Stability and change in intelligence from age 11 to ages 70, 79, and 87: The Lothian Birth Cohorts of 1921 and 1936

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Alan J. Gow, Wendy Johnson, Alison Pattie, Caroline E. Brett, Beverly Roberts, John M. Starr and Ian J. Deary

University of Edinburgh, Edinburgh

Author note

Alan J. Gow, Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh; Wendy Johnson, Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh; Alison Pattie, Department of Psychology, University of Edinburgh; Caroline E. Brett, Department of Psychology, University of Edinburgh; Beverly Roberts, Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh; John M. Starr, Centre for Cognitive Ageing and Cognitive Epidemiology, Geriatric Medicine Unit, University of Edinburgh; Ian J. Deary Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh.

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Correspondence concerning this article should be addressed to Alan J. Gow, Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh, EH8 9JZ. E-mail: Alan.Gow@ed.ac.uk

Abstract

Investigating the predictors of age-related cognitive change is a research priority. However, it is first necessary to discover the long-term stability of measures of cognitive ability because prior cognitive ability level might contribute to the amount of cognitive change experienced within old age. These two issues were examined in the Lothian Birth Cohorts of 1921 and 1936. Cognitive ability data were available from age 11 years when the participants completed the Moray House Test No. 12 (MHT). The Lothian Birth Cohort 1936 (LBC1936) completed the MHT a second time at age 70. The Lothian Birth Cohort 1921 (LBC1921) completed the MHT at ages 79 and 87. We examined cognitive stability and change from childhood to old age in both cohorts, and within old age in the LBC1921. Raw stability coefficients for the MHT from 11-70, 11-79 and 11-87 years were .67, .66 and .51, respectively; and larger when corrected for range restriction in the samples. Therefore, minimum estimates of the variance in later-life MHT accounted for by childhood performance on the same test ranged from 26-44%. This study also examined, in the LBC1921, whether MHT score at age 11 influenced the amount of change in MHT between ages 79 and 87. It did not. Higher intelligence from early life was apparently protective of intelligence in old age due to the stability of cognitive function across the lifespan, rather than because it slowed the decline experienced in later life.

Keywords: cognitive ability, stability, change, cognitive aging

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There is increasing interest in identifying the determinants of successful aging and, in particular, the aging of mental functions (Kirkwood, Bond, May, McKeith, & Teh, 2008). This research is driven by the ever greater number of individuals in both developed and developing nations living into their 60s, 70s and beyond (Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat, 2005). Increasingly aged populations may incur higher healthcare and other service costs relative to those in the workforce. It is therefore incumbent upon researchers to identify areas in which individuals can be helped to age more successfully (Hertzog, Kramer, Wilson, & Lindenberger, 2009). Retaining cognitive abilities is central to living independently in old age, maintaining control over one's life and decisions, and sustaining an acceptable quality of life. As people age, however, there are observable changes in cognitive abilities.

Cognitive functions decline with age

Longitudinal studies have provided clear demonstrations of the way in which some cognitive abilities decline across time (Schaie, Willis, & Caskie, 2004; Hedden & Gabrieli, 2004; Salthouse, 2006). Nevertheless, "there is no uniform pattern of age-related changes across all intellectual abilities" (Schaie et al., 2004, p. 309); different domains of cognitive function begin to show marked decrements at different ages, and these declines occur at different rates. Abilities such as reasoning, processing speed and memory decline at earlier ages than those abilities representing the application of accrued knowledge, such as vocabulary. The latter are likely to decline after about 70 years of age, whilst processing speed has been shown to decline from the late 20s-early 30s.

In one of the landmark cognitive aging studies—the Seattle Longitudinal Study—noticeable declines were generally apparent across the range of diverse abilities tested when individuals reached 50-60 years old and, by the mid 70s, declines in all abilities were evident. Perceptual speed was found to exhibit almost linear decline from young adulthood onwards. Nevertheless, at age 81 "less than half of all observed individuals experienced reliable decremental change on a particular ability over the preceding seven years" (Schaie et al., 2004, p. 310). Therefore, whereas, in general, cognitive abilities decline with age, there is considerable individual variation in the extent to which this is experienced (Salthouse, 2006).

Individual variation in cognitive aging trajectories

This striking feature of cognitive aging—the marked degree of individual variation that exists—is of great interest. It is likely that a number of factors are responsible for these differences in cognitive ageing trajectories, including genetic influences and those from the environment (Christensen et al., 2004; Hertzog et al., 2009). A key goal is to determine if mutable factors accounting for these differences can be identified. Such factors could be promoted or discouraged as appropriate, or manipulated as part of delivered interventions to delay, reduce, or indeed reverse this decline.

There is a determined research effort to identify the factors that might be associated with the level of ability in later life, and the change in ability over time (Hertzog et al., 2009; Hendrie et al., 2006). Indeed, the National Institutes of Health in the United States formed a committee to evaluate critically the extant literature pertaining to the promotion of cognitive and emotional health in older adults. The report stated that "identifying the demographic, biological, and psychosocial factors

that can help people maintain or enhance their cognitive…health as they grow older becomes a major public health goal" (Hendrie et al., 2006, p. 13).

The problem of identifying such factors is not, however, straightforward. This is because mental ability shows large individual differences not only in level and change in later life but also throughout the lifespan. Previous estimates indicate that about 50% of the variance in later life ability is accounted for by the lifetime stability of cognitive ability (Deary, Whiteman, Starr, Whalley, & Fox, 2004). Thus, though the potentially malleable determinants of later life mental ability are the principal focus of much research, these cannot be identified definitively without knowing the extent to which later life abilities differ from those in earlier life. Ability in early life is the baseline from which change in later life takes place.

Early life cognitive ability and later ability and change

There is high stability of intellectual function from one occasion to the next across several decades (Riley, Snowdon, Desrosiers, & Markesbery, 2005; Deary et al., 2004). It is as yet unresolved, however, whether prior ability predicts the degree of *change* in cognitive functions over time in later life. Indeed, although it is 50 years since Owens (1959) asked the question "is age kinder to the initially more able?", and despite its being posed again in subsequent articles with the same title, it is an issue which still demands further exploration, not least because of the methodological difficulties involved in answering this question (Deary, MacLennan, & Starr, 1998). Owens's early findings suggested that those of higher ability had no advantage in terms of changes in the Army Alpha examination over a period of 30 years (Owens, 1959). This is in agreement with more recent work in which a Scottish cohort of individuals was followed longitudinally. Cognitive ability measured at age 11 was strongly associated with level of cognitive ability at ages 79 and 83 years (from a

battery of 3 cognitive tests), but it was not related to cognitive change observed across this 4-year period (Gow et al., 2008).

Results from similar types of study have, however, suggested that childhood ability does predict reduced cognitive decline over 10 years in midlife (Richards, Shipley, Fuhrer, & Wadsworth, 2004), or 2-3 years in the 60-70 age range (Bourne, Fox, Deary, & Whalley, 2007). The discrepancy may be explained partly by different tests and follow-up periods, although analytical methodology is also an important consideration. In Gow et al. (2008), the longitudinal data were analyzed using 2 methods that produced different results: linear regression suggested that childhood ability *was* related to 4-year cognitive change, in agreement with Richards et al. (2004) and Bourne et al. (2007). Yet, when more accurate growth curve modeling was applied to the dataset, childhood ability *was not* significantly related to cognitive change. Growth curve modeling is more appropriate for such longitudinal analyses as it allows a simultaneous examination of *level* and *change* in the outcome of interest, in this case cognitive function. In addition, growth curve modeling makes it possible to control the effects of demographic and lifestyle variables on both level and change in cognitive function, and to measure the correlation between them. But the largest barrier to resolving the issue as to whether early-life cognitive ability influences the trajectory of cognitive ability in later life is the lack of studies which have appropriate data.

Much as “the process of healthy aging cannot be understood without considering the entire human life history” (Westendorp & Wimmer, 2005, p. 420), so cognitive aging cannot be investigated optimally without a knowledge of the studied individuals’ cognitive ability histories. Because cognitive ability shows such large individual differences that are highly stable throughout the lifespan, a measure or

validated estimate of prior ability is required when one begins to look for predictors of the level of, and change in, later-life mental function. Prior ability may itself confound—or indeed precede and predict—other determinants of cognitive aging. Failing to control for this may allow spurious relations to be reported between a variable of interest and cognitive ability and/or change in old age. This is often termed reverse causation, in which an association is wholly or partly determined by the level of an antecedent (and often unmeasured) factor. For example, inflammatory processes have been implicated in age-related cognitive changes, and markers of inflammation, such as C-reactive protein, have been shown to predict poorer performance on tests of cognitive ability. However, when childhood ability was included in one such analysis, the cross-sectional associations between markers of inflammation and cognitive ability in old age were markedly attenuated and no longer significant (Luciano, Marioni, Gow, Starr, & Deary, 2009b). The stability of cognitive ability across the lifespan accounted for the later cognition-inflammation association (this, of course, does not preclude the possibility that inflammatory processes are themselves set in motion at an earlier point in time).

The present study

Studies that can account for the stability of cognitive abilities are rare; requirements include a valid early-life measure of cognitive ability, and an extended period across which stability and change can be assessed. In the current analysis, we examined the stability of an omnibus test of general intelligence—the Moray House Test No. 12—over a re-test period of up to 76 years using data from two similar cohorts. Participants in the Lothian Birth Cohort 1921 (LBC1921) completed the Moray House Test No. 12 on three occasions: at ages 11, 79 and 87 years. Participants

in the Lothian Birth Cohort 1936 (LBC1936) completed this Moray House Test on two occasions: at ages 11 and 70.

In an earlier report of the stability of the Moray House Test in the LBC1921, which suggested that about 43% of the variance in the later life test performance was accounted for by earlier performance, Deary and colleagues (2004, p. 135) reported:

> This is an important result to establish, because the current, intense interest in discovering the determinants of age-related cognitive change…must first be informed by the basic stability that exists in psychometric intelligence across the life span.

We extended this analysis by a further 8 years in the LBC1921, and replicated these estimates in a sample twice as large, albeit nine years younger at the first occasion of testing (70 versus 79 years old)—the LBC1936.

In addition to examining the stability of the Moray House Test over several decades, we examined cognitive stability and change within old age (79 to 87 years) in the Lothian Birth Cohort 1921, and the influence of prior ability (age 11) on the rate of change.

Method

Participants

Lothian Birth Cohort 1921. The recruitment and testing of the Lothian Birth Cohort 1921 (LBC1921) at waves 1 and 2 have been reported in detail previously (Deary et al., 2004; Gow et al., 2008; Deary, Whalley, & Starr, 2009b). In summary, the individuals recruited into the LBC1921 were all born in 1921 and had taken part in the Scottish Mental Survey 1932 when aged 11 (N = 87,498: Scottish Council for Research in Education, 1933). The LBC1921 study was initiated in 1999 and identified surviving participants of the Scottish Mental Survey 1932 from Edinburgh

and the surrounding areas, either through the Community Health Index or as volunteers replying to media calls. This initial wave of recruitment and testing ran from 1999-2001, during which time 550 individuals (234 men and 316 women) were tested individually at the Wellcome Trust Clinical Research Facility, Edinburgh, by two trained researchers (Deary et al., 2004).

For the 2nd wave (data from this wave are not reported in the current analysis but is described for completeness), all LBC1921 participants, except those who had withdrawn or were known to have died, were invited to participate. Of the 454 participants invited, 335 participants agreed to attend, and 321 were tested (145 men and 176 women). Testing was conducted from 2003-05 by two trained researchers in the Visual Psychophysics Laboratory at the Princess Alexandra Eye Pavilion, Royal Infirmary of Edinburgh (Gow et al., 2008).

The 3rd wave of testing, which ran from 2007-08, is reported here in detail for the first time. All LBC1921 participants who had completed both waves 1 and 2, excluding those who had withdrawn or were known to have died since wave 2, were invited to participate in the next stage of follow-up. The invitation letter was accompanied by a detailed information leaflet. Invitation letters were sent to 268 participants, in batches of about 20 at a time. Participants were then phoned a week later and given an opportunity to ask any questions about the clinic visit before an appointment was arranged. Transport was offered to bring them to and from the clinic, as required. In total, 196 participants were tested at the Wellcome Trust Clinical Research Facility, Edinburgh, by three trained researchers. Each participant was tested individually in a session lasting about 3.5-4 hours, including breaks (the cognitive testing element lasted about 30-40 minutes, with an additional 45 minutes for completion of the Moray House Test; all participants were given a scheduled

break of 20-30 minutes about halfway through the appointment, although participants were able to request more frequent breaks, as required). Participants who were unable to attend the clinic due to illness, infirmity, or caring for a partner, were offered a home visit. All the equipment used at the home visits was identical to that used at the clinic and participants were given the same battery of cognitive tests. Eleven subjects were visited at home.

Of the participants who were invited but who did not participate in wave 3, 42 were unable to come to the clinic or receive a home visit; 8 died over the course of the follow-up; contact was lost with 4 who were no longer living at their previous address; 3 who had been diagnosed with dementia or memory problems since the previous wave were withdrawn from the study; and 4 withdrew of their own accord. The participants who were unable to attend the clinic or receive a home visit were asked to complete a questionnaire booklet (also completed by the other participants prior to their appointments) and a Townsend disability scale. Twenty-nine completed questionnaires and 30 Townsend scales were received from these otherwise non-participants. The total number of LBC1921 participants interviewed at wave 3, including home visits, was 207; including those who were able to complete only the questionnaires, the response was 237 (109 men and 128 women).

The mean age of the LBC1921 when tested as children in the Scottish Mental Survey 1932 was 10.9 years old (*sd* = 0.3). In late adulthood, the follow-ups occurred at mean ages of 79.1 (*sd* = 0.6: wave 1), 83.4 (*sd* = 0.5: wave 2), and 86.6 years old (*sd* = 0.4: wave 3). For simplicity, these are referred to as ages 11, 79, 83 and 87 throughout. The mean follow-up time from wave 1 to wave 2 was 4.3 years (*sd* = 0.4), ranging from 3.1 to 5.8 years; from wave 2 to wave 3 was 3.2 years (*sd* = 0.4),

ranging from 2.1 to 4.5 years; and from wave 1 to wave 3 was 7.6 years (*sd* = 0.4), ranging from 6.7 to 8.9 years.

Lothian Birth Cohort 1936. The recruitment and testing of the Lothian Birth Cohort 1936 (LBC1936) is described extensively in an open-access article (Deary et al., 2007). To summarize, individuals born in 1936 who had participated in the Scottish Mental Survey 1947 when aged 11 (N = 70,805: Scottish Council for Research in Education, 1949) were identified and recruited into the LBC1936 from 2004-07. This was achieved by using the Community Health Index, and latterly, media advertisements. In total, 1091 relatively healthy participants (548 men and 543 women) were recruited and tested individually at the Wellcome Trust Clinical Research Facility, Edinburgh. A second wave of testing is due for completion in 2010.

The mean age of the LBC1936 when tested as children in the Scottish Mental Survey 1947 was 10.9 years old (*sd* = 0.3), and the first follow-up in old age was conducted at a mean age of 69.5 years old (*sd* = 0.8). These will be referred to as ages 11 and 70 throughout.

Procedure

At each clinic appointment, the LBC1921 and LBC1936 participants were asked to complete a battery of cognitive tests administered by trained psychology research associates, undergo a physical examination with a research nurse (at the LBC1921 wave 2 assessment the physical measures were taken by the psychology research staff), and provide demographic and other psychosocial lifestyle information (the latter often via self-report measures). Only the variables relevant to the current analysis are reported in detail. Further details can be obtained from: Deary et al., 2004; Deary et al., 2007; Gow et al., 2008; Deary et al., 2009b.

Moray House Test Number 12. The participants in the LBC1921 and LBC1936 had taken part in the nationwide Scottish Mental Surveys of 1932 and 1947, respectively. In both surveys, the Moray House Test Number 12 was used (Scottish Council for Research in Education, 1933; Scottish Council for Research in Education, 1949). It is a paper and pencil, group-administered test, which has a 45-minute time limit. The test has 71 numbered items, 75 items in total, of a variety of types: following directions (14 items), same-opposites (11), word classification (10), analogies (8), practical items (6), reasoning (5), proverbs (4), arithmetic (4), spatial items (4), mixed sentences (3), cypher decoding (2), and other items (4). The maximum possible score in the Moray House Test (MHT) is 76.

The MHT was validated in a sub-sample of 1000 children (500 boys and 500 girls) drawn from the Scottish Mental Survey 1932. This group completed the Stanford revision of the Binet-Simon Test (as standardized by Terman). Correlations between the MHT and Binet IQ were recorded as .80 for the boys and .76 for the girls, providing concurrent validity for the MHT (Deary et al., 2009b; Scottish Council for Research in Education, 1933).

The MHT was re-administered to the LBC1921 at ages 79 and 87 (waves 1 and 3 as described above) and to the LBC1936 at age 70. The same instructions and 45-minute time limit were used.

MMSE. The MMSE (Folstein, Folstein, & McHugh, 1975) is commonly used as a first-stage screen for dementia. It is a brief measure, and a score of less than 24 correct (out of 30) is often used as an indicator of potential dementia (Lezak, Howieson, & Loring, 2004). In this study, the MMSE was used for descriptive purposes only.

Demographics. At the first occasion of testing in the LBC1921 (similar information was obtained from the LBC1936 but is not reported in the current analysis), participants were asked to provide the number of years spent in full-time formal education; their main occupation to allow social class coding (see below); whether they were current, ex- or never-smokers; and the frequency, amount and type of alcohol currently consumed per week to allow the number of units of alcohol to be calculated. This variable was capped at 49 units per week (6 outliers above this were recoded accordingly).

Participants were asked to describe the highest occupational position achieved to allow a social class category to be assigned (using a year-appropriate coding system). Married women were asked to provide description of their husband's occupation and they were assigned the higher of their own or husbands' social class. For the LBC1921, social class was coded according to the 1951 Classification of Occupations (General Register Office, 1956), ranging from I (professional) to V (unskilled).

Statistical analyses

All analyses were conducted in PASW Statistics Version 17.0, except the growth curve modeling, which was carried out in Mplus Version 5. Due to the potential for restriction of range in the LBC samples, the MHT correlations were corrected following the description of Wiberg and Sundstrom (2009). Correction for range restriction used the standard deviation of the MHT in the restricted and unrestricted samples, and the MHT correlations in the restricted samples (see Wiberg and Sundstrom (2009), p. 4 for details). To examine later life cognitive change in the LBC1921, we implemented a latent difference score model which generated latent intercept and slope terms. This allowed the separate measurement of the effects of the

demographic variables and age-11 IQ on the overall level (intercept) of cognitive ability across both ages, and on change (slope) in cognitive ability from age 79 to age 87. We could also measure the correlation between intercept and slope. All participants who were present at age 79 but not at age 87 were included under the assumption that they were missing at random, using full information maximum likelihood.

Results

Descriptives

Both cohorts were relatively cognitively healthy, as shown by the MMSE means obtained (Table 1). On the first occasion of testing, 9 LBC1921 participants and 11 LBC1936 participants scored less than 24 (often used as an indicator of possible dementia). No participant scored below 18 in the LBC1921, or 20 in the LBC1936. The MMSE data are included in the LBC studies for descriptive purposes only to allow comparisons with other aged cohorts.

The LBC1921 participants had a mean of 10.9 years of education (*sd* = 2.5), and at age 79, the mean weekly alcohol consumption was 5.8 units (*sd* = 10.7). Of the 549 who provided information on their smoking status at 79 years, 40 participants (7.3%) were current smokers, 271 (49.4%) were ex-smokers and 238 (43.4%) had never smoked. The majority of participants were grouped in social classes 1-3: of the 548 with social class data, 129 participants (23.5%) were in social class 1 (the highest grouping), 183 (33.4%) were in 2, and 217 (39.6%) were in 3; social classes 4 and 5 consisted of 12 (2.2%) and 7 (1.3%) participants, respectively.

In Table 1, the raw MHT scores are listed for the LBC1921 (3 occasions) and the LBC1936 (2 occasions). When the groups completed the same test at 11 years old the individuals in the LBC1936 scored on average 2.6 points more (out of 76) than

those in the LBC1921. The LBC1921 and LBC1936 thus showed the same pattern as the populations from which they were drawn, whereby the average MHT score obtained in the SMS1947 was 36.74 (16.1) compared to 34.46 (15.5) in the SMS1932 (Scottish Council for Research in Education, 1949; Deary et al., 2009b): see the Discussion for further comment.

[Table 1 here]

Comparing performance on the MHT at age 11 to the first occasion it was repeated within old age, both cohorts showed significant improvement. For the LBC1921, the difference between average MHT scores at 11 and 79 years old was 12.8 points (46.4 vs. 59.2, t(485) = 28.31, $p < .001$, Cohen's $d = 1.12$); for the LBC1936, the difference between average MHT scores at 11 and 70 years old was 15.2 points (49.0 vs. 64.2, t(1016) = 56.19, $p < .001$, Cohen's $d = 1.46$).

In the LBC1921, a further MHT score was available at age 87. On this occasion, participants scored significantly less than they did 8 years previously (54.1 vs. 59.2: $t(200) = -11.26$, $p < .001$, Cohen's $d = -.41$). Because the LBC1921 participants were assessed on separate occasions in old age, it was also possible to compare MHT performance at ages 11 and 79 in those who did and did not complete all follow-ups. As shown in Table 1, the LBC1921 wave 3 returnees scored significantly higher on the MHT at ages 11 and 79 than those who did not return for this follow-up.

Stability of the Moray House Test

Correlating MHT performance across several decades provides an indication of its stability. The correlation of MHT scores from 11 and 79 years old in the LBC1921 has been reported previously and indicated substantial stability across 68 years (Deary et al., 2004). The stability coefficient of .66 rose to .76 after correction

for the restriction of range in the LBC1921 sample versus the full SMS1932 from which it was drawn (Wiberg & Sundstrom, 2009). The inclusion of a 3rd wave of data collection extends the period over which the stability of the MHT can be examined by 8 years: a period now spanning 11 to 87 years old, or 76 years. This is reported for the first time and is believed to be the longest period over which a stability coefficient has been computed for intelligence. The correlation of .51 between MHT scores obtained at ages 11 and 87 indicates that 26-51% of the variance was shared. We state a range because there is debate as to whether one should square a correlation coefficient as a means of determining the percentage of shared variance; in some cases, using the raw correlation may be more appropriate, hence the range of possible values for the shared variance (Ozer, 1985). Furthermore, correcting for range restriction, the stability coefficient of the MHT from age 11 to 87 increased to .61 (the LBC1921 participants tested at age 87 were even less representative of the original SMS1932 population than the LBC1921 participants at age 79).

The correlation between the MHT at ages 79 and 87 years old in the LBC1921 of .70 suggested a high degree of stability across the 8-year period; again, the percentage of stable variance ranged from 50-70%. When corrected for range restriction the correlation increased to .75. This correlation reflected the restriction in range that took place between ages 79 and 87 in the sample, but not the restriction in range from ages 11 to 79. The correlations were also attenuated because the MHT, like all tests of cognitive ability, is not completely reliable (Ozer, 1985).

The LBC1936 data allowed us to examine the stability of MHT performance from childhood to old age in a sample almost twice as large (although previously reported, this was as a partial eta squared from an ANOVA including other covariates and not referenced in the main text: Luciano et al., 2009a). The stability coefficient

across 59 years (11 to 70 years old) was .67 ($p < .001$, N = 1017), rising to .78 after disattenuation for restriction of range.

Modeling the influence of childhood intelligence on MHT level and change in the LBC1921

We examined the influence of MHT performance in childhood on the degree to which MHT performance changed from age 79 to 87. In the growth curve model there were two outcomes: overall cognitive ability *level* across the 8-year time span from ages 79 and 87 (intercept); and the *change* in cognitive ability from age 79 to 87 (slope). For the analysis, the MHT scores in the LBC1921 from ages 11, 79 and 87 were adjusted to remove the effects of age. The raw MHT score (11, 79 or 87) was entered as the dependent variable in a linear regression whilst age in days at the time the test was completed was the independent variable. The standardized residual (the age-corrected MHT score) was then converted to an IQ-type score (mean = 100, sd = 15). In addition to age-11 IQ, a number of other potential contributors to the level of, and change in, late-life cognitive ability were included: sex, social class, number of years of education, smoking status at age 79, and alcohol consumption at age 79. All variables were standardized prior to the modeling analysis.

The growth curve model was essentially saturated, so it fit well (RMSEA = .000, TLI = 1.000 and CFI = 1.000). The parameter estimates are shown in Table 2 and illustrated in Figure 1. The mean slope parameter of -.03 indicates that IQ (as standardized for the model) declined by .03 sd each year from 79 to 87 years old. The variance in the mean slope of .01 was small, but compared to the mean level of change, it was substantial. For example, 32% of the individual slope parameters fell outside the range -.13 to .07. To examine whether the variance in slope was significantly different from zero, further models were run specifying this parameter.

The slope variance could not be reduced to zero without substantial deterioration in model fit.

[Table 2 here]

[Figure 1 here]

Age-11 IQ was the strongest single predictor of the intercept (overall level) of IQ at ages 79 and 87, explaining about 32.6% of the variance independently of the other predictors. The other factors contributing to the level of IQ in old age were sex (male advantage) and education (positive relationship between years of education and IQ), explaining about 4.0% and 1.4% of the variance, respectively. None of the predictors made a significant contribution to the slope parameter (the change in IQ from 79 to 87 years old). Combining their effects, age-11 IQ and the demographic variables accounted for 45.6% of the variance in the level of IQ, but only a non-significant 3.3% of the variance in change across 8 years.

Discussion

The results present the longest available evaluation of the stability of a test of intelligence, covering a 76-year period between age 11 and age 87 (although the retest at age 79 means this is not an unbroken span). These data from the Lothian Birth Cohort 1921 illustrate the high level of stability in cognitive ability across several decades of the human lifespan. The cumulative evidence suggests that at least, if not more than, 50% of the variance in later life cognitive ability test performance is accounted for by childhood cognitive ability. Given this stability, it is important that researchers investigating the determinants of cognitive ability in later life account for it. The stability of cognitive ability across a period of 8 years within old age was also high. Early cognitive ability is generally the largest individual predictor of the *level* of cognitive ability in later life; however, in this sample, early ability did not predict

change in cognitive ability over a period of 8 years in old age. Therefore, prior ability predicted the level, but not change in, later life ability. This is important information for those investigating the contributors to age-related cognitive decline.

Flynn and other effects

Like the Scottish Mental Survey populations from which they were drawn (Maxwell, 1961; Deary et al., 2009b; Deary et al., 2004), the participants of the LBC1936 performed better than those from the LBC1921, although both groups were aged 11 at time of testing. This increase in test performance across almost a generation is an early example of what is now known as the Flynn effect (Flynn, 1999). The causes and consequences of these increases are still a matter of debate.

Those who joined the LBC research in later life performed better as children than those who were not recruited; the individuals recruited in their 70s scored almost 12 points higher at age 11 than their peers. However, the mean childhood test scores from the Edinburgh area in the Scottish Mental Surveys were the highest in the country, and so this likely reflects a stable lifelong regional difference to some degree (the mean for the 1947 survey population was 36.70 compared with the educational area including Edinburgh of 40.28: Scottish Council for Research in Education, 1958). The higher ability of the LBC participants compared to the whole population who took part in the Scottish Mental Survey of 1947 may also be partly explained by a survivor effect, whereby higher-ability individuals were more likely to reach their 70s and 80s in relatively good health and thus be eligible for recruitment into the follow-up studies (Whalley & Deary, 2001). The male advantage in MHT performance at age 79 in the LBC1921 is probably another example of a survivor effect. The differential survival of men and women would suggest that, at any given

age, the men in a sample are likely to be healthier than their female contemporaries. Health in this context would include higher cognitive ability.

Higher-ability individuals are more likely to self-select to be part of studies such as the LBCs (Nishiwaki, Clark, Morton, & Leon, 2005). This lack of representativeness is often unresolved and not properly accounted for in research examining cognitive abilities. In this case, however, we knew the populations from which our participants were drawn, so it was possible to correct for their selected nature. That is, the archival data allow the stability coefficients to be disattenuated for the restriction of range.

High stability across almost 80 years

The current analyses from the Lothian Birth Cohorts of 1921 and 1936 significantly extend—in terms of the period of time and the size of the sample—previous findings from these and similar studies of the high degree of stability in cognitive functions over time (Gow et al., 2008; Deary et al., 2009b; Deary et al., 2004). It has again been highlighted that childhood cognitive ability is by far the largest independent predictor of the level of cognitive ability in later life.

Although previously reported, it is important to reaffirm this stability. Many studies lacking a valid measure of premorbid ability use demographic measures as a proxy of this, most commonly educational attainment and/or socioeconomic position. However, in the current analysis using data from the LBC1921 and previous work with the LBC1936 (Johnson, Gow, Corley, Starr, & Deary, 2009), it was shown that these variables contribute only small independent effects. In addition, without a measure of prior cognitive ability, the advantages of education or social class in terms of cognitive outcomes can be overstated. These factors are themselves outcomes predicted by earlier cognitive ability, and so are partly mediating its effects (Deary et

al., 2005). This has been discussed more fully in the context of modelling analyses conducted on the LBC1936 data (Johnson et al., 2009). That these factors have independent effects is not in question, but it is likely they each contribute small amounts of variance in the level of later cognitive ability, once an earlier measure of ability is taken into account.

Intellectual stability as a major confounder in cognitive aging research

This argument is also prescient when considering other determinants of the level of cognitive ability in later life. If these determinants are themselves outcomes of early ability, then any contemporaneous associations with later functioning may be wholly or partly spurious. The earlier discussion of inflammatory markers is but one example (Luciano et al., 2009b). Indeed, to cite an ongoing debate, there are a number of studies reporting a link between participation in mentally stimulating activities and cognitive function (Fratiglioni, Paillard-Borg, & Winblad, 2004). Here too, prior ability is a frequently unmeasured confounder. The outcomes of such research are therefore often open to question, as it is not possible to answer:

> does participation in stimulating activities promote cognitive performance or is it that better performing cognitively capable subjects tend to participate in more intellectual, social and physical activities? (Scarmeas & Stern, 2003, p. 626).

This centrally important issue has been highlighted by two Canadian research teams, working on different longitudinal samples. The Veterans study followed up men tested 40 years after their initial screening assessment as army recruits (Pushkar et al., 1995); the Victoria Longitudinal Study recruited participants from middle age onwards, testing them every 3 years (Hultsch, Hertzog, Small, & Dixon, 1999). The analyses and subsequent reanalyses of these datasets have resulted in a fertile and

productive debate as it has been suggested that it is equally likely that increased intellectual engagement maintains cognitive function in later life (Pushkar et al., 1995), or that cognitive decline itself causes decreased participation in intellectual activities (Hultsch et al., 1999). Hertzog and co-workers suggest that researchers should not be too eager to accept the 'engaged lifestyle' hypothesis, as the available evidence is presently not conclusive. The inclusion of a valid measure of premorbid ability in longitudinal studies is but one method to allow the independent effects of a range of psychosocial, demographic and other lifestyle factors on the aging mind to be assessed. Whist not the only possible way to resolve such issues, it should be clear that the stability of cognitive ability measures is not just a niche psychometric interest, but something which has implications for those exploring the determinants of cognitive ability and change. If this is within the context as full a lifespan perspective as possible, including information from earlier periods still, such findings would be even more valuable; that is, there may be important factors prior to the teens which may impact on lifelong health and intelligence.

Prior ability does not predict subsequent decline

Although prior ability predicts the level of later ability, the current analysis suggests it is not related to the degree of change experienced across time. This distinction between level and change is an important one, and the determinants of each require discrete attention. The stability of cognitive ability over large periods of time is well-established (from the LBC and other longitudinal studies); however, reports of the association between early ability and cognitive *change* measured some decades later have been inconsistent (for example, Gow et al., 2008; Bourne et al., 2007). The current results suggesting no association are between early ability and cognitive aging are consistent with previous work in the LBC1921 evaluating 4-year

change in a general factor based on a battery of 3 cognitive tests (Gow et al., 2008). Though this study doubled the observation period, it also included many fewer participants due to death and disability, thus reducing statistical power considerably. The variance in the slope parameter was small in absolute terms, which made detection of predictive association by covariates difficult. The lack of a relationship between early ability and decline is in contrast to that reported by other research teams (Bourne et al., 2007; Richards et al., 2004). As noted, methodological variation and analytical techniques may account for such discrepancies. Growth curve analyses is ideally suited to such datasets, and is therefore the preferred means of analysis when there is an interest in investigating both level and change in an outcome (regression and similar techniques are adequate when level at a single point in time is the outcome). It will be necessary to replicate these analyses in further cohorts and with diverse tests of cognitive ability, whilst utilizing the most appropriate techniques for the data available.

Given the lack of an association of early cognitive ability, education and social class with cognitive aging, the question returns to what might be protective. Indeed, many factors emerging later in the lifespan are of interest (Kirkwood et al., 2008; Deary et al., 2009a). These might include, but are not limited to non-shared environmental effects, age-related diseases, or terminal decline phenomena. Furthermore, as cognitive function at age 11 is not fully developed, it is worth considering measures of cognitive ability into adolescence and early adulthood as potential predictors of later change.

Strengths and limitations

Clearly, the presence of measures of premorbid cognitive ability in large samples of individuals was an advantage in this study. Most research studies are

subject to selective recruitment, and the LBC are no exception. Variations in where the participants are enrolled and/or tested may skew the composition of the sample under investigation (whether contacted through, and visited at, institutional or care settings, for example, or volunteers who are required to visit an external facility); this will have consequences for the patterns of age-related change observed (Hedden et al., 2004; Hertzog, 1996). Additionally, longitudinal studies may underestimate cognitive changes due to non-random attrition, or practice effects over numerous testing sessions (Hedden et al., 2004). Selective attrition is likely to bias any sample over time; however, the growth curve models made use of all available data including those who were present at age 79 but not at age 87. Furthermore, the bias introduced is unlikely to be that those participants lost to follow-up have a different set of associations among the variables, but rather that there has been some restriction of range and therefore some underestimation of the parameters as a result of this. Practice or retest effects can be present even over extended periods of time and these are likely to have affected the results reported. There is limited short-term test-retest data on the MHT and so it is not possible to estimate the extent of this. That said, the MHT is a relatively long and varied test (71 items over 45 minutes) so it might be that participants are less likely to remember individual items or types of items (compared with standardized psychometric tests of intelligence which are individually short and generally consist of a single type of item). The non-representativeness of the sample can be partly accounted for by correcting for range restriction. Knowing the composition of the population from which the participants were drawn makes it possible to do this more accurately.

Although the use of the same test across several decades is a strength of the current analysis in evaluating overall stability of cognitive ability, it did not make it

possible to investigate differential rates of aging across varied cognitive domains. Furthermore, the use of a single test precluded the possibility of separating change from measurement error in the growth curve models. This issue can be addressed by defining cognitive ability at a given age as the latent trait from a number of tests, where possible.

Conclusions

Cognitive ability is highly stable across the lifespan. It is important to recognize this in searching for the contributors to the level of cognitive ability in later life. Though overall level of late-life cognitive ability was substantially related to early life ability, cognitive change within old age, in the current LBC1921 sample at least, was not related to the level of prior ability nor a range of other covariates. Future studies should examine whether this is because there is little variance in rates of cognitive aging or whether other variables besides those considered here may predict change.

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Table 1

Mean (sd) MMSE and Moray House Test scores for Lothian Birth Cohorts of 1921 and 1936

	Age	MMSE	Moray House Test			
			All	Wave 3 returnees	Wave 3 non-returnees	T-test
LBC1921	11		46.4 (12.0) N = 496	47.7 (11.5) N = 213	45.5 (12.4) N = 283	t(467) = -2.47, p = .014
	79	28.2 (1.70) N = 548	59.2 (10.8) N = 542	61.9 (9.3) N = 234	57.2 (11.5) N = 308	t(495.66) = -5.23, p < .000
	87	27.8 (2.3) N = 207	54.1 (13.8) N = 202			
LBC1936	11		49.0 (11.8) N = 1028			
	70	28.8 (1.4)	64.2 (8.8)			

	N = 1090	N = 1079

Note. The Mini-Mental State Examination (MMSE) has a maximum of 30; the Moray House Test has a maximum of 76. The t-tests shown compare the performance of the LBC1921 participants who returned for all 3 waves of cognitive testing versus those who did not.

Table 2

Estimates derived from the linear growth curve model of Moray House Test (MHT) IQ score between age 79 and age 87

			Standardized regression coefficients (*p* value)	
	Mean	Variance	Intercept	Slope
Model intercept	.02	1.06	-	-
Model slope	-.03	.01	-	-
Sex	.58	.24	-.10 (.004)	.08 (.237)
Alcohol status	-.03	.75	-.03 (.470)	-.02 (.780)
Smoking status	.00	1.00	-.02 (.498)	.11 (.124)
Social class	.00	1.00	-.06 (.129)	.03 (.661)
Education	.00	1.00	.12 (.001)	-.07 (.374)
Age-11 IQ	.03	1.00	.57 (.000)	-.03 (.666)

Note. The model slope expressed the change per year. For sex, the reference category was male. Smoking status at age 79 was defined as never, ex or current; Education was the number of years in full-time formal education. The latent means differed slightly from 0 because the model did

not completely account for the selectivity in sample attrition over the 8-year follow-up. All continuous variables were standardized before analysis.

Figure 1. Growth curve model of the level and change of Moray House Test (MHT) IQ score between age 79 and age 87. Items in rectangles are measured variables, those in ellipses are latent traits. The numbers adjacent to the arrows leading from latent traits to measured variables were fixed (the 8 refers to the 8-year period between age 79 and 87). The other numbers—beside those arrows going from measured variables to latent traits, and beside arrows between latent traits—are parameters estimated by the program. These can be treated like standardized partial beta weights, and when squared give the proportion of variance shared by adjacent variables. For the covariates, all measured paths are shown, but parameter estimates are only given for those paths that were significant $p < .01$. All parameter estimates are standardized and given to two decimal places. For sex, the reference category was male. Education is the number of years in full-time formal education; smoking status at age 79 is defined as never, ex or current.