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The effect of age on fluid intelligence is fully mediated by physical health

Ingvar Bergman [a,*], Ove Almkvist [b,c]

[a] Department of Neurobiology, Care Sciences and Society, Division of Clinical Geriatrics, Karolinska Institutet, Karolinska University Hospital, Huddinge, Sweden
[b] Department of Neurobiology, Care Sciences and Society, Division of Alzheimer Neurobiology Center, Karolinska Institutet, Karolinska University Hospital, Huddinge, Sweden
[c] Department of Psychology, Stockholm University, Stockholm, Sweden

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ABSTRACT

The present study investigated the extent to which the effect of age on cognitive ability is predicted by individual differences in physical health. The sample consisted of 118 volunteer subjects who were healthy and ranging in age from 26 to 91. The examinations included a clinical investigation, magnetic resonance imaging (MRI) brain neuroimaging, and a comprehensive neuropsychological assessment. The effect of age on fluid IQ with and without visual spatial praxis and on crystallized IQ was tested whether being fully-, partially- or non-mediated by physical health. Structural equation analyses showed that the best and most parsimonious fit to the data was provided by models that were fully mediated for fluid IQ without praxis, non-mediated for crystallized IQ and partially mediated for fluid IQ with praxis. The diseases of the circulatory and nervous systems were the major mediators. It was concluded from the pattern of findings that the effect of age on fluid intelligence is fully mediated by physical health, while crystallized intelligence is non-mediated and visual spatial praxis is partially mediated, influenced mainly by direct effects of age. Our findings imply that improving health by acting against the common age-related circulatory- and nervous system diseases and risk factors will oppose the decline in fluid intelligence with age.

1. Introduction

Normal cognitive aging is characterized by a primary decline of *fluid* intelligence, with only a minimal decline of *crystallized* intelligence (Schaie, 2005). Fluid intelligence is a concept that refers to intellectual abilities required for logical problem solving in novel situations, whereas crystallized intelligence refers to accumulated skills, knowledge and experience (Horn & Cattell, 1967). Some recent research has tended to strongly support the distinction between fluid and crystallized intelligence (Blair, 2006; Horn & McArdle, 2007).

Age-related disease has been found to be a significant predictor of low fluid ability in normal aging (e.g., Christensen et al., 1994) though its impact has typically been reported as small in cross-sectional research (e.g. Christensen et al., 1994; Jelicic, Jonker, & Deeg, 1999; Salthouse, Kausler, & Saults, 1990; van Boxtel et al., 1998; Verhaegen, Borchelt, & Smith, 2003; Zelinski, Crimmins, Reynolds, & Seeman, 1998) and in longitudinal research (Aarts et al., 2011; Verhaegen et al., 2003). For instance, van Boxtel et al. (1998) reported that medical conditions only accounted for up to 1.6% of the variation in cognitive performance. Quite contrary to these findings, substantial effects of age-related disease on a broad array of cognitive capacities have been reported by our own research group (Bergman, Blomberg, & Almkvist, 2007): Based on cross-sectional design and, with respect to medical and neuropsychological function, a carefully investigated sample of 118 healthy volunteers ranging in age from 26 to 91, health-related cognitive impairment predicted on average 10% of the variation in cognitive performance.

More substantial effects on cognitive test performance have also been associated with so-called biological markers of aging (i.e., forced expiratory volume, grip strength, and sensory functions such as vision, hearing, and vibration sense) in cross-sectional research (e.g., Anstey & Smith, 1999; Lindenberger & Baltes, 1994; Wahlin, MacDonald, deFrias, Nilsson, & Dixon, 2006) and in longitudinal research (Valentijn et al., 2005). Such markers of aging have been reported as being directly affected by physical health

* Corresponding author at: Trafikmedicinskt Centrum S31, Karolinska University Hospital, S-14186 Stockholm, Sweden. Tel.: +46 8585 86416; fax: +46 8585 86490.
E-mail address: ingvar.bergman@karolinska.se (I. Bergman).

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and as mediating age-related variance in cognitive tests (Anstey & Smith, 1999). Other factors reported to be associated with deterioration of test performance are exposure to free radicals (Droge, 2002), presence of apoliproprotein E genotype ε4 (Deary et al., 2002), abnormalities associated with Alzheimer's disease such as the presence of senile plaques and neurofibrillary tangles (Gómez-Isla & Hyman, 2003), and physical and mental inactivity (Davenport, Hogan, Eskes, Longman, & Poulin, 2012; Jak, 2012).

However, the cross-sectional designs in previous research on cognitive aging showing small effects of physical health on cognitive performance, as well as the cross-sectional design in our own study, did not allow for differentiation between shared and unique age- and health-related variance, or in other words, the establishment of the degree to which age has direct effects on cognitive abilities vis-à-vis indirect effects mediated by physical health. This is important because if the relationship between physical health and cognitive performance is entirely explained by chronological age, then it may be argued that the association is a spurious statistical finding (Salthouse, Hambrick, & McGuthry, 1998). Hofer and Sliwinski (2001) have elaborated on this critique and shown that cross-sectional study designs imply a positive bias toward finding significant associations among variables when there is variation in age within the sample and the key variables of interest are associated with age. This means that in simple bivariate prediction (e.g., by use of multiple regression analyses), the shared age-related variance among factors associated with cognitive performance may in part be due to a statistical artifact in age-related mean trends.

To avoid such bias from age-related mean trends, Hofer and Sliwinski (2001) and Hofer, Flaherty, and Hoffman (2006) have suggested age-homogenous or sequential narrow-age cohort designs (i.e., multiple age-homogenous groups). Ideally, age is a constant in age-homogenous groups, but in narrow-age cohort samples with some within-group age variance, the influence of mean trends on estimates of association can be eliminated by partialing for age within groups. As the authors point out, however, this approach is not making full use of the available data, in that information regarding the changing patterns of covariation between processes across time is lost.

Moreover, it is well-established that impaired brain integrity may explain important amounts of individual variation in cognitive tests (e.g., Gunning-Dixon & Raz, 2000; Machner, Sprenger, Kompf, & Heide, 2005; Mungas et al., 2002; Vermeer et al., 2003). It has also been shown that even subclinical cardiovascular disease or cardiovascular risk factors, as well as e.g. subclinical vitamin B12 levels, has been associated with decrease in cognitive performance (Leritz, McGlinchey, Kellison, Rudolph, & Milberg, 2011; Moore et al., 2012; Saxton et al., 2000). However, comprehensive objective examination of health and brain imaging has typically not been included in the same study (e.g., those referred to in the first paragraph above), leaving common cerebrovascular and central nervous system (CNS) changes in normal aging undetected.

Spiro and Brady (2008) have called attention to the problem of undiagnosed diseases and risk factors in cognitive aging research. They argue exhaustively for an overarching hypothesis holding that the effects of age on cognitive ability are related, at least in part, to vascular disease and its risk factors that selectively affect frontal system function (that is assumed critical for fluid ability; see e.g., Leahy, Suchy, Sweet, & Lam, 2003). The authors base their hypothesis upon their own and others research on the effects of age, vascular risk factors (e.g., hypertension) and vascular disease on cognitive performance (e.g., Elias et al., 2003, 2004; Raz, Rodrigue, & Acker, 2003; Waldstein, Giggey, Thayer, & Zonderman, 2005), research on vascular cognitive impairment (e.g., Bowler, 2005; Hachinski et al., 2006), and research on vascular dementia (e.g., Roman et al., 1993).

Based on this theoretical foundation, we re-analyzed our cross-sectional and age-heterogeneous clinical data from the previous study (Bergman et al., 2007). The sample is rather unique in that it includes the combination of MRI of the brain and a comprehensive objective examination of health. We regarded the sample too small for stratified analyses, so the cross-sectional design was retained.

The objective was to investigate the extent to which the effect of age on cognitive ability is predicted by individual differences in physical health. To counteract the problem of confusion with mean age trends, we increased the sophistication of the statistical analyses by applying Structural Equation Modeling (SEM). It makes full use of the covariance matrix for available data and allows for a shift of focus from simple bivariate prediction to mediational relationships (Kelloway, 1998). A three-factor cognitive model (fluid IQ with visual spatial praxis, fluid IQ without visual spatial praxis and crystallized IQ) was hypothesized based on the finding reported in our previous study that age and health dissociate with regard to cognitive function affected. Praxis refers to the ability to plan and then execute movement (The free dictionary by Farlex, 2013) and visual spatial praxis is required by tasks combining manual and visual spatial elements. Three alternative structural models in nested relationships were analyzed, where the effect of age on cognitive ability was tested whether being fully-, partially- or non-mediated by physical health, with the expectation that it is at least partially mediated for fluid IQ (i.e., according to the hypothesis by Spiro & Brady, 2008).

2. Methods

2.1. Participants

In all 131 individuals were recruited as healthy subjects between 1987 and 1996 to volunteer as control subjects at the Geriatric Clinic, Karolinska University Hospital at Huddinge, Stockholm, or the Geropsychiatric ward, St. Görans Hospital in Stockholm. They were respondents to a call on five occasions (regarding two studies on clinical trials, one on Alzheimer's disease, one on optimal health and one study on car driving ability). All individuals were given a comprehensive medical examination (see below), which resulted in the exclusion of 13 individuals (7 with a history of significant head trauma, 5 with dyslexia, and one with a history of evident brain infarction).

The remaining 118 individuals (48 women and 70 men) had an age range from 26 to 91 years with an average of 69.3 (±12.8) years. This was a negative age distribution, though not to an extreme (skewness = −1.5; kurtosis = 2.1). The level of formal education ranged from 6 to 19 years, with an average of 10.4 (±3.2) years. That was significantly higher than the national mean of approximately 8.5 years for corresponding generations and age distribution ($p < 0.001$; Statistics Sweden, 1995). Age and education were scored as continuous variables, while gender was scored as 0 for man and 1 for woman.

2.2. Health assessment

The medical examination was based on a comprehensive objective assessment of physical health. It included the following elements: self-reports on medical history and present health status, coronary and pulmonary examination, blood-pressure test ($n = 112$), neurological status ($n = 108$), electrocardiogram (ECG; $n = 73$), and Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975; $n = 106$). Laboratory analyses were carried out for blood and cerebrospinal fluid ($n = 42$). Brain neuroimaging was conducted using T1 and T2 weighted MRI