 2006 | 10.05 | 3.33 | .20 | -.37 | .13 | .35 | .20 | .22 | .10 | .22 | -.15 | -.005 | -.15 | .13 | -.08 |
| Memory: 2010 | 9.66 | 3.42 | .15 | -.40 | .15 | .34 | .15 | .20 | .05 | .18 | -.18 | .01 | -.14 | .15 | -.08 |

Note. $N = 4,177$. Grip strength was measured in kilograms (kg). Forced expiratory volume was measured in liters per minute (L/min). Systolic blood pressure was measured in mmHg. Pulse rate was measured in beats per minute. Hemoglobin A_{1c} was measured in %. High density lipoprotein cholesterol was measured in mg/dL. Waist circumference was measured in inches. Correlations above |.03| are significant at the $p < .05$ level.

(all $ps > .05$). Although the effect size of the differences found was relatively small ($\eta^2 < .01$ for all comparisons), we note that our sample may be positively select in that they reported more perceived control and were younger, more educated, and more physically and cognitively fit.

Measures

Outcome. The target outcome variable was episodic memory, which was indexed by a sum of performance scores on immediate and delayed free-recall tests. Detailed information can be found in Ofstedal, Fisher, and Herzog (2005) and Gerstorff, Hoppmann, Kadlec, and McArdle (2009). In brief, participants were presented a list of 10 nouns, and asked to recall as many words as possible both immediately after presentation and after a delay of approximately 5 min. Scores were calculated as the total sum (ranging from 0 to 20) of words correctly remembered in both tasks, with higher scores indicating better performance. We used episodic memory assessments from 2006 and 2010.

Perceived control. We used an average composite of 10 items that assessed one's feelings of control over one's life to index perceived control (see Clarke et al., 2008; Pearlin & Schooler, 1978). Participants were asked to indicate the extent to which they agree with each of the items using a 6-point scale (1 = *strongly disagree* to 6 = *strongly agree*; e.g., "I can do just about anything I really set my mind to"). Negatively valenced items were reverse coded, so that higher scores reflected perceiving more control (Cronbach's $\alpha = .87$). Perceived control was assessed in 2006.

Behavioral. Our behavioral factor was the level of and 2-year change in *physical activity*, which was comprised of three separate items assessing how often participants partake in vigorous, moderate, and mild activity. The specific item wording was, "We would like to know the type and amount of physical activity involved in your daily life"; "How often do you take part in sports or activities that are *vigorous*, such as running or jogging, swimming, cycling, aerobics, or gym workout, tennis, or digging with a space or shovel?"; "How often do you take part in sports or activities that are *moderately* energetic such as gardening, cleaning the car, walking at a moderate pace, dancing, floor or stretching exercises?"; "How often do you take part in sports or activities that are *mildly* energetic, such as vacuuming, laundry, and home repairs?" Participants rated each item using a 5-point scale (1 = *every day* to 5 = *hardly ever or never*). The items were reverse coded and averaged, with higher scores indicating more physical activity (Cronbach's $\alpha = .54$). We used measurements taken in 2006 and 2008; level represents physical activity in 2006, and change in physical activity was modeled in our latent change score model as 2-year change from 2006 to 2008 (details on latent change score models in Statistical Analyses section). Substantively similar findings were observed when we first weighted the three items (vigorous activity, .9; moderate activity, .5; and light activity, .3) and then averaged them.

Biological health. Physical fitness was measured using two separate indicators. Grip strength was measured using a Smedley spring-type hand dynamometer in kilograms (Anstey et al., 2001; Crimmins et al., 2008). The dynamometer was fit to the participant's hand, and the person was instructed to stand and squeeze the meter as hard as they were able to for a couple of seconds and then let go. Participants completed two measurements with each hand,

alternating hands, while standing, or if a participant was unable to stand, the measurement was completed with the participant seated. The participants' maximum score out of their total measurement trials was selected. Forced expiratory volume was measured in L/min using a Mini Wright Peak Flow Meter with a disposable mouthpiece. Respondents were instructed to stand up, take a deep breath, place their lips around the mouthpiece, and blow as hard and as fast as possible. The interviewer recorded the value indicated by the pointer and reset the meter. Up to three readings were obtained at 30-s intervals. The Mini Wright Peak Flow Meter does not indicate the quality of the trials, and therefore we do not have information on quality of trial. However, there is information as to reasons for not having a complete forced expiratory volume reading. Reasons for not completing forced expiratory volume included the following: (a) the participant felt it would not be safe ($n = 77$); (b) the interviewer felt it would not be safe ($n = 11$); (c) the participant refused or was not willing to complete the test ($n = 177$); (d) the participant tried but was unable to complete the test ($n = 9$); (e) the participant did not understand the instructions ($n = 4$); (f) there was a problem with equipment or supplies ($n = 44$); (g) the participant had surgery, injury, or other health condition that prevented participant from completing the measurement ($n = 13$); (h) an unsuitable location ($n = 14$); and (i) other reasons ($n = 10$). The participants' maximum score out of their total measurement trials was selected.

Cardiovascular functioning was quantified with participants' SBP and PR, which were measured using an Omron HEM-780 Intellisense Automatic blood pressure monitor with ComFit cuff (see Crimmins et al., 2008). Respondents were instructed to sit down, with both feet on the floor and their left arm comfortably supported with the palm facing up. The cuff was adjusted to the respondent's arm, ensuring that it made direct contact with the skin; the bottom of the cuff was approximately half an inch above the elbow, and the air tube ran down the middle of the respondent's arm. Three measurements were taken, 45 s apart, on the respondent's left arm. We used the average of three measurements for SBP and PR.

Metabolic functioning was represented by three separate indicators. Blood acquisition and determination was performed using instructions and kits from Biosafe Laboratories (Chicago, IL; for details, see Crimmins et al., 2009). Blood was taken by pricking the participant's finger with a sterile lancet after cleansing the finger with an alcohol swab and analyzed for concentrations of hemoglobin A_{1c} and HDL-C. Droplets of blood were directly placed on specially treated filter paper, within circles printed on the paper. The blood spots on filter paper were then placed in special foil envelopes with a desiccant packet, and then within mailing containers, and then shipped to Biosafe Labs. Repeated measures within a specific laboratory run showed a coefficient of variation of less than 3.5% for HDL-C and less than 7% between runs (Crimmins et al., 2009). During quality control studies, the correlation between finger prick and serum levels was 0.949 for HDL-C. Hemoglobin A_{1c} is a summary measure of blood glucose metabolism that covers the last 90 days. Waist circumference was measured with a tape measure at the level of the respondent's navel. Respondents were asked to stand up and remove any bulky clothing, and then the interviewer placed the tape measure around the waist at the level of their navel. The respondent was instructed to inhale and slowly exhale, and waist circumference was mea-

sured while holding the exhale (see Crimmins et al., 2008). Physical fitness and cardiovascular and metabolic functioning were assessed in 2006.

Statistical Analyses

Our research questions revolved around whether perceived control was associated with 4-year memory change and the role of physical activity and biological health factors in mediating such associations. Figure 1 illustrates a schematic representation of our model that shows when each of the factors was assessed. To address these questions, we used latent change score (LCS) models (see Ferrer & McArdle, 2010; McArdle, 2009; McArdle & Neselrode, 2003). In LCS models, the targeted outcome variable is the change score. The model for our first research question was specified as:

$$\begin{aligned} \text{Memorychange}_i = & \beta_0 + \beta_1(\text{Memory06}_i) + \beta_2(\text{Control06}_i) \\ & + \beta_3(\text{Age}_i) + \beta_4(\text{Gender}_i) + \beta_5(\text{Education}_i) \\ & + \beta_6(\text{Control06}_i \times \text{Age}_i) + e_i \end{aligned}$$

where $\text{Memorychange}_i = \text{Memory in 2010} - \text{Memory in 2006}$; β_0 is the sample-level average of 4-year memory change when all the predictors are zero; β_1 is the effect of memory in 2006 on memory change; β_2 is the effect of control in 2006 on memory change; $\beta_3 - \beta_5$ is the effect of age, gender, and education on memory change, respectively; β_6 is whether the effect of control on memory change differs across chronological age; and e_i are residual errors. In a subsequent model, we included physical activity and biological health factors to examine whether they moderated 4-year memory change.

For our second research question, we tested a mediation model in which behavior and biological health factors were regressed onto perceived control, and memory change was regressed onto

each mediator (MacKinnon et al., 2007). The model for research question two was specified as

$$\begin{aligned} \text{Memorychange}_i = & \beta_0 + \beta_1(\text{Memory06}_i) + \beta_2(\text{Control06}_i) \\ & + \beta_3(\text{Age}_i) + \beta_4(\text{Gender}_i) + \beta_5(\text{Education}_i) \\ & + \beta_6(\text{Control06}_i \times \text{Age}_i) + \beta_7(\text{Level physical activity}_i) \\ & + \dots + \beta_{15}(\text{Waist circumference}_i) + e_i \end{aligned}$$

where $\beta_7 - \beta_{15}$ is the direct effect of each of the mediators on memory change. The effect for control on each of the mediators was specified as

$$\text{Level physical activity}_i = \beta_0 + \beta_1(\text{Control06}_i) + e_i$$

...

$$\text{Waist circumference}_i = \beta_0 + \beta_1(\text{Control06}_i) + e_i$$

where the mediators (e.g., level physical activity to waist circumference) are the outcome, β_0 are the intercepts, β_1 is the effect of control on the specified mediator, and e_i are residual errors. A nonparametric resampling or bootstrapping procedure was applied using Mplus (Muthén & Muthén, 1998–2007) to test whether the indirect effect of perceived control through the hypothesized mediators (behavior and biological health factors) and memory change were reliably different from zero. To acknowledge the possible skew of the distribution of the indirect effect, our models did not impose normality assumptions (see Preacher & Hayes, 2008).

Results

Associations Between Perceived Control and Cognition

Table 2 shows results from our LCS models examining whether perceived control was associated with 4-year memory change. Results from Model 1 suggest that, on average, memory declined over the 4-year period ($\Delta = -0.82, p < .05$) and there were sizable between-person differences in change after controlling for memory in 2006 ($\sigma^2 = 6.59, p < .05$). We observed that perceived control predicted 4-year memory change ($\beta_2 = 0.15, p < .05$), suggesting that reporting higher levels of perceived control was associated with experiencing less steep declines in memory, independent of age, gender, and education. The perceived Control \times Age interaction was not reliably different from zero, suggesting that the protective effect of control on memory change did not differ across chronological age. Follow-up analyses also tested interaction effects of control beliefs with gender and education, but none of these terms was found to be reliably different from zero. Figure 2 illustrates that, compared with participants who reported lower levels of control ($-1 SD$; dotted line), participants who reported higher levels of perceived control ($+1 SD$; block line) showed, on average, less-steep memory declines. Based on the estimates from Model 1 in Table 2, the average person remembered 10.05 words in 2006, and individuals reporting $\pm 1 SD$ in perceived control remembered 10.73 and 9.37 words, respectively (difference between high and low control = 1.36 words in 2006). In 2010, the average person remembered 9.23 words, whereas individuals reporting $\pm 1 SD$ in perceived control in 2006 remembered 10.05 and 8.41 words, respectively (difference between high

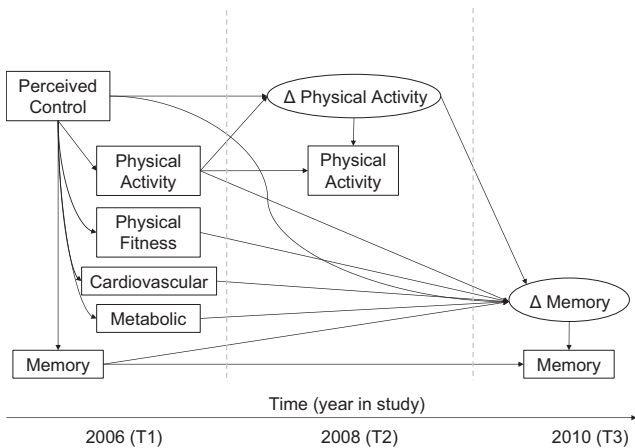


Figure 1. Overview of data collected in the Health and Retirement Study, as used in the present study and for our latent change score mediation analyses. We used perceived control data collected in 2006 to predict 4-year memory change (between 2006 and 2010), and whether behavior and biological health (physical fitness, cardiovascular, and metabolic) factors collected in 2006 and physical activity change (between 2006 and 2008) mediated such associations.

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Table 2
Latent Change Score Model of Perceived Control and Physical Activity and Biological Health Factors Predicting 4-Year Memory Change: Standardized and Unstandardized Regression Coefficients

| Variable | Model 1 | | Model 2 | |
|--|--------------------------|--------------|--------------------------|--------------|
| | Parameter estimates (SE) | Std. β | Parameter estimates (SE) | Std. β |
| Fixed effect | | | | |
| Change, β_0 | -0.82* (0.05) | | -0.81*(0.05) | |
| Predictor | | | | |
| Episodic memory in 2006, β_1 | -0.50* (0.02) | -.52 | -0.53* (0.02) | -.59 |
| Perceived control, β_2 | 0.15* (0.05) | .04 | 0.10* (0.05) | .03 |
| Age, β_3 | -0.08* (0.01) | -.26 | -0.07* (0.01) | -.24 |
| Women, β_4 | 0.59* (0.09) | .09 | 0.84* (0.14) | .14 |
| Education, β_5 | 0.18* (0.02) | .17 | 0.16* (0.02) | .16 |
| Perceived Control \times Age, β_6 | 0.001 (0.01) | .003 | 0.002 (0.01) | .01 |
| Correlates | | | | |
| Physical activity | | | | |
| Level of physical activity, β_7 | | | 0.17* (0.06) | .05 |
| 2-year change in physical activity, β_8 | | | 0.19* (0.06) | .05 |
| Physical fitness | | | | |
| Grip strength, β_9 | | | 0.001 (0.01) | .004 |
| Forced expiratory volume, β_{10} | | | 0.002* (0.000) | .07 |
| Cardiovascular | | | | |
| Systolic blood pressure, β_{11} | | | -0.004* (0.002) | -.03 |
| Pulse rate, β_{12} | | | -0.004 (0.004) | -.01 |
| Metabolic | | | | |
| Hemoglobin A _{1c} , β_{13} | | | -0.12* (0.05) | -.03 |
| High-density lipoprotein cholesterol, β_{14} | | | 0.01* (0.003) | .04 |
| Waist circumference, β_{15} | | | 0.02* (0.008) | .05 |
| Random effect | | | | |
| Residual variance, σ^2 | 6.59* (0.21) | | 6.50* (0.19) | |

Note. $N = 4,177$. $SE =$ standard error; std. $\beta =$ standardized beta.
 * $p < .05$.

and low control = 1.64 words in 2010, resulting in an effect size of $d = .49$ (1.64/ 3.33). Furthermore, results indicate that a 1-SD increase in perceived control relates to 1.75 fewer years of aging on 4-year memory change, in the absence of any change in perceived control and the included covariates.

Behavioral and Biological Health Correlates of Cognition

We next included physical activity and biological health factors as predictors to examine whether they moderated 4-year memory change, independent of sociodemographic factors and perceived control. Results are shown in Model 2 in Table 2. We observed that the effect of perceived control remained reliably different from zero ($\beta_2 = 0.10, p < .05$). We observed that higher levels of and maintenance of physical activity ($\beta_7 = 0.17, p < .05$; $\beta_8 = 0.19, p < .05$) were associated with less-steep declines in 4-year memory change. Because the physical activity construct consisted of three items, follow-up analyses included the three items separately, and we observed that the effect was largely driven by light and vigorous physical activity. Additionally, stronger forced expiratory volume ($\beta_{10} = 0.002, p < .05$), lower SBP ($\beta_{11} = -0.004, p < .05$), lower hemoglobin A_{1c} ($\beta_{13} = -0.12, p < .05$), higher HDL-C ($\beta_{14} = 0.01, p < .05$), and higher waist circumference ($\beta_{15} = 0.02, p < .05$) were all protective against 4-year memory declines.

Behavioral and Biological Health Mediators of Control-Cognition Associations

We next tested a mediation model to examine whether indicators representing physical activity and biological health factors mediated associations between perceived control and memory change. To begin with, we observed that perceived control was linked to level (estimate = 0.21, $SE = 0.02, p < .05$) and 2-year change in physical activity (estimate = 0.11, $SE = 0.01, p < .05$), as well as grip strength (estimate = 1.77, $SE = 0.17, p < .05$), forced expiratory volume (estimate = 28.18, $SE = 2.03, p < .05$), PR (estimate = -0.58, $SE = 0.19, p < .05$), hemoglobin A_{1c} (estimate = -0.07, $SE = 0.01, p < .05$), HDL-C (estimate = 1.02, $SE = 0.24, p < .05$), and waist circumference (estimate = -0.28, $SE = 0.11, p < .05$), but not with SBP (estimate = -0.04, $SE = 0.35, p > .05$).

Table 3 shows results from the nonparametric bootstrapping technique, quantifying the indirect effect of control through each mediator. Most importantly for our research question, the nonparametric bootstrapping technique allowed us to quantify whether the indirect effect of control through each mediator for memory change was reliably different from zero. Analyses revealed that level of and 2-year change in physical activity, forced expiratory volume, hemoglobin A_{1c}, and HDL-C reliably mediated the relationship between perceived control and 4-year memory change. For example, in Table 3, the total sum of the indirect effect of the

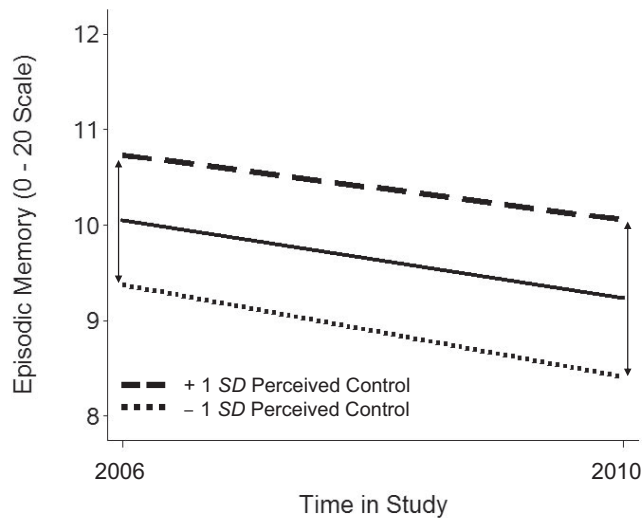


Figure 2. Illustrating the predictive effects of perceived control for memory in the Health and Retirement Study. Participants with more perceived control performed better on the episodic memory test in 2006 and experienced shallower memory declines over 4 years. In 2006, the average person remembered 10.05 words and individuals reporting ± 1 SD in perceived control remembered 10.73 and 9.37 words, respectively (difference between high and low control = 1.36 words in 2006). In 2010, the average person remembered 9.23 words, and individuals reporting ± 1 SD in perceived control in 2006 remembered 10.05 and 8.41 words, respectively (difference between high and low control = 1.64 words in 2010).

behavioral and biological health factors is .12, of which level of physical activity accounts for .04. Our results suggest that the direct effect of perceived control onto memory change is (partly) attributable to physical activity, physical fitness, and metabolic factors. Figure 3 illustrates the mediation effects of forced expiratory volume on control–memory change associations. The mediated effect is the change in the regression line relating forced expiratory volume to memory change for a change in forced expiratory volume through a 1-SD increase in perceived control, as shown in the graph (mediated effect). Following procedures outlined by Preacher and Kelley (2011), we quantified the observed effect size for the indirect effect of perceived control through the level of and 2-year change in physical activity, forced expiratory volume, PR, hemoglobin A_{1c}, and HDL-C, which was .01, .01, .02, .01, .01, and .01, respectively. The effect size metric discussed by Preacher and Kelley follows the same distribution as Cohen's *d*, suggesting that all mediation effects were in the small range of effect sizes.

The Role of Age in Moderating Control–Cognition Associations

In a final step, we estimated a multiple-age-group model to examine whether the effects of perceived control, and the behavioral and biological health factors and mediation, differed between people in midlife (ages <70 in 2006; *n* = 2,534) and old age (ages ≥ 70 in 2006; *n* = 1,643). Table 4 and Table 5 show results from our multiple-age-group model. First, we found evidence to suggest that individuals in old age were more likely to experience stronger

4-year memory declines (old age, $\Delta = -1.45$ vs. midlife, $\Delta = -0.33$). Furthermore, in both midlife and old age, indicators of behavioral and biological health were associated with 4-year memory change. More specifically, in midlife, higher levels and maintenance of physical activity, higher forced expiratory volume, lower SBP, and higher HDL-C were each protective against 4-year memory declines in midlife. In old age, higher levels of physical activity, better grip strength, and lower hemoglobin A_{1c} were each protective against 4-year memory declines.

Focusing on possible age differences in mediation, we found that in midlife, the level of and 2-year change in physical activity, as well as grip strength, forced expiratory volume, and HDL-C were more likely to mediate the effect of perceived control on 4-year memory change. Conversely, in old age, we found that only level of physical activity was more likely to mediate the association between perceived control and 4-year memory change. However, in follow-up analyses, we used Sobel tests to help determine whether the differences were reliably different from zero and found that the differences between the mediators in midlife and old age were not reliably different from zero. We note that the associations were reliably different from zero for the middle-aged group and not for the old age group, but we cannot secure with statistical tests that these are indeed different from one another.

Table 3
Mediation of the Effect of Perceived Control on 4-Year Memory Changes Through Physical Activity, Physical Fitness, Cardiovascular, and Metabolic Factors

| Indirect effect | Estimate | SE | Percentile 95% CI | |
|--------------------------------------|----------|-------|-------------------|-------|
| | | | Lower | Upper |
| Physical activity | | | | |
| Level of physical activity | 0.04* | 0.01 | 0.01 | 0.06 |
| 2-year change in physical activity | 0.02* | 0.01 | 0.01 | 0.06 |
| Physical fitness | | | | |
| Grip strength | 0.003 | 0.01 | -0.02 | 0.03 |
| Forced expiratory volume | 0.05* | 0.01 | 0.02 | 0.07 |
| Cardiovascular | | | | |
| Systolic blood pressure | 0.000 | 0.002 | -0.003 | 0.004 |
| Pulse rate | 0.002 | 0.003 | -0.002 | 0.01 |
| Metabolic | | | | |
| Hemoglobin A _{1c} | 0.01* | 0.004 | 0.002 | 0.02 |
| High-density lipoprotein cholesterol | 0.01* | 0.004 | 0.002 | 0.02 |
| Waist circumference | -0.01 | 0.003 | -0.02 | 0.000 |
| Total indirect effect | 0.12* | 0.02 | 0.07 | 0.16 |

Note. *N* = 4,177. Parameters for mediator regressed on perceived control—level of physical activity: 0.21 (0.02), std. β = .23, R^2 = .05; 2-year change in physical activity: 0.11 (0.01), std. β = .12, R^2 = .19; grip strength: 1.77 (0.17), std. β = .15, R^2 = .02; forced expiratory volume: 28.18 (2.03), std. β = .21, R^2 = .04; systolic blood pressure: -0.04 (0.35), std. β = -.002, R^2 = .00; pulse rate: -0.58 (0.19), std. β = -.05, R^2 = .002; hemoglobin A_{1c}: -0.07 (0.01), std. β = -.08, R^2 = .01; high-density lipoprotein cholesterol: 1.02 (0.24), std. β = .07, R^2 = .004; waist circumference: -0.28 (0.10), std. β = -.04, R^2 = .002. SE = standard error.

* $p < .05$.

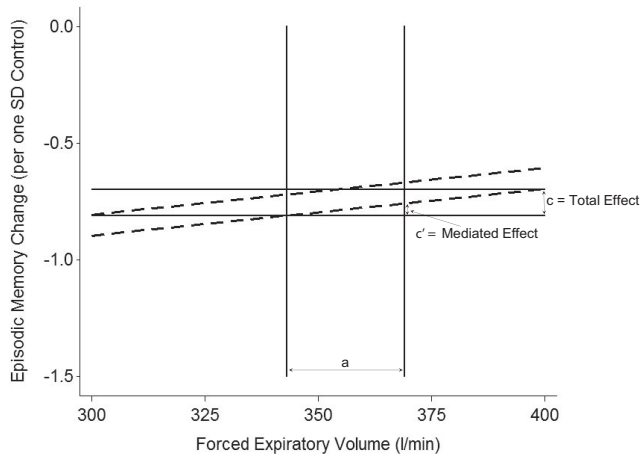


Figure 3. Illustrating the mediation effects of forced expiratory volume on control–memory change associations. The mediated effect is the change in the regression line relating forced expiratory volume to memory change for a change in forced expiratory volume of 1-*SD* increase in perceived control as shown in the graph (mediated effect). The (*a*) parameter refers to the effect of a one standard deviation increase in perceived control on forced expiratory volume. The (*c*) parameter denotes the total effect or the direct effect of perceived control on memory change, independent of mediators. The (*c'*) parameter denotes the amount of the association between perceived control and episodic memory change that is accounted for by forced expiratory volume.

Discussion

Our objective in this article was to shed light on psychological, behavioral, and biological health factors that are linked to change in cognitive functioning and possible factors that underlie control–cognition associations in adulthood and old age. To do so, we used data from the nationwide HRS to first examine whether perceived control was associated with 4-year memory change. Consistent with previous research (Agrigoroaei & Lachman, 2011; Caplan & Schooler, 2003; Windsor & Anstey, 2008), we found that higher levels of perceived control were associated with exhibiting shallower 4-year memory declines. In the next step, we included behavioral and biological health factors, and found that higher levels and maintenance of physical activity over 2 years of time, and better physical fitness, and cardiovascular and metabolic health, were associated with less-steep declines in 4-year memory change. In an exploratory step, we conducted mediation analyses to determine whether physical activity (level of and 2-year change in) and biological health (physical fitness, cardiovascular, and metabolic) factors underlie control–memory associations. Results revealed that level of and change in physical activity, forced expiratory volume, hemoglobin A_{1c}, and HDL-C were contributing to control–memory associations. In a final step, we examined whether control–memory associations and mediators of such associations differed across chronological age. We found that the protective effect of perceived control for memory change did not differ across chronological age. However, we note that there was some evidence to suggest that the mediating effects of change in physical activity and biological health factors (grip strength, forced expiratory volume, and HDL-C) were more often evident in midlife than in old age. Our discussion focuses on pathways

through which perceived control may be associated with cognitive change and targets directions for future research to more thoroughly examine the perceived control–cognition association.

Associations Between Perceived Control and Cognition

Our findings are consistent with previous research reporting that perceived control was linked to better cognitive functioning (Agrigoroaei & Lachman, 2011; Bielak et al., 2007; Caplan & Schooler, 2003; Lachman, 1986; Windsor & Anstey, 2008). We provide evidence that perceived control can be construed as a protective factor not only for health outcomes but also for changes in cognition in both midlife and old age. Our results demonstrate the importance of one's attitudes, beliefs, and motivations for cognitive functioning in adulthood and old age, and contribute to the growing interest in and discussion of risk and protective factors for cognitive aging (Spiro & Brady, 2011). We found that perceived control was linked to 4-year memory change in the context of observing memory change that was similar to previous studies. For example, our findings of individuals, on average, declining 0.25 *SD* in memory over the 4-year time period is similar to research from Windsor and Anstey (2008), who found in a sample of older adults aged 60 to 64 at baseline that immediate recall declined about 0.13 *SD* for processing speed and 0.04 *SD* for immediate recall over 4 years of time.

Interestingly, we observed that perceived control was protective against 4-year memory declines independent of behavioral and biological health correlates. The link between perceived control and cognition may also operate via pathways and correlates beyond that of physical activity and biological health. First, perceived control may be linked to better cognitive functioning through strategy use. For example, individuals with higher levels of control may exhibit better memory performance through the utilization of strategies such as clustering (Hertzog et al., 1998; Lachman & Andreoletti, 2006). Second, perceived control may be associated with better cognition through decreased anxiety and distraction (Agrigoroaei, Neupert, & Lachman, 2013). Lower perceived control may be associated with higher anxiety or expectation of failure, resulting in individuals putting forth less effort during memory tasks (Berry & West, 1993; Desrichard & Köpetz, 2005). Third, facets of one's social network (e.g., social support, integration, or activity) may underlie control–cognition associations (Gerstorf et al., 2011). For example, various theories of control argue that control beliefs allow people to mobilize social support, particularly in times of strain, thereby serving as a buffer against the effects of stress that have subsequent effects on cognition and health (see Antonucci, 2001; Juster, McEwen, & Lupien, 2010).

Behavioral and Biological Health Correlates of Cognition

We observed that components of behavioral and biological health factors were independently associated with 4-year memory change. More specifically, level of and change in physical activity, forced expiratory volume, as well as SBP, hemoglobin A_{1c}, and HDL-C were each independently associated with 4-year memory change. Physical activity was found to be one of the strongest predictors of 4-year memory change in our analyses, and these

Table 4
Latent Change Score Model of Perceived Control and Physical Activity and Biological Health Factors Predicting 4-Year Memory Change in Midlife and Old Age: Standardized and Unstandardized Regression Coefficients

| Variable | Midlife (ages < 70) | | Old age (age ≥ 70) | |
|---|--------------------------|--------|--------------------------|--------|
| | Parameter estimates (SE) | Std. β | Parameter estimates (SE) | Std. β |
| Fixed effect | | | | |
| Change, β ₀ | -0.33* (0.06) | | -1.45* (0.10) | |
| Predictor | | | | |
| Episodic memory in 2006, β ₁ | -0.54* (0.02) | -.57 | -0.47* (0.03) | -.48 |
| Perceived control, β ₂ | 0.07 (0.06) | .02 | 0.12 (0.09) | .04 |
| Women, β ₃ | 1.05* (0.08) | .18 | 1.24* (0.25) | .19 |
| Education, β ₄ | 0.18* (0.18) | .17 | 0.14* (0.03) | .14 |
| Correlates | | | | |
| Physical activity | | | | |
| Level of physical activity, β ₅ | 0.17* (0.08) | .05 | 0.23* (0.11) | .07 |
| 2-year change in physical activity, β ₆ | 0.26* (0.08) | .07 | 0.17 (0.10) | .05 |
| Physical fitness | | | | |
| Grip strength, β ₇ | 0.004 (0.01) | .01 | 0.03* (0.01) | .10 |
| Forced expiratory volume, β ₈ | 0.003* (0.001) | .11 | 0.001 (0.001) | .03 |
| Cardiovascular | | | | |
| Systolic blood pressure, β ₉ | -0.01* (0.003) | -.05 | -0.01 (0.004) | -.04 |
| Pulse rate, β ₁₀ | -0.001 (0.01) | -.002 | -0.003 (0.01) | -.01 |
| Metabolic | | | | |
| Hemoglobin A _{1c} , β ₁₁ | -0.09 (0.06) | -.03 | -0.24* (0.10) | -.06 |
| High-density lipoprotein cholesterol, β ₁₂ | 0.01* (0.004) | .05 | 0.01 (0.01) | .02 |
| Waist circumference, β ₁₃ | 0.02 (0.01) | .04 | 0.03 (0.02) | .05 |
| Random effect | | | | |
| Residual variance, σ ² | 6.04* (0.24) | | 7.48* (0.31) | |

Note. N = 4,177. Age groups: midlife (ages < 70), N = 2,534; old age (ages ≥ 70), N = 1,643. SE = standard error.

* p < .05.

findings are consistent with previous results showing the importance of remaining physical active in adulthood and old age and its implications for cognition (Anstey & Christensen, 2000; Hultsch, Hertzog, Small, & Dixon, 1999; Small et al., 2012; Spiro & Brady,

2011). We found evidence to suggest that the effect of physical activity was primarily driven by light and vigorous activity. Our results focusing on vigorous activity are in line with previous research showing the importance of physical exercise for brain

Table 5
Mediation of the Effect of Perceived Control on 4-Year Memory Changes Through Physical Activity, Physical Fitness, Cardiovascular, and Metabolic Factors in Midlife and Old Age

| Indirect effect | Midlife (ages < 70) | | | | Old age (age ≥ 70) | | | |
|--------------------------------------|---------------------|-------|-------------------|-------|--------------------|-------|-------------------|-------|
| | Estimate | SE | Percentile 95% CI | | Estimate | SE | Percentile 95% CI | |
| | | | Lower | Upper | | | Lower | Upper |
| Physical activity | | | | | | | | |
| Level of physical activity | 0.03* | 0.02 | 0.004 | 0.07 | 0.05* | 0.02 | 0.01 | 0.10 |
| 2-year change in physical activity | 0.02* | 0.01 | 0.01 | 0.04 | 0.02 | 0.01 | -0.003 | 0.05 |
| Physical fitness | | | | | | | | |
| Grip strength | 0.004 | 0.01 | -0.01 | 0.02 | 0.06 | 0.03 | 0.01 | 0.12 |
| Forced expiratory volume | 0.06* | 0.01 | 0.03 | 0.09 | 0.03 | 0.03 | -0.03 | 0.09 |
| Cardiovascular | | | | | | | | |
| Systolic blood pressure | -0.002 | 0.003 | -0.01 | 0.004 | -0.003 | 0.004 | -0.02 | 0.002 |
| Pulse rate | 0.000 | 0.004 | -0.01 | 0.01 | 0.002 | 0.004 | -0.01 | 0.02 |
| Metabolic | | | | | | | | |
| Hemoglobin A _{1c} | 0.01 | 0.01 | -0.002 | 0.02 | 0.01 | 0.01 | 0.001 | 0.03 |
| High density lipoprotein cholesterol | 0.012* | 0.006 | 0.003 | 0.03 | 0.002 | 0.004 | -0.002 | 0.02 |
| Waist circumference | -0.01 | 0.01 | -0.02 | 0.00 | 0.000 | 0.01 | -0.01 | 0.01 |
| Total indirect effect | 0.12* | 0.03 | 0.08 | 0.18 | 0.17* | 0.04 | 0.09 | 0.26 |

Note. N = 4,177. Age groups: midlife (ages < 70), N = 2,534; old age (ages ≥ 70), N = 1,643. SE = standard error.

* p < .05.

health and cognitive function (see Colcombe & Kramer, 2003; Cotman & Berchtold, 2002). Similarly, light physical activity in the form of passive, integrative, and novel information processing is beneficial for cognitive function (see Hultsch et al., 1999; Parslow, Jorm, Christensen, & Mackinnon, 2006).

Physical activity may be linked to cognition through various pathways, including biological and neuronal health. First, physical activity affects nerve growth factors and the ability to repair neurons and maintain health (Cotman & Berchtold, 2002). Second, physical exercise can lead to maintenance of cognition over time through increasing levels of brain-derived neurotrophic factor (BDNF) and other growth factors, stimulating neurogenesis, increasing resistance to brain insult, and improving learning and mental performance (Colcombe et al., 2004). Furthermore, exercise mobilizes gene expression profiles that would be predicted to benefit brain plasticity processes (Cotman & Berchtold, 2002).

Physical fitness—in particular, forced expiratory volume—was associated with cognition. Forced expiratory volume is a marker of pulmonary function that has previously been shown to be associated with better levels of and changes in cognitive functioning across adulthood and old age (Albert et al., 1995; Anstey, 2012; MacDonald et al., 2011; Wahlin et al., 2006). Pulmonary functioning may have several pathways that underlie its salutary effects on cognitive functioning. First, stronger pulmonary functioning is indicative of better blood flow to the brain, which decreases white matter hyperintensities and brain atrophy (Sachdev et al., 2006). Second, pulmonary functioning or, more broadly, physical fitness represents a proxy for physiological resilience that exerts its influence through physical activity levels (Cotman & Berchtold, 2002).

In line with previous research, we found that better cardiovascular and metabolic functioning, as indexed by lower SBP, lower hemoglobin A_{1c}, and higher HDL-C were associated with less steep cognitive declines (Thorvaldsson et al., 2012; Yaffe et al., 2004). Elevated SBP over the course of years can have cumulative effects for later cognition through atherosclerosis and impaired blood circulation (Kennelly, Lawlor, & Kenny, 2009; Waldstein, Brown, Maier, & Katzel, 2005). Poor metabolic control may impair the central nervous system functioning and subsequent cognitive function. For example, poor cardiometabolic health directly affects neuronal health through glycosylation of red blood cells, which impairs perfusion and directly damages nerve cells (peripheral neuropathy; Yaffe et al., 2011). Finally, we note that the effect sizes for the biomarkers were modest. One possible reason why biomarkers could have had a modest effect in our study is that biomarkers are considered an objective measure of health and may not have shared variance with subjective measures of control and health.

Behavioral and Biological Health Mediators of Control–Cognition Associations

Our findings suggested that perceived control may have broader effects in midlife and old age, especially independent of behavioral and biological health correlates of cognition. To elucidate some of the possible factors underlying why perceived control was associated with cognitive change, we conducted mediation analyses to determine whether physical activity and biological health factors underlie such associations. Our findings provide preliminary em-

pirical support for conceptual models of control that have delineated that physical activity and biological health underlie links between control and aging-related outcomes, but have lacked the empirical evidence to test such claims conjointly in panel surveys. However, we note that we are only at the beginning of understanding the factors that may underlie control–cognition associations.

Along the lines suggested, we found that the level of and 2-year change in physical activity, and physical fitness and metabolic health systems, mediated control–memory associations. As a first pathway, we targeted physical activity and observed that both level and 2-year change mediated control–memory change associations. Findings provide support for the vascular hypothesis that engagement in physical activities can indeed protect against cognitive decline in adulthood and old age (Bielak et al., 2012; Fratiglioni et al., 2004; Hertzog, Kramer, Wilson, & Lindenberger, 2008; Hultsch et al., 1999; Spiro & Brady, 2008). Our study demonstrates that perceived control may be a factor that promotes this process. Individuals who report higher levels of perceived control have an increased likelihood of partaking in and maintaining physical activity over time, such as strenuous exercise, which subsequently leads to maintaining cognitive functioning. Physical activity is likely associated with preserved cognition through better cardiovascular (i.e., lower blood pressure, better PR), physical fitness (i.e., better pulmonary functioning) and lower risk of metabolic syndrome (Infurna & Gerstorf, in press; Penedo & Dahn, 2005; Small et al., 2012; Yaffe et al., 2009). We made use of a measure that assessed physical activity very broadly and whose measurement properties were less than ideal (e.g., Cronbach's $\alpha = .54$). To reach the next level of insights, researchers should target specific activities and other health behaviors, such as diet, smoking, and alcohol. Is it partaking in strenuous exercise or is it the total amount of activity individuals are involved in throughout the day (e.g., as measured through actigraphy, Buchman, Wilson, & Bennett, 2008; or through nonexercise activity thermogenesis, Levine et al., 1999) that have the most beneficial effects for cognition? Finally, the relationship between perceived control and physical activity is likely bidirectional, which should be disentangled further in longitudinal studies. More perceived control may be associated with increased likelihood of physical activity and, in turn, partaking in physical activity boosts one's ability and perceptions to attain desired outcomes.

We also found that physical fitness—more specifically, forced expiratory volume—mediated control–memory change associations. Perceived control is associated with motivational beliefs and processes that provide individuals intrinsic vigor, leading to better physical fitness (Brach et al., 2003; Rantanen et al., 1999). Pulmonary functioning may result in better cognition via better blood flow to the brain, decreasing white matter hyperintensities and physiological resilience (Cotman & Berchtold, 2002; Sachdev et al., 2006). In contrast to previous research, grip strength was not predictive of memory change and did not mediate control–memory change associations. Discrepant findings may be due to our one-time assessment of grip strength, whereas previous research examined wave-to-wave couplings at more frequent intervals (e.g., MacDonald et al., 2011). Additionally, we included grip strength in our models conjointly in the context of other behavior and biological health factors, with shared variance across other factors possibly leading to little predictive effects.

Markers of metabolic, but not cardiovascular, health mediated associations between perceived control and memory change. Perceived control may be associated with metabolic systems through health-promoting behaviors (Infurna & Gerstorf, *in press*; Roepke & Grant, 2011). For example, perceived control is associated with eating a healthy and balanced diet (i.e., lower sugar consumption, fewer saturated fats), which suppresses glucose production, promotes antioxidant functioning, and promotes pre high-density lipoprotein production, thereby providing protection against atherosclerosis and possible cardiac events (Penedo & Dahn, 2005; Selvin et al., 2010; Stefanick et al., 1998). Furthermore, we found that one of our markers of cardiovascular functioning, SBP, was linked to 4-year memory change, but did not mediate control–memory change associations. One possible reason may be that resting blood pressure (as opposed to dynamic assessments in the lab or throughout the day) may be a suboptimal operational definition to examine control–SBP associations. For example, perceived control buffers increases in SBP during times of acute stress, such as in experimental settings or among people who experience chronic stress, such as caregivers (Roepke & Grant, 2011). Furthermore, our measures of blood pressure and PR were only taken at one point in time, which may not be reliable enough to exhibit associations with cognition. It may be that we need to measure blood pressure and PR repeatedly and concomitantly to show how they are interrelated with cognition (e.g., Schwartz & Stone, 1998).

One way to advance the field further would be to examine multidirectional dynamics between perceived control, physical activity, and biological health. The mediation analyses focusing on biological health focused on one point in time (i.e., 2006), and to better understand such associations, research needs to test bidirectional longitudinal associations among perceived control with biological health factors (see Lindenberger, von Oertzen, Ghisletta, & Hertzog, 2011; Maxwell & Cole, 2007). Mediation processes occur over time, and in order to better understand the role of perceived control for cognition, the longitudinal nature of control–biological health associations needs to be disentangled further. First and foremost, researchers need longitudinal data on all the variables of interest to fully address the possibility of mediation (for discussion, see Imai, Keele, & Tingley, 2010; Maxwell & Cole, 2007; Valeri & VanderWeele, 2013). There are several techniques that could allow us to address questions about temporal ordering (e.g., bivariate dual change score model, McArdle & Hamagami, 2001; continuous time modeling, Voelkle, Oud, Davidov, & Schmidt, 2012) or address third-variable accounts (e.g., propensity score matching; Coffman, 2011; Foster, 2010; Stuart, 2010; West et al., 2008). The bivariate dual change score model permits for examining the temporal precedence among the variables of interest (Grimm, An, McArdle, Zonderman, & Resnick, 2012; McArdle & Hamagami, 2001), but still leaves open the possibility of unobserved factors to confound the observed relationships. Propensity score matching would be able to tackle the possibility of factors confounding the observed relationships. Second, based on the present data, we do not know whether mediation of the kind reported does indeed occur; to what extent it actually runs in the opposite direction, in the sense that control mediates the association between physical activity and memory change; or to what extent common causes affecting the mediator and the dependent variable are at work. This is in line with empirical evidence that suggests that the relationship among perceived control, the

targeted mediators (behavioral and biological health factors), and cognition is bidirectional. For example, more perceived control is linked to engaging in more physical activity (Lachman & Firth, 2004; White et al., 2012), which, in turn, may enhance or maintain perceived control through repeated successes and positive cognitive appraisals of one's own abilities (Bandura, 1977). Additionally, poorer levels of or exhibiting declines in cognitive functioning over time may decrease feelings of control through memory lapses and the perceived inability to master and complete everyday tasks (Agrigoroaei, Neupert, & Lachman, 2013; Hertzog et al., 1998).

The Role of Age in Moderating Control–Cognition Associations

Our findings suggest that the direct, protective effect of perceived control for memory change is consistent in midlife and old age. Our results are in line with previous research that has documented little evidence to suggest control–cognition associations to be stronger in older ages (Agrigoroaei & Lachman, 2011), but are in contrast to studies reporting that control–cognition associations are stronger in older ages (Windsor & Anstey, 2008). Differences in our findings could be due to the measure of cognition. Previous studies that found control–cognition associations to be stronger in older ages focused on processing speed and cognitive difficulties, which could be more sensitive to differences by one's point in the adult life span. For indices of processing speed that involve a motor component, perceived control may be an enabling factor through the use of compensatory strategies (see Windsor & Anstey, 2008).

As a corollary, our findings suggest that much can be learned from assessing whether associations between psychological factors and aging-related outcomes are similar across the adult life span (Deary, Weiss, & Batty, 2010; Smith & Infurna, 2011). It may be that differences in physical activity and biological health factors begin in adolescence and young adulthood, which are then exacerbated in midlife and old age. For example, findings from the 1970 British Cohort Study found that higher levels of perceived control at age 10 were linked to a better health profile at age 30 (e.g., reduced risk of obesity, more physically active, and not a current smoker; Gale, Batty, & Deary, 2008). Recently, the Dunedin study found that children with more self-control in childhood and adolescence were more likely to have a better cardiovascular health profile in young adulthood (Moffitt et al., 2011). Similarly, research in children has shown that children who report higher levels of control are likely to exhibit lower levels of hemoglobin A_{1c} and higher forced expiratory volume (Griffin & Chen, 2006; Nabors, McGrady, & Kichler, 2010). Therefore, a life-span approach can help contribute to studying how and why people experience different trajectories of cognitive change in midlife and old age. For example, vigorous physical activity may decline with age, but for persons who still manage to exercise in very old age, it may be associated with maintenance of levels of health and cognition.

Conceptual Implications

We explored the role of psychological, behavioral, and biological health factors in determining how and why cognition changes

in adulthood and old age. We next discuss future research opportunities to enhance our understanding of the interplay among perceived control, physical activity, biological health, and cognition in adulthood and old age. Our study was done over a macro timescale of years, examining how between-person differences in perceived control were associated with between-person differences in memory change (for discussion, see Gerstorf, Lövdén, Röcke, Smith, & Lindenberger, 2007; Molenaar, Huizenga, & Nesselroade, 2003). From this perspective, inferences cannot be made regarding within-person relations; it is upon future research to examine ergodicity questions about the equivalence of structural relations based on between-person and within-person variation (for discussion, see Molenaar et al., 2003; Sliwinski & Mogle, 2008). One avenue to move toward this would be to utilize the strengths of measurement-burst designs to assess developmental processes that transpire over the course of days or weeks that can be linked to cognitive change taking place over the course of years (for discussion, see Nesselroade, 1991; Ram & Gerstorf, 2009). This would be akin to recent findings from Neupert and Allaire (2012), who examined within-person coupling between control beliefs and cognition over 60 days, and found that control was coupled with concurrent and subsequent cognitive performance (see also, Agrigoroaei et al., 2013). The next step would be to incorporate whether various physical activity and biological health factors may underlie control–cognition associations at the daily level. For example, higher levels of perceived control at the daily level may be associated with increased likelihood of performing strenuous exercise or eating a healthier diet, which has implications for biological health. If such burst designs are embedded in studies examining long-term longitudinal change, this would allow us to track whether daily processes surrounding perceived control, physical activity, biological health, and their across-domain interplay account for observed longitudinal changes in cognition.

Limitations and Outlook

In closing, we note several limitations of our study. First, although we conjointly targeted various physical activity and biological health factors as mediators of control–memory associations, there are other factors that were not available for our analyses. For example, we did not have access to indicators of immune system functioning, such as C-reactive protein or pro-inflammatory markers that are associated with the level of and change in cognition in adulthood and old age (Aiello et al., 2006). This would allow for assessing whether biological health pathways underlying control–cognition associations also operate via immune system functioning, in addition to physical fitness and metabolic health. In a similar vein, we targeted each behavior and biological health indicator separately, but there may be synergistic effects across behavior, physical fitness, cardiovascular, and metabolic factors with genetic components (e.g., Bender & Raz, 2012; Podewils et al., 2005). For example, the protective effects of perceived control on cognitive declines in adulthood and old age may differ for individuals who are carriers of the $\epsilon 4$ allele apolipoprotein E gene (Small, Rosnick, Fratiglioni, & Bäckman, 2004). Second, cognition is a multidimensional construct, and we only focused on memory. For example, if data for crystallized abilities or processing speed had been available, we could have examined whether our find-

ings generalize to other dimensions of cognitive functioning. Third, as panel surveys begin to incorporate biological health factors into their battery of assessments, it will be essential for researchers to target bidirectional associations between psychosocial and biological indicators. For example, perceived control is predictive of physical fitness and metabolic health, but the association may also operate in the reverse direction, with people who are biologically fitter having stronger beliefs that they can exert influence onto their life circumstances. Such tests of bidirectional associations can be done with only two waves of data, with the advancement of latent change score models (Ferrer & McArdle, 2010). Fourth, our subsample from the larger HRS was a select group of participants in that they were younger and overall healthier than the group from which they were selected. In our view, this provides a conservative estimate of associations present in the general population. Finally, we have deliberately chosen a very liberal criterion of $p < .05$, with a large sample size, because of the exploratory nature of our (mediator) analyses. Future research needs to examine whether or not the effects observed here for a national sample are particularly pronounced among certain subgroups. For example, it is possible that perceived control may have particularly pronounced protective effects against cognitive declines among subgroups of the population who exhibit high cardio-metabolic risk in young adulthood and midlife, which are precursors for declines in cognition and development of cardiovascular disease in old age (see Grundy et al., 2005; Spiro & Brady, 2011).

In sum, our study highlights the importance of perceptions of control for cognitive change in adulthood and old age. Our study adds to conceptual models of control and extant empirical evidence that shows higher levels of perceived control are predictive of better levels and more positive change in cognitive functioning (Caplan & Schooler, 2003; Lachman, 2006; Windsor & Anstey, 2008). As an extension of previous research, our study has shed light on possible behavior and biological health factors that underlie control–cognition associations. We take our results to provide impetus for future research to examine how and why risk and protective factors, like perceived control, are associated with changes in cognition in adulthood and old age.

References

- Agrigoroaei, S., & Lachman, M. E. (2011). Cognitive functioning in midlife and old age: Combined effects of psychosocial and behavioral factors. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, *66*, i130–i140. doi:10.1093/geronb/gbr017
- Agrigoroaei, S., Neupert, S. D., & Lachman, M. E. (2013). Maintaining a sense of control in the context of cognitive challenges: Greater stability in control beliefs benefits working memory. *Geropsych: The Journal of Gerontopsychology and Geriatric Psychiatry*, *26*, 49–59.
- Aiello, A. E., Haan, M. N., Blythe, L., Moore, K., Gonzalez, J. M., & Jagust, W. (2006). The influence of latent viral infection on rate of cognitive decline over 4 years. *Journal of the American Geriatrics Society*, *54*, 1046–1054. doi:10.1111/j.1532-5415.2006.00796.x
- Albert, M. S., Jones, K., Savage, C. R., Berkman, L., Seeman, T., Blazer, D., & Rowe, J. W. (1995). Predictors of cognitive change in older persons: MacArthur Studies of Successful Aging. *Psychology and Aging*, *10*, 578–589. doi:10.1037/0882-7974.10.4.578
- Anstey, K. J. (2012). Biomarkers and memory aging: A lifecourse per-

- spective. In M. Naveh-Benjamin & N. Ohta (Eds.), *Memory and aging* (pp. 349–372). New York, NY: Psychology Press.
- Anstey, K., & Christensen, H. (2000). Education, activity, health, blood pressure and apolipoprotein E as predictors of cognitive change in old age: A review. *Gerontology, 46*, 163–177. doi:10.1159/000022153
- Anstey, K. J., Luszcz, M. A., Giles, L. C., & Andrews, G. R. (2001). Demographic, health, cognitive, and sensory variables as predictors of mortality in very old adults. *Psychology and Aging, 16*, 3–11. doi:10.1037/0882-7974.16.1.3
- Antonucci, T. C. (2001). Social relations: An examination of social networks, social support, and sense of control. In J. E. Birren & K. W. Schaie (Eds.), *Handbook of the psychology of aging* (5th ed., pp. 427–453). San Diego, CA: Academic Press.
- Baltes, P. B., Lindenberger, U., & Staudinger, U. M. (2006). Life-span theory in developmental psychology. In R. M. Lerner (Ed.), *Handbook of child psychology Vol. 1: Theoretical models of human development* (6th ed., pp. 569–664). New York, NY: Wiley.
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215. doi:10.1037/0033-295X.84.2.191
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York, NY: Freeman.
- Bandura, A. (2004). Health promotion by social cognitive means. *Health Education & Behavior, 31*, 143–164. doi:10.1177/1090198104263660
- Barter, P. J., & Rye, K.-A. (1996). High density lipoproteins and coronary heart disease. *Atherosclerosis, 121*, 1–12. doi:10.1016/0021-9150(95)05675-0
- Bender, A. R., & Raz, N. (2012). Age-related differences in episodic memory: A synergistic contribution of genetic and physiological vascular risk factors. *Neuropsychologia, 50*, 704–714. doi:10.1016/j.neuropsychologia.2011.12.025
- Berry, J. M., & West, R. L. (1993). Cognitive self-efficacy in relation to personal mastery and goal setting across the life span. *International Journal of Behavioral Development, 16*, 351–379. doi:10.1177/016502549301600213
- Bielak, A. A. M. (2010). How can we not “lose it” if we still don’t understand how to “use it”? Unanswered questions about the influence of activity participation on cognitive performance in older age – A mini-review. *Gerontology, 56*, 507–519. doi:10.1159/000264918
- Bielak, A. A. M., Christensen, H., & Windsor, T. D. (2012). Activity engagement is related to level, but not change in cognitive ability across adulthood. *Psychology and Aging, 27*, 219–228. doi:10.1037/a0024667
- Bielak, A. A. M., Hultsch, D. F., Levy-Ajzenkopf, J., MacDonald, S. W. S., Hunter, M. A., & Strauss, E. (2007). Short-term changes in general and memory-specific control beliefs and their relationship to cognition in younger and older adults. *The International Journal of Aging & Human Development, 65*, 53–71. doi:10.2190/G458-X101-0338-746X
- Brach, J. S., Fitzgerald, S., Newman, A. B., Kelsey, S., Kuller, L., VanSwearingen, J. M., & Kriska, A. M. (2003). Physical activity and functional status in community-dwelling older women: A 14-year prospective study. *Archives of Internal Medicine, 163*, 2565–2571. doi:10.1001/archinte.163.21.2565
- Brady, C. B., Spiro, A., & Graziano, J. M. (2005). Effects of age and hypertension status on cognition: The Veterans Affairs Normative Aging Study. *Neuropsychology, 19*, 770–777. doi:10.1037/0894-4105.19.6.770
- Buchman, A. S., Wilson, R. S., & Bennett, D. A. (2008). Total daily activity is associated with cognition in older persons. *The American Journal of Geriatric Psychiatry, 16*, 697–701. doi:10.1097/JGPM.0b013e31817945f6
- Caplan, L. J., & Schooler, C. (2003). The roles of fatalism, self-confidence, and intellectual resources in the disablement process in older adults. *Psychology and Aging, 18*, 551–561. doi:10.1037/0882-7974.18.3.551
- Casserly, I., & Topol, E. (2004). Converge of atherosclerosis and Alzheimer’s disease: Inflammation, cholesterol, and misfolded proteins. *The Lancet, 363*, 1139–1146. doi:10.1016/S0140-6736(04)15900-X
- Clarke, P., Fisher, G., House, J., Smith, J., & Weir, D. (2008). *Guide to content of the HRS psychosocial leave-behind participant lifestyle questionnaires: 2004 & 2006*. Documentation report version 2.0. Ann Arbor, MI: University of Michigan. doi:10.7826/ISR-UM.06.585031.001.05.0016.2008
- Coffman, D. L. (2011). Estimating causal effects in mediation analysis using propensity scores. *Structural Equation Modeling, 18*, 357–369. doi:10.1080/10705511.2011.582001
- Colcombe, S. J., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science, 14*, 125–130. doi:10.1111/1467-9280.t01-1-01430
- Colcombe, S. J., Kramer, A. F., Erickson, K. I., Scalf, P., McAuley, E., Cohen, N. J., . . . Elavsky, S. (2004). Cardiovascular fitness, cortical plasticity, and aging. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 101*, 3316–3321.
- Cotman, C. W., & Berchtold, N. C. (2002). Exercise: A behavioral intervention to enhance brain health and plasticity. *Trends in Neurosciences, 25*, 295–301. doi:10.1016/S0166-2236(02)02143-4
- Crimmins, E., Guyer, H., Langa, K., Ofstedal, M. B., Wallace, R., & Weir, D. (2008). *Documentation of physical measures, anthropometrics and blood pressure in the Health and Retirement Study*. HRS documentation report DR-011. Ann Arbor, MI: University of Michigan.
- Crimmins, E., Guyer, H., Langa, K., Ofstedal, M. B., Wallace, R., & Weir, D. (2009). *Documentation of biomarkers in the Health and Retirement Study*. HRS documentation report. Ann Arbor, MI: University of Michigan. doi:10.7826/ISR-UM.06.585031.001.05.0018.2009
- Danner, D. D., Snowdon, D. A., & Friesen, W. V. (2001). Positive emotions in early life and longevity: Findings from the Nun Study. *Journal of Personality and Social Psychology, 80*, 804–813. doi:10.1037/0022-3514.80.5.804
- Deary, I. J., Weiss, A., & Batty, G. D. (2010). Intelligence and personality as predictors of illness and death: How researchers in differential psychological and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychological Science in the Public Interest, 11*, 53–79. doi:10.1177/1529100610387081
- de la Torre, J. C. (2004). Is Alzheimer’s disease a neurodegenerative or a vascular disorder? Data, dogma, and dialectics. *Lancet Neurology, 3*, 184–190. doi:10.1016/S1474-4422(04)00683-0
- Després, J.-P., Lemieux, I., Bergeron, J., Pibarot, P., Mathieu, P., Larose, E., . . . Poirier, P. (2008). Abdominal obesity and the metabolic syndrome: Contribution to global cardiometabolic risk. *Arteriosclerosis, Thrombosis, and Vascular Biology, 28*, 1039–1049. doi:10.1161/ATVBAHA.107.159228
- Desrichard, O., & Köpetz, C. (2005). A threat in the elder: The impact of task-instructions, self-efficacy and performance expectations on memory performance in the elderly. *European Journal of Social Psychology, 35*, 537–552. doi:10.1002/ejsp.249
- Diehl, M., & Hay, E. L. (2010). Risk and resilience factors in coping with daily stress in adulthood: The role of age, self-concept incoherence, and personal control. *Developmental Psychology, 46*, 1132–1146. doi:10.1037/a0019937
- Ferrer, E., & McArdle, J. J. (2010). Longitudinal modeling of developmental changes in psychological research. *Current Directions in Psychological Science, 19*, 149–154. doi:10.1177/0963721410370300
- Foster, E. M. (2010). Causal inference and developmental psychology. *Developmental Psychology, 46*, 1454–1480. doi:10.1037/a0020204
- Fratiglioni, L., Paillard-Borg, S., & Winblad, B. (2004). An active and social integrated lifestyle in late life might protect against dementia. *Lancet Neurology, 3*, 343–353. doi:10.1016/S1474-4422(04)00767-7
- Gale, C. R., Batty, G. D., & Deary, I. J. (2008). Locus of control at age 10 years and health outcomes and behaviors at age 30 years: The 1970

- British Cohort Study. *Psychosomatic Medicine*, 70, 397–403. doi:10.1097/PSY.0b013e31816a719e
- Gerstorff, D., Hoppmann, C. A., Kadlec, K. M., & McArdle, J. J. (2009). Memory and depressive symptoms are dynamically linked among married couples: Longitudinal evidence from the AHEAD Study. *Developmental Psychology*, 45, 1595–1610. doi:10.1037/a0016346
- Gerstorff, D., Lövdén, M., Röcke, C., Smith, J., & Lindenberger, U. (2007). Well-being affects changes in perceptual speed in advanced old age: Longitudinal evidence for a dynamic link. *Developmental Psychology*, 43, 705–718. doi:10.1037/0012-1649.43.3.705
- Gerstorff, D., Röcke, C., & Lachman, M. E. (2011). Antecedent-consequent relations of perceived control to health and social support: Longitudinal evidence for between-domain associations across adulthood. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 66, 61–71. doi:10.1093/geronb/gbq077
- Griffin, M. J., & Chen, E. (2006). Perceived control and immune and pulmonary outcomes in children with asthma. *Psychosomatic Medicine*, 68, 493–499. doi:10.1097/01.psy.0000221367.96439.da
- Grimm, K. J., An, Y., McArdle, J. J., Zonderman, A. B., & Resnick, S. M. (2012). Recent changes leading to subsequent changes: Extensions of multivariate latent difference score models. *Structural Equation Modeling*, 19, 268–292. doi:10.1080/10705511.2012.659627
- Grundy, S. M., Cleeman, J. I., Daniels, S. R., Donato, K. A., Eckel, R. H., Franklin, B. A., . . . Costa, F. (2005). Diagnosis and management of the metabolic syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation*, 112, 2735–2752. doi:10.1161/CIRCULATIONAHA.105.169404
- Hay, E. L., & Diehl, M. (2010). Reactivity to daily stressors in adulthood: The importance of stressor type in characterizing risk factors. *Psychology and Aging*, 25, 118–131. doi:10.1037/a0018747
- Hertzog, C., Kramer, A. F., Wilson, R. S., & Lindenberger, U. (2008). Enrichment effects on adult cognitive development: Can the functional capacity of older adults be preserved and enhanced? *Psychological Science in the Public Interest*, 9, 1–65.
- Hertzog, C., McGuiire, C. L., & Lineweaver, T. T. (1998). Aging, attributions, perceived control, and strategy use in a free recall task. *Aging, Neuropsychology, and Cognition*, 5, 85–106. doi:10.1076/anec.5.2.85.601
- Hultsch, D. F., Hertzog, C., Small, B. J., & Dixon, R. A. (1999). Use it or lose it: Engaged lifestyle as a buffer of cognitive decline in aging? *Psychology and Aging*, 14, 245–263. doi:10.1037/0882-7974.14.2.245
- Imai, K., Keele, L., & Tingley, D. (2010). A general approach to causal mediation analysis. *Psychological Methods*, 15, 309–334. doi:10.1037/a0020761
- Infurna, F. J., & Gerstorff, D. (in press). Perceived control relates to better functional health and lower cardio-metabolic risk: The mediating role of physical activity. *Health Psychology*.
- Infurna, F. J., Gerstorff, D., & Zarit, S. H. (2011). Examining dynamic links between perceived control and health: Longitudinal evidence for differential effects in midlife and old age. *Developmental Psychology*, 47, 9–18. doi:10.1037/a0021022
- Infurna, F. J., Ram, N., & Gerstorff, D. (in press). Level and change in perceived control predict 19-year mortality: Findings from the Americans' Changing Lives Study. *Developmental Psychology*.
- Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, 35, 2–16. doi:10.1016/j.neubiorev.2009.10.002
- Kennelly, S. P., Lawlor, B. A., & Kenny, R. A. (2009). Blood pressure and risk for dementia: A double edged sword. *Ageing Research Reviews*, 8, 61–70. doi:10.1016/j.arr.2008.11.001
- Khaw, K.-T., & Wareham, N. (2006). Glycated hemoglobin as a marker of cardiovascular risk. *Current Opinion in Lipidology*, 17, 637–643. doi:10.1097/MOL.0b013e3280106b95
- Kramer, A. F., Bherer, L., Colcombe, S. J., Dong, W., & Greenough, W. T. (2004). Environmental influences on cognitive and brain plasticity during aging. *The Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*, 59, M940–M957. doi:10.1093/gerona/59.9.M940
- Kumari, M., Brunner, E., & Fuhrer, R. (2000). Minireview: Mechanisms by which the metabolic syndrome and diabetes impair memory. *The Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*, 55, B228–B232. doi:10.1093/gerona/55.5.B228
- Lachman, M. E. (1986). Locus of control in aging research: A case for multidimensional and domain-specific assessment. *Psychology and Aging*, 1, 34–40. doi:10.1037/0882-7974.1.1.34
- Lachman, M. E. (2006). Perceived control over aging-related declines: Adaptive beliefs and behaviors. *Current Directions in Psychological Science*, 15, 282–286. doi:10.1111/j.1467-8721.2006.00453.x
- Lachman, M. E., & Agrigoroaei, S. (2010). Promoting functional health in midlife and old age: Long-term protective effects of control beliefs, social support, and physical exercise. *PLoS ONE*, 5, e13297.
- Lachman, M. E., & Andreoletti, C. (2006). Strategy use mediates the relationship between control beliefs and memory performance for middle-aged and older adults. *Journals of Gerontology: Psychological Sciences*, 61B, P88–P94.
- Lachman, M. E., & Firth, K. M. (2004). The adaptive value of feeling in control during midlife. In O. G. Brim, Jr., C. D. Ryff, & R. C. Kessler (Eds.), *How healthy are we? A national study of well-being at midlife* (pp. 320–349). Chicago, IL: University of Chicago Press.
- Lachman, M. E., Jette, A., Tennstedt, S., Howland, J., Harris, B. A., & Peterson, E. (1997). A cognitive-behavioural model for promoting regular physical activity in older adults. *Psychology, Health & Medicine*, 2, 251–261. doi:10.1080/13548509708400583
- Lachman, M. E., Neupert, S. D., & Agrigoroaei, S. (2011). The relevance of control beliefs for health and aging. In K. W. Schaie & S. L. Willis (Eds.), *Handbook of the psychology of aging* (7th ed., pp. 175–190). San Diego, CA: Academic Press. doi:10.1016/B978-0-12-380882-0.00011-5
- Levine, J. A., Eberhardt, N. L., & Jensen, M. D. (1999). Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science*, 283, 212–214. doi:10.1126/science.283.5399.212
- Lindenberger, U., von Oertzen, T., Ghisletta, P., & Hertzog, C. (2011). Cross-sectional age variance extraction: What's change got to do with it? *Psychology and Aging*, 26, 34–47. doi:10.1037/a0020525
- MacDonald, S. W. S., DeCarlo, C. A., & Dixon, R. A. (2011). Linking biological and cognitive aging: Toward improving characterizations of developmental time. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 66, i59–i70. doi:10.1093/geronb/gbr039
- MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual Review of Psychology*, 58, 593–614. doi:10.1146/annurev.psych.58.110405.085542
- Maxwell, S. E., & Cole, D. A. (2007). Bias in cross-sectional analyses of longitudinal mediation. *Psychological Methods*, 12, 23–44. doi:10.1037/1082-989X.12.1.23
- McArdle, J. J. (2009). Latent variable modeling of differences in changes with longitudinal data. *Annual Review of Psychology*, 60, 577–605. doi:10.1146/annurev.psych.60.110707.163612
- McArdle, J. J., Fisher, G. G., & Kadlec, K. M. (2007). Latent variable analyses of age trends from the Health and Retirement Study, 1992–2004. *Psychology and Aging*, 22, 525–545. doi:10.1037/0882-7974.22.3.525
- McArdle, J. J., & Hamagami, F. (2001). Linear dynamic analyses of incomplete longitudinal data. In L. Collins & A. Sayer (Eds.), *Methods for the analysis of change* (pp. 139–175). Washington, DC: APA Press. doi:10.1037/10409-005
- McArdle, J. J., & Nesselroade, J. R. (2003). Growth curve analysis in contemporary psychological research. In J. Shinka & W. Velicer (Eds.), *Comprehensive handbook of psychology: Research methods in psychol-*

- ogy (Vol. 2, pp. 447–480). New York, NY: Wiley. doi:10.1002/0471264385.wei0218
- McAuley, E. (1993). Self-efficacy and the maintenance of exercise participation in older adults. *Journal of Behavioral Medicine, 16*, 103–113. doi:10.1007/BF00844757
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine, 338*, 171–179. doi:10.1056/NEJM199801153380307
- Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 108*, 2693–2698. doi:10.1073/pnas.1010076108
- Molenaar, P. C. M., Huizenga, H. M., & Nesselroade, J. R. (2003). The relationship between the structure of interindividual and intraindividual variability: A theoretical and empirical vindication of developmental systems theory. In U. M. Staudinger & U. Lindenberger (Eds.), *Understanding human development: Dialogues with lifespan psychology* (pp. 339–360). Dordrecht, Netherlands: Kluwer. doi:10.1007/978-1-4615-0357-6_15
- Muthén, L. K., & Muthén, B. O. (1998–2007). *Mplus user's guide* (4th ed.). Los Angeles, CA: Author.
- Nabors, L., McGrady, M. E., & Kichler, J. (2010). Children's attitudes toward their diabetes, locus of control, and HbA1c levels. *Journal of Developmental and Physical Disabilities, 22*, 475–484. doi:10.1007/s10882-009-9183-3
- Nesselroade, J. R. (1991). The warp and woof of the developmental fabric. In R. Downs, L. Liben, & D. Palermo (Eds.), *Visions of development, the environment, and aesthetics: The legacy of Joachim F. Wohlwill* (pp. 213–240). Hillsdale, NJ: Erlbaum.
- Neupert, S. D., & Allaire, J. C. (2012). I think I can, I think I can: Examining the within-person coupling of control beliefs and cognition in older adults. *Psychology and Aging, 27*, 742–749. doi:10.1037/a0026447
- Ofstedal, M. B., Fisher, G. G., & Herzog, A. R. (2005). *Documentation of cognitive functioning measures in the Health and Retirement Study*. HRS/AHEAD documentation report DR-006. Ann Arbor, MI: University of Michigan.
- Parslow, R. A., Jorm, A. F., Christensen, H., & Mackinnon, A. (2006). An instrument to measure engagement in life: Factor analysis and associations with sociodemographic, health and cognition measures. *Gerontology, 52*, 188–198.
- Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior, 19*, 2–21. doi:10.2307/2136319
- Penedo, F. J., & Dahn, J. R. (2005). Exercise and well-being: A review of mental and physical health benefits associated with physical activity. *Current Opinion in Psychiatry, 18*, 189–193. doi:10.1097/00001504-200503000-00013
- Podewils, L. J., Guallar, E., Kuller, L. H., Fried, L. P., Lopez, O. L., Carlson, M., & Lyketsos, C. G. (2005). Physical activity, APOE Genotype, and dementia risk: Findings from the Cardiovascular Health Cognition Study. *American Journal of Epidemiology, 161*, 639–651. doi:10.1093/aje/kwi092
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*, 879–891. doi:10.3758/BRM.40.3.879
- Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods, 16*, 93–115. doi:10.1037/a0022658
- Qiu, C., Winblad, B., & Fratiglioni, L. (2005). The age-dependent relation of blood pressure to cognitive function and dementia. *The Lancet Neurology, 4*, 487–499. doi:10.1016/S1474-4422(05)70141-1
- Ram, N., & Gerstorf, D. (2009). Time-structured and net intraindividual variability: Tools for examining the development of dynamic characteristics and processes. *Psychology and Aging, 24*, 778–791. doi:10.1037/a0017915
- Rantanen, T., Guralnik, J. M., Foley, D., Masaki, K., Leveille, S., Curb, J. D., & White, L. (1999). Midlife hand grip strength as a predictor of old age disability. *Journal of American Medical Association, 281*, 558–560. doi:10.1001/jama.281.6.558
- Rantanen, T., Volpato, S., Ferrucci, L., Heikkinen, E., Fried, L. P., & Guralnik, J. M. (2003). Handgrip strength and cause-specific and total mortality in older disabled women: Exploring the mechanism. *Journal of American Geriatric Society, 51*, 636–641.
- Reich, J. W., & Zautra, A. J. (1990). Dispositional control beliefs and the consequences of a control-enhancing intervention. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences, 45*, P46–P51.
- Rodin, J. (1986). Aging and health: Effects of the sense of control. *Science, 233*, 1271–1276. doi:10.1126/science.3749877
- Roepke, S. K., & Grant, I. (2011). Toward a more complete understanding of the effects of personal mastery on cardio-metabolic health. *Health Psychology, 30*, 615–632. doi:10.1037/a0023480
- Rowe, J. W., & Kahn, R. L. (1987). Human aging: Usual and successful. *Science, 237*, 143–149. doi:10.1126/science.3299702
- Sachdev, P. S., Anstey, K. J., Parslow, R. A., Wen, W., Maller, J., Kumar, R., . . . Jorm, A. F. (2006). Pulmonary function, cognitive impairment and brain atrophy in a middle-aged community sample. *Dementia and Geriatric Cognitive Disorders, 21*, 300–308. doi:10.1159/000091438
- Schwartz, J. E., & Stone, A. A. (1998). Data analysis for EMA studies. *Health Psychology, 17*, 6–16. doi:10.1037/0278-6133.17.1.6
- Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco, CA: Freeman.
- Selvin, E., Steffes, M. W., Zhu, H., Matsushita, K., Wagenknecht, L., Pankow, J., . . . Brancati, F. L. (2010). Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. *New England Journal of Medicine, 362*, 800–811. doi:10.1056/NEJMoa0908359
- Skaff, M. M. (2007). Sense of control and health: A dynamic duo in the aging process. In C. M. Aldwin, C. L. Park, & A. Spiro (Eds.), *Handbook of health psychology and aging* (pp. 186–209). New York, NY: Guilford Press.
- Skinner, E. A. (1996). A guide to constructs of control. *Journal of Personality and Social Psychology, 71*, 549–570. doi:10.1037/0022-3514.71.3.549
- Sliwinski, M. J., & Mogle, J. (2008). Time-based and process-based approaches to analysis of longitudinal data. In S. M. Hofer & D. F. Alwin (Eds.), *Handbook of cognitive aging: Interdisciplinary perspectives* (pp. 477–491). Thousand Oaks, CA: Sage. doi:10.4135/9781412976589.n28
- Small, B. J., Dixon, R. A., McArdle, J. J., & Grimm, K. J. (2012). Do changes in lifestyle engagement moderate cognitive decline in normal aging? Evidence from the Victoria Longitudinal Study. *Neuropsychology, 26*, 144–155. doi:10.1037/a0026579
- Small, B. J., Rosnick, C. B., Fratiglioni, L., & Bäckman, L. (2004). Apolipoprotein E and cognitive performance: A meta-analysis. *Psychology and Aging, 19*, 592–600. doi:10.1037/0882-7974.19.4.592
- Smith, J., & Infurna, F. J. (2011). Early precursors of later health. In K. L. Fingerhut, C. A. Berg, J. Smith, & T. C. Antonucci (Eds.), *Handbook of lifespan development* (pp. 213–238). New York, NY: Springer.
- Soederberg Miller, L. M., & Lachman, M. E. (1999). The sense of control and cognitive aging: Toward a model of mediational processes. In T. Hess & F. Blanchard-Fields (Eds.), *Social cognition* (pp. 17–41). New York, NY: Academic Press. doi:10.1016/B978-012345260-3/50003-3
- Soldo, B. J., Hurd, M., Rodgers, W., & Wallace, R. (1997). Asset and health dynamics among the oldest old: An overview of the AHEAD

- study. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 52, 1–20. doi:10.1093/geronb/52B.Special_Issue.1
- Spiro, A., III, & Brady, C. B. (2008). Integrating health into cognitive aging research and theory: Quo vadis? In S. M. Hofer & D. F. Alwin (Eds.), *Handbook of cognitive aging: Interdisciplinary perspectives* (pp. 260–283). Thousand Oaks, CA: Sage. doi:10.4135/9781412976589.n16
- Spiro, A., III, & Brady, C. B. (2011). Integrating health into cognitive aging: Toward a preventive cognitive neuroscience of aging. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 66, i17–i25. doi:10.1093/geronb/gbr018
- Stampfer, M. J. (2006). Cardiovascular disease and Alzheimer's disease: Common links. *Journal of Internal Medicine*, 260, 211–223. doi:10.1111/j.1365-2796.2006.01687.x
- Stefanick, M. L., Mackey, S., Sheehan, M., Ellsworth, N., Haskell, W. L., & Wood, P. D. (1998). Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *The New England Journal of Medicine*, 339, 12–20. doi:10.1056/NEJM199807023390103
- Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society*, 8, 448–460. doi:10.1017/S1355617702813248
- Stuart, E. A. (2010). Matching methods for causal inference: A review and a look forward. *Statistical Science*, 25, 1–21. doi:10.1214/09-STS313
- Thorvaldsson, V., Skoog, I., Hofer, S. M., Börjesson-Hanson, A., Östling, S., Sacuiu, S., & Johansson, B. (2012). Nonlinear blood pressure effects on cognition in old age: Separating between-person and within-person associations. *Psychology and Aging*, 27, 375–383. doi:10.1037/a0025631
- Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine*, 29, 377–387. doi:10.1007/s10865-006-9056-5
- Valeri, L., & VanderWeele, T. J. (2013). Mediation analysis allowing for exposure–mediator interactions and causal interpretation: Theoretical assumptions and implementation with SAS and SPSS macros. *Psychological Methods*. Advance online publication. doi:10.1037/a0031034
- Verhaegen, P., Borchelt, M., & Smith, J. (2003). Relation between cardiovascular and metabolic disease and cognition in very old age: Cross-sectional and longitudinal findings from the Berlin Aging Study. *Health Psychology*, 22, 559–569. doi:10.1037/0278-6133.22.6.559
- Voelkle, M. C., Oud, J. H. L., Davidov, E., & Schmidt, P. (2012). An SEM approach to continuous time modeling of panel data: Relating authoritarianism and anomia. *Psychological Methods*, 17, 176–192. doi:10.1037/a0027543
- Wahlin, Å., MacDonald, S. W. S., de Frias, C. M., Nilsson, L.-G., & Dixon, R. A. (2006). How do health and biological age influence chronological age and sex differences in cognitive aging: Moderating, mediating, or both? *Psychology and Aging*, 21, 318–332. doi:10.1037/0882-7974.21.2.318
- Waldstein, S. R., Brown, J. R. P., Maier, J. K., & Katzel, L. I. (2005). Diagnosis of hypertension and high blood pressure levels negatively affect cognitive function in older adults. *Annals of Behavioral Medicine*, 29, 174–180.
- West, S. G., Duan, N., Pequegnat, W., Gaist, P., Des Jarlais, D. C., Holtgrave, D., . . . Mullen, P. D. (2008). Alternatives to the randomized controlled trial. *American Journal of Public Health*, 98, 1359–1366. doi:10.2105/AJPH.2007.124446
- White, S. M., Wójcicki, T. R., & McAuley, E. (2012). Social cognitive influences on physical activity behavior in middle-aged and older adults. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 67, P18–P26. doi:10.1093/geronb/gbr064
- Windsor, T. D., & Anstey, K. A. (2008). A longitudinal investigation of perceived control and cognitive performance in young, midlife, and older adults. *Aging, Neuropsychology, and Cognition*, 15, 744–763. doi:10.1080/13825580802348570
- Yaffe, K., Fiocco, A. J., Lindquist, K., Vittinghoff, E., Simonsick, E. M., Newman, A. B., . . . Harris, T. B. (2009). Predictors of maintain cognitive function in older adults: The Health ABC Study. *Neurology*, 72, 2029–2035. doi:10.1212/WNL.0b013e3181a92c36
- Yaffe, K., Kanaya, A., Lindquist, K., Simonsick, E. M., Harris, T., Shorr, R. I., . . . Newman, A. B. (2004). The metabolic syndrome, inflammation and risk of cognitive decline. *JAMA: Journal of the American Medical Association*, 292, 2237–2242. doi:10.1001/jama.292.18.2237
- Yaffe, K., Lindquist, K., Schwartz, A. V., Vitartas, C., Vittinghoff, E., Satterfield, S., . . . Harris, T. (2011). Advanced glycation end product level, diabetes, and accelerated cognitive aging. *Neurology*, 77, 1351–1356. doi:10.1212/WNL.0b013e3182315a56

Received January 25, 2013

Revision received May 1, 2013

Accepted May 8, 2013 ■