Available online at www.sciencedirect.com
ScienceDirect
Personality and Individual Differences 42 (2007) 1551–1559
PERSONALITY AND INDIVIDUAL DIFFERENCES
www.elsevier.com/locate/paid

Does childhood intelligence predict variation in cognitive change in later life?

Victoria J. Bourne [a,*], Helen C. Fox [b], Ian J. Deary [c], Lawrence J. Whalley [b]

[a] *School of Psychology, University of Dundee, Scotland, UK*
[b] *Department of Mental Health, University of Aberdeen, Scotland, UK*
[c] *Department of Psychology, University of Edinburgh, Scotland, UK*

Received 24 April 2006; received in revised form 13 October 2006; accepted 26 October 2006
Available online 13 December 2006

Abstract

Lower childhood cognitive ability may be a risk factor for greater cognitive decline in late life and progression to dementia. To assess variation in age-related cognitive change, it is helpful to have valid measures of cognitive ability from early life. Here, we examine the relation between childhood intelligence and cognitive change in later life in two samples, one born in 1921 and the other in 1936. All participants completed the same test of mental ability (one of the Moray House Test series) at age about 11 years, and were re-examined on Raven's Progressive Matrices at age 77 (1921-born) or age 64 (1936-born). Where possible, the 1921 sample was re-tested at the age of about 80 years old and the 1936 sample re-tested at about 66 years. After taking into account various covariates, including sex, education and occupation, childhood intelligence was a significant predictor of cognitive change in later life. Results were in the direction that participants with lower childhood mental ability experienced relatively greater cognitive decline, whereas those of higher childhood mental ability showed improved performance. This result suggests that higher premorbid cognitive ability is protective of decline in later life.

Keywords: Ageing; Childhood cognitive ability; Cognitive decline

* Corresponding author. Tel.: +44 1328 384 617.
E-mail address: v.bourne@dundee.ac.uk (V.J. Bourne).

0191-8869/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.paid.2006.10.030

1. Introduction

Understanding the possible determinants of individual differences in age-related cognitive change is important. Cognitive decline can impact greatly upon an individual's life, leading to reduced independence, lower quality of life (Logsdon, Gibbons, McCurry, & Teri, 2002) and increased risk of dementia and death (Hui et al., 2003). Cognitive ability is relatively stable and life-long. Indeed, some research has suggested that across over 60 years, about 50% of the variance in cognitive ability may be stable whereas the remaining 50% may represent change and measurement error (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). There are important individual differences in people's cognitive change with age: With regard to their rank order, some improve, some decline and some stay about the same. The sources of these individual differences in age-related cognitive change are not fully understood, but are highly relevant to understanding how best cognitive abilities may be retained even into advanced old age.

One possible contributor to variation in age-related cognitive change is the initial level of cognitive ability itself. Thus, several papers have asked the question: "Is age kinder to the initially more able?" (see Deary, Starr, & MacLennan, 1999, for a review and data on this question). One problem in addressing this research question, however, is gaining a valid measure of cognitive ability prior to old age. A number of methods have been developed that can 'estimate' premorbid cognitive ability (e.g., NART) and these have been frequently used to estimate change in cognitive ability. However, such methods are not without their limitations. Some studies have shown that NART performance can be impaired in dementia patients (e.g., Cockburn, Keene, Hope, & Smith, 2000). Also, while such methods aim to provide an estimate of cognitive ability in earlier adult life, there is evidence that various lifestyle factors (e.g., smoking, alcohol consumption, exercise, diet) may affect cognitive abilities, even in early adulthood (see Gottfredson, 2004). Consequently, the earliest possible direct measure of cognitive ability is likely to provide the least contaminated estimate of premorbid cognitive ability. A direct examination of the relation between cognitive ability from early life and age-related cognitive decline would allow the related question of the "cognitive reserve hypothesis of cognitive ageing" to be tested in a manner that is not possible with other data sets. This hypothesis proposes that adults with higher initial cognitive ability are better able to compensate for the effects of ageing and dementia (Stern, 2003). The cognitive reserve hypothesis predicts that individuals of higher childhood cognitive ability will experience less cognitive decline in later life.

In order to achieve a direct examination of the relation between childhood mental ability and the amount of cognitive decline, it is preferable to obtain an accurate measure of childhood ability from earlier in life. Few studies of older people have cognitive data from early life. Some have used estimates of prior ability and have found evidence that people with lower initial ability decline more in verbal ability in old age (Deary et al., 1999). The "Nun study" (Snowdon et al., 1996) found that higher linguistic ability in early adulthood was associated with higher cognitive ability in later life and lower incidence of dementia. A follow-up study of the UK 1946 birth cohort examined the relation between cognitive ability at age 15 years and cognitive decline from 43 to 53 years (Richards, Shipley, Fuhrer, & Wadsworth, 2004). They found that childhood mental ability was predictive of decline, with participants of higher childhood ability experiencing less cognitive decline in later adulthood. Although Richards et al. found a relation between cognitive ability in adolescence and cognitive decline in mid-life; their sample was re-examined at an age

when age-related cognitive decline would be slight in comparison to an older sample. Longer delays between early and later ability measurements will enable the relation between childhood cognitive ability and variation in cognitive change in later adulthood and old age (i.e., 65 years and older) to be examined.

The Aberdeen 1921 longitudinal follow-up study obtained cognitive measures at about age 11 years and in old age. Reports from this sample have provided evidence for the relative stability of mental ability from childhood to old age (Deary et al., 2000). Lower childhood intelligence predicted increased risk of early death (Whalley & Deary, 2001), psychiatric contact (Walker, McConville, Hunter, Deary, & Whalley, 2002) and late onset, but not early onset, dementia (Whalley et al., 2000). In the present study, the Aberdeen 1921 sample was used to examine the relation between childhood intelligence and variation in cognitive change in old age. A second sample, born in 1936, also completed tests of mental ability at age 11 years and returned for re-examination in later adulthood when cognitive ability was re-examined. For the 1921 sample, re-examination occurred at ages about 77 and about 80 years. The 1936 sample was re-examined at ages about 64 and 66 years.

2. Methods

Study participants all took part, at age 11, in the Scottish Mental Surveys 1932 or 1947 (SMS32 or SMS47; see Deary, Whiteman, Starr, Whalley, & Fox, 2004) and completed the Moray House Test of general intelligence. The Moray House Test comprised 71 items including arithmetic, reasoning, spatial items and word classification. The maximum possible score was 76. The Moray House Test is highly correlated (at about $r = 0.8$) with the Stanford–Binet intelligence test. These archived mental test results were later matched with health registers by name, sex and date of birth to approach people as potential participants in this longitudinal study of ageing. 235 participants were recruited from the SMS32 sample and 506 from the SMS47 sample. SMS32 participants were re-examined twice, at the ages of about 77 and 80 years old with a mean interval of 34 months and 14 days (SD = 3.5 months). SMS47 participants were re-examined after a two-year interval from age about 65 to about 67 years with a mean interval of 25 months and 21 days (SD = 4.1 months). At each re-examination, participants completed the Mini Mental State Examination, which is widely used as a screening test for dementia (Folstein, Folstein, & McHugh, 1975). These scores were used to exclude possible dementia cases from some analyses. Participants also completed Raven's Progressive Matrices (RPM; Raven, Court, & Raven, 1977), a test of nonverbal reasoning and intelligence. RPM showed high test–retest reliability ($r = .8$, $p < .001$).

The subjects of this investigation were 91 participants (39%) from the SMS32 sample (46 men, 45 women) and 349 participants (69%) from the SMS47 sample (166 men, 183 women) who provided complete data for the analyses. The difference in sample size between initial recruitment and testing is due to either attrition between waves (e.g., due to illness, death or moving away from the area) or inability to complete RPM at both waves of testing (e.g., due to fatigue or lack of cognitive capability). From the 1921 birth sample 59 people (25%) refused to attend the second wave of testing without providing a reason, 5 people (2%) did not attend as they had moved out of the area, 39 people (17%) had died, 12 people (5%) did not attend due to personal circumstances and 29 people (12%) were unable to provide complete data sets. In the 1936 birth sample 69 people

(14%) refused to attend the second wave of testing without providing a reason, 20 people (4%) did not attend as they had moved out of the area, 17 people (3%) had died, 19 people (4%) did not attend due to personal circumstances and 32 people (6%) were unable to provide complete data sets.

Data were analysed using multiple linear regression, with RPM at wave two as the outcome variable. RPM at wave one was the first variable entered into the model. The second block comprised a number of covariates, entered simultaneously, that may be implicated in cognitive ageing and/or dementia: sex (Deary et al., 2004), years of education (Staff, Murray, Deary, & Whalley, 2004), highest occupational status (Li, Wu, & Sung, 2002), whether they were a smoker at wave one of testing (Whalley, Fox, Deary, & Starr, 2005) and units of alcohol consumed a week at wave one of testing (Richards, Hardy, & Wadsworth, 2005). Cohort (SMS32 or SMS47) and interval between testing session in days were also included as possible covariates. Childhood cognitive ability was then entered into the model in a separate block. By running the analysis in this way, the variance in RPM at wave two that is accounted for by performance on RPM at wave one (i.e., the change in cognitive ability from wave one to wave two) is analysed within the first block and possible covariates are taken into account in the second block. Significant findings in the third block can subsequently be interpreted as predictors of change in cognitive ability from wave one to wave two, after statistically controlling for any variance explained by the covariates.

3. Results

Descriptive statistics of the measures can be seen in Table 1. A summary of the regression analysis is presented in Table 2. RPM at wave one was a significant predictor of RPM at wave two

Table 1
Demographic and cognitive characteristics of the two samples, born in either 1921 or 1936

	Men	Women	Total
1921 Birth sample			
N	46	45	91
MHT (11 years)	42.2 (13.3)	43.1 (10.6)	42.6 (12.0)
RPM (77 years)	31.3 (8.7)	29.1 (8.0)	30.2 (8.4)
MMSE (77 years)	28.5 (1.7)	28.9 (1.2)	28.7 (1.5)
RPM (80 years)	30.6 (8.3)	29.3 (8.3)	30.0 (8.2)
MMSE (80 years)	28.1 (1.8)	28.4 (1.5)	28.3 (1.7)
Change in RPM score	−.70 (6.7)	.18 (5.7)	−.26 (6.2)
1936 Birth sample			
N	166	183	349
MHT (11 years)	43.2 (12.6)	44.6 (12.0)	44.0 (12.3)
RPM (64 years)	38.0 (7.7)	36.2 (8.7)	37.0 (8.3)
MMSE (64 years)	29.0 (1.4)	29.0 (1.3)	29.0 (1.4)
RPM (66 years)	38.4 (8.1)	36.4 (8.7)	37.4 (8.5)
MMSE (66 years)	28.8 (1.4)	28.9 (1.2)	28.8 (1.3)
Change in RPM score	.45 (5.2)	.26 (4.9)	.35 (5.0)

All scores are expressed as means (SD) of raw scores. Maximum score on the Moray House Test (MHT) was 76, on the Mini Mental State Examination (MMSE) was 30, and on Raven's Progressive Matrices (RPM) was 60.

Table 2
Findings from the multiple regression analysis

	β	t	p	Block significance
Block one:				
RPM at wave one	.842	29.80	<.001	$F(1,433) = 888.3$, $p < .001$
Block two:				
Cohort	1.660	1.94	.053	$F(7,426) = 2.9$, $p = .006$
Sex	−.517	−.98	.326	
Interval (days)	.001	−.47	.640	
Years of education	.278	1.98	.048	
Occupation	−.218	−1.60	.109	
Units of alcohol per week	.019	−.59	.553	
Smoker (yes/no)	−.162	−.25	.804	
Block three:				
Cognitive ability age 11 years	.129	5.29	<.001	$F(1,425) = 28.0$, $p < .001$

RPM = Raven's Progressive Matrices.

($\beta = .84$, $p < .001$) accounting for 67.2% of the variance. Within the covariates block only years of education ($\beta = .28$, $p = .048$) was significant, cohort approached significance ($\beta = 1.7$, $p = .053$) and all other covariates were not significant ($p > .1$ for all). The covariates accounted for a further 1.5% of the variance. Participants with fewer years of education tended to show greater cognitive decline and individuals from the 1921 birth sample showed greater decline than those from the 1936 birth sample. Childhood cognitive test score was entered into the third block and was a significant predictor ($\beta = .13$, $p < .001$) explaining a further 2% of the variance. Because the variance explained by performance on RPM at wave one has already been taken into account in the fist block, this third block can be interpreted in terms of childhood cognitive ability predicting change in cognitive ability from wave one to wave two. There is a positive relationship between the two variables (evident in the β coefficient). As such, with each increase of one point on the MHT, an individual's performance on RPM at wave two would be expected to increase by .13, over and above the variance explained by RPM at wave one. Therefore, the higher an individual's childhood cognitive ability, the greater their improvement in performance from wave one to wave two. In order to examine whether this relationship was apparent in both cohorts, partial correlations between childhood cognitive ability and the raw change score, controlling for each of the variables controlled for in the regression analysis, were conducted for each of the cohorts separately. A significant correlation was found for both cohorts (SMS32: $r(82) = .315$, $p = .003$; SMS47: $r(335) = .231$, $p < .001$) indicating that the relationship between childhood cognitive ability and change in cognitive ability in later life is true of both samples, although stronger in the SMS32 sample.

It is possible that our findings may have been inflated by the inclusion of possible dementia cases or by individuals who have shown dramatic cognitive decline between the waves of testing. Childhood cognitive ability is predictive of increased risk of dementia (Whalley et al., 2000). It is possible that the relationship we have identified may not exist for all participants, but rather only in more extreme cases of cognitive decline. Does the relationship between childhood cognitive ability and change in cognitive performance in later life remain for participants who show no signs

of dementia? In order to address this possibility, we ran the analysis again excluding any possible dementia cases.

Five participants had MMSE scores less than 24, indicating possible dementia and three participants had outlying scores. One participant fell into both of these groups; hence seven participants (six males and one female) may have skewed our data. The analysis was re-run excluding these participants. Childhood cognitive ability was still identified as a significant predictor ($\beta = .11$, $p < .001$). Both analyses were also run using raw change score as the outcome variable. Again, childhood cognitive ability was a significant predictor when including all participants ($\beta = .13$, $p < .001$) and when excluding the seven outliers ($\beta = .11$, $p < .001$). By analysing the data in this way, the relationship between childhood cognitive ability and change in cognitive performance in later life can be explored further. Individuals of lower childhood cognitive ability tended to show cognitive decline from wave one to wave two (i.e., a negative change score) and individuals of higher childhood cognitive ability tended to show improved cognitive performance (i.e., a positive change score).

4. Discussion

We found a significant relation between lower childhood mental ability and change in cognitive ability in later life. People of higher childhood intelligence showed improved cognitive performance, whereas those of lower childhood intelligence experienced cognitive decline. Thus, we did find that age is kinder to the initially more able (Deary et al., 1999). Other studies have also demonstrated a relation between cognitive ability in earlier life and variation in cognitive change in later life (Richards et al., 2004; Snowdon et al., 1996). This inverse relation was present in both samples, 15 years apart in age. It is important to acknowledge that not all participants will have experienced decline. Some participants will have experienced decline, some will have remained stable and others may have shown improved performance. Inspection of Table 1 shows only a small mean change in performance. This may be explained by such individual differences in change in cognitive ability. Our results may reflect a combination of age-related cognitive decline in some participants and practice effects in others (see Ferrer, Salthouse, Stewart, & Schwartz, 2004; Rabbitt, Diggle, Smith, Holland, & McInnes, 2001). That is we cannot choose between childhood IQ being associated with a greater practice effect or less cognitive decline, or a combination of both. It is not possible to separate and quantify these two processes with our data set; however this issue clearly warrants further consideration.

The regression analysis showed that individuals from the SMS32 sample experienced greater decline than the SMS47 sample. This result is most likely to be because the SMS32 sample was older. A number of studies have demonstrated that cognitive decline is non-linear, with accelerated decline occurring from the age of about 65–70 years (Finkel, Pedersen, Plomin, & McClearn, 1998, 2003; McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; Verhaeghen & Salthouse, 1997). It is likely that some of the SMS47 participants had not yet begun to experience age related cognitive decline. As such, when they reach the age of 77 years, it would be predicted that they would show a comparable degree of decline over the following three years as the SMS32 sample did in this analysis. Further, cognitive decline may have occurred prior to wave one of testing. This is particularly relevant to the SMS32 sample. Since we used wave one performance effectively

as a baseline for later cognitive change, any decline prior to wave one may have biased our findings.

Why are the initially more able cognitively protected into later life? There are two possible accounts. First, it may be that those with higher cognitive ability have a greater "cognitive reserve" and are more able to compensate for the social, psychological and/or neurobiological effects of ageing and dementia (Stern, 2003). The relation identified in the present study may be interpreted as resulting from increased neural plasticity in individuals with high childhood mental ability, and consequently increased ability to compensate for the neuropathological effects of ageing and cognitive decline. Studies investigating a neurobiological basis of cognitive reserve point to the possible functional re-organisation, in old people, of those neural networks used by young adults to complete the same tasks (Stern et al., 2005). An alternative account suggests that use-dependent processes afford cognitive protection: that is one must "use it or lose it" (e.g., Christensen & Henderson, 1991). In this way, our findings may have arisen because those with high childhood mental ability may have received more education and worked at more intellectually demanding tasks (Staff et al., 2004). These individuals may, by these means, have acquired a repertoire of life skills that support a mentally stimulating and socially engaged life style, thus "exercising" their mental faculties more over their lifetime than those who were initially less cognitively able. Such attributes might even contribute to better cognitive test performance and improvement between the two testing phases. It is not possible to distinguish between these two accounts with the current data set and analyses.

One limitation of this study is that cognitive performance in old age was just measured at two time points. This poses a possible problem as it may be difficult to adequately distinguish baseline performance from change in cognitive performance. A statistical model which included more than two waves of cognitive testing in old age might provide greater insights into the relationship between childhood cognitive ability and change in cognitive ability in old age. Although this analysis is not possible with the current data set, it is anticipated that such analyses may be possible in the future. A further advantage of including more than two time points would be that non-linear models could be examined. Our findings are limited to analysis of linear change in cognitive performance. Various studies have suggested that cognitive decline is better represented as non-linear (e.g., Finkel, Reynolds, McArdle, Gatz, & Pedersen, 2003, 2005; Lovden, Ghisletta, & Lindenberger, 2004; McArdle et al., 2004; Rabbitt et al., 2001). Again, such analyses may be possible in the future following further waves of data collection. While the current analysis provides a preliminary and unique insight into the relationship between childhood cognitive ability and change in cognitive abilities in later life, it is obvious that deeper understanding will come with further waves of testing and more complex methods of analysis.

Longitudinal studies of cognitive ageing are typically subject to an important source of bias: retention of volunteer participants. Our study is certainly no exception. Indeed, we were only able to analyse 39% of the data from the SMS32 cohort and 69% of the data from the SMS47 cohort. The restricted number of participants that we were able to include in the analysis, particularly in the SMS32 cohort, is a considerable limitation of this study. Whether participants return to repeat waves of testing is influenced by a number of factors including general health, cognitive ability, social integration and ease of returning for re-examination. These factors serve to increase the representation within follow up samples of participants of higher cognitive ability. Indeed, many longitudinal studies of cognitive ageing have found that participants who 'drop out' are of lower

cognitive ability (e.g., Matthews, Chatfield, & Brayne, 2006). It is likely that a similar pattern would be identified in our data. Our sample may not, therefore, have included people of lower cognitive ability, who also may experience more extreme levels of cognitive decline. Given these potential limitations, it is possible that our estimates of the relation between childhood cognitive ability and cognitive decline in later life may therefore be an underestimate.

One particular strength of this paper is that the relationship between childhood cognitive ability and change in cognitive ability in later life was found even after accounting for the variance explained by frequently cited risk factors for cognitive ageing. Whilst we attempted to control for a number of possible covariates, there are still a number of other possible factors that could influence cognitive decline in later life. A further issue to consider is whether the relationship between childhood IQ and cognitive decline in later life is a direct one, or whether childhood intelligence might influence other factors. These include health behaviours (e.g., smoking, alcohol consumption, diet), which may subsequently impact on cognitive variation in later life (Gottfredson, 2004). Poor physical health has also been identified as a risk factor for cognitive decline (van Hooren et al., 2005). In conclusion, whilst estimators of prior cognitive ability such as education and occupation are often described as predictors of cognitive decline in later life, our study demonstrates that childhood cognitive ability makes an important additional contribution to cognitive ageing.

References

Christensen, H., & Henderson, A. S. (1991). Is age kinder to the initially more able? A study of eminent scientists and academics. *Psychological Medicine, 21*, 935–946.

Cockburn, J., Keene, J., Hope, T., & Smith, P. (2000). Progressive decline in NART score with increasing dementia severity. *Journal of Clinical and Experimental Neuropsychology, 22*(4), 508–517.

Deary, I. J., Starr, J. M., & MacLennan, W. J. (1999). Is age kinder to the initially more able? Differential aging of verbal ability in the HOPE study. *Intelligence, 26*, 357–375.

Deary, I. J., Whalley, L. J., Lemmon, H., Crawford, J. R., & Starr, J. M. (2000). The stability of individual differences in mental ability from childhood to old age: follow-up of the 1932 Scottish mental survey. *Intelligence, 28*(1), 49–55.

Deary, I. J., Whiteman, M. C., Starr, J. M., Whalley, L. J., & Fox, H. C. (2004). The impact of childhood intelligence on later life: following up the Scottish mental surveys of 1932 and 1947. *Journal of Personality and Social Psychology, 86*(1), 130–147.

Ferrer, E., Salthouse, T. A., Stewart, W. F., & Schwartz, B. S. (2004). Modeling age and retest processes in longitudinal studies of cognitive abilities. *Psychology and Aging, 19*(2), 243–259.

Finkel, D., Pedersen, N. L., Plomin, R., & McClearn, G. E. (1998). Longitudinal and cross-sectional twin data on cognitive abilities in adulthood: the Swedish adoption/twin study of aging. *Developmental Psychology, 34*(6), 1400–1413.

Finkel, D., Reynolds, C. A., McArdle, J. J., Gatz, M., & Pedersen, N. L. (2003). Latent growth curve analyses of accelerating decline in cognitive abilities in late adulthood. *Developmental Psychology, 39*(3), 535–550.

Finkel, D., Reynolds, C. A., McArdle, J. J., & Pedersen, N. L. (2005). The longitudinal relationship between processing speed and cognitive ability: genetic and environmental influences. *Behavior Genetics, 35*(5), 535–549.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini–Mental state a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Gottfredson, L. S. (2004). Intelligence: is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *Journal of Personality and Social Psychology, 86*(1), 174–199.

Hui, J. S., Wilson, R. S., Bennett, D. A., Bienias, J. L., Gilley, D. W., & Evans, D. A. (2003). Rate of cognitive decline and mortality in Alzheimer's disease. *Neurology, 61*(10), 1356–1361.

Li, C. Y., Wu, S. C., & Sung, F. C. (2002). Lifetime principal occupation and risk of cognitive impairment among the elderly. *Industrial Health, 40*, 7–13.

Logsdon, R. G., Gibbons, L. E., McCurry, S. M., & Teri, L. (2002). Assessing quality of life in older adults with cognitive impairment. *Psychosomatic Medicine, 64*(3), 510–519.

Lovden, M., Ghisletta, P., & Lindenberger, U. (2004). Cognition in the Berlin aging study (BASE): the first 10 years. *Aging Neuropsychology and Cognition, 11*(2–3), 104–133.

Matthews, F. E., Chatfield, M., & Brayne, C. (2006). An investigation of whether factors associated with short-term attrition change or persist over ten years: data from the medical research council cognitive function and ageing study. *BMC Public Health, 6*, 185.

McArdle, J. J., Ferrer-Caja, E., Hamagami, F., & Woodcock, R. W. (2002). Comparative longitudinal structural analyses of the growth and decline of multiple intellectual abilities over the life span. *Developmental Psychology, 38*(1), 115–142.

McArdle, J. J., Hamgami, F., Jones, K., Jolesz, F., Kikinis, R., Spiro, A., et al. (2004). Structural modeling of dynamic changes in memory and brain structure using longitudinal data from the normative aging study. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences, 59*(6), P294–P304.

Rabbitt, P., Diggle, P., Smith, D., Holland, F., & McInnes, L. (2001). Identifying and separating the effects of practice and of cognitive ageing during a large longitudinal study of elderly community residents. *Neuropsychologia, 39*(5), 532–543.

Raven, J. C., Court, J. H., & Raven, J. (1977). *Manual for Raven's Progressive Matrices and Vocabulary Scales*. London: HK Lewis & Co. Ltd.

Richards, M., Hardy, R., & Wadsworth, M. E. J. (2005). Alcohol consumption and midlife cognitive change in the British 1946 birth cohort study. *Alcohol and Alcoholism, 40*, 112–117.

Richards, M., Shipley, B., Fuhrer, R., & Wadsworth, M. E. J. (2004). Cognitive ability in childhood and cognitive decline in mid-life: longitudinal birth cohort study. *British Medical Journal, 328*(7439), 552–554B.

Snowdon, D. A., Kemper, S. J., Mortimer, J. A., Greiner, L. H., Wekstein, D. R., & Markesbery, W. R. (1996). Linguistic ability in early life and cognitive function and Alzheimer's disease in late life. Findings from the Nun study. *Journal of the American Medial Association, 275*(7), 528–532.

Staff, R. T., Murray, A. D., Deary, I. J., & Whalley, L. J. (2004). What provides cerebral reserve? *Brain, 127*, 1191–1199.

Stern, Y. (2003). The concept of cognitive reserve: a catalyst for research. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 589–593.

Stern, Y., Habeck, C., Moeller, J., Scarmeas, N., Anderson, K. E., Hilton, H. J., et al. (2005). Brain networks associated with cognitive reserve in healthy young and old adults. *Cerebral Cortex, 15*(4), 394–402.

van Hooren, S. A. H., Valentijn, S. A. M., Bosma, H., Ponds, R. W. H. M., van Boxtel, M. P. J., & Jolles, J. (2005). Relation between health status and cognitive functioning: a 6-year follow-up of the Maastricht aging study. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences, 60*, 57–60.

Verhaeghen, P., & Salthouse, T. A. (1997). Meta-analyses of age-cognition relations in adulthood: estimates of linear and nonlinear age effects and structural models. *Psychologial Bulletin, 122*(3), 231–249.

Walker, N. P., McConville, P. M., Hunter, D., Deary, I. J., & Whalley, L. J. (2002). Childhood mental ability and lifetime psychiatric contact – a 66-year follow-up study of the 1932 Scottish mental ability survey. *Intelligence, 30*(3), 233–245.

Whalley, L. J., & Deary, I. J. (2001). Longitudinal cohortstudy of childhood IQ and survival up to age 76. *British Medical Journal, 322*(7290), 819–822.

Whalley, L. J., Fox, H. C., Deary, I. J., & Starr, J. M. (2005). Childhood IQ, smoking, and cognitive change from age 11 to 64 years. *Addictive Behaviours, 30*(1), 77–88.

Whalley, L. J., Starr, J. M., Athawes, R., Hunter, D., Pattie, A., & Deary, I. J. (2000). Childhood mental ability and dementia. *Neurology, 55*(10), 1455–1459.