Intelligence 37 (2009) 520–528

Contents lists available at ScienceDirect

Intelligence

The association of childhood intelligence with mortality risk from adolescence to middle age: Findings from the Aberdeen Children of the 1950s cohort study

D.A. Leon [a,*], D.A. Lawlor [b], H. Clark [c], G.D. Batty [d,e], S. Macintyre [d]

[a] Department of Epidemiology and Population Health, London School of Hygiene & Tropical Medicine, UK
[b] MRC Centre for Causal Analyses in Translational Epidemiology, Department of Social Medicine, University of Bristol, UK
[c] The Dugald Baird Centre for Research on Women's Health, University of Aberdeen, Scotland, UK
[d] MRC Social and Public Health Sciences Unit, University of Glasgow, Scotland, UK
[e] MRC Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh, Edinburgh, UK

ARTICLE INFO

Article history:
Received 11 August 2008
Received in revised form 28 October 2008
Accepted 17 November 2008
Available online 7 January 2009

Keywords:
Mortality
IQ
Cohort study
Epidemiology

ABSTRACT

There is growing evidence that childhood IQ is inversely associated with mortality in later life. However, the specificity of this association in terms of causes of death, whether it is continuous over the whole range of IQ scores and whether it is the same according to age and sex is not clear. In a large cohort ($N = 11{,}603$) of a complete population of children born in one city in the UK in the early 1950s, IQ measured at age 7 years (using a routinely administered picture test) was found to be inversely associated with mortality between the ages of 15 and 57 years. For every 1 SD increase in IQ at 7, the all cause mortality hazard ratio was 0.79 (95% CI 0.73, 0.85). On adjustment for a range of perinatal factors, father's social class at birth, number of sibs in the household and childhood height and weight, this was attenuated slightly to 0.81 (0.74, 0.88). Almost identical associations of IQ with mortality were seen for men and women as well as at younger (15–39) and older (40+) ages. These associations were across the entire IQ range, although some of the high mortality in the lowest category of IQ (<70) was accounted for by causes associated with congenital disorders. Overall, external causes of death showed the strongest association, with weaker associations being seen for cancer. Further work is required to understand the mechanisms whereby childhood IQ has such a robust association with mortality in later life.

1. Introduction

It has been known for several decades that individuals who are severely cognitively impaired have increased rates of mortality in childhood, adolescence (Simila, von Wendt, & Rantakallio, 1986) and adulthood (Patja, Molsa, & Iivanainen, 2001; Patja, Iivanainen, Vesala, Oksanen, & Ruoppila, 2000). More recently, attention has been focussed on the inverse association of childhood cognition with mortality in adult life that appears to extend over the entire range of IQ (Batty, Deary, & Gottfredson, 2007). This association has been replicated in a number of independent studies (Hart et al., 2003; Osler et al., 2003; Kuh, Richards, Hardy, Butterworth, & Wadsworth, 2004; Pearce, Deary, Young, & Parker, 2006; Whalley & Deary, 2001), and is even seen to hold even among individuals whose IQ in childhood was in the range 135 to 160 or more (based on the Stanford–Binet test) (Martin & Kubzansky, 2005). While most studies of childhood cognition as a predictor of adult mortality have only been able to look at deaths into middle age, an inverse association has also been found with mortality up to age 76 years in subjects included in the 1932 Scottish Mental Survey (Hart et al., 2003; Whalley & Deary, 2001). Childhood or early adulthood intelligence has been reported to be inversely associated with risk for

* Corresponding author. LSHTM, Keppel St, London WC1E 7HT.
E-mail address: david.leon@lshtm.ac.uk (D.A. Leon).

0160-2896/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.intell.2008.11.004

cardiovascular disease (Hart et al., 2004; Batty, Mortensen, Nybo Andersen, & Osler, 2005; Lawlor, Batty, Clark, Macintyre, & Leon, 2008; Batty, Gale et al., 2008b), risk factors for adult mortality (Lawlor, Clark, Davey Smith, & Leon, 2006; Chandola, Deary, Blane, & Batty, 2006; Batty, Deary, & Macintyre, 2007), and hospital admission from unintentional injury (Lawlor, Clark, & Leon, 2007). However, a recent study found no association of early adult IQ with cancer risk in adulthood (Batty et al., 2007).

A number of hypotheses, which are not mutually exclusive, have been put forward to explain the inverse associations of intelligence measured in childhood with adult mortality (Batty & Deary, 2004; Whalley & Deary, 2001). One type of explanation is that childhood IQ and later mortality risk may share common antecedents rather than being directly causally related. This possibility has also been formulated in terms of childhood IQ being a marker of individual constitution or "system integrity". Genetic factors would be included, as would perinatal problems giving rise to irreversible damage to organ systems including the brain. However, these early life factors could include nutritional or other insults as indicated by impaired fetal or growth in infancy and/or childhood. A second class of alternative explanation is that there may be confounding factors such as socioeconomic position and parental intelligence or education, all of which are associated with intelligence in early life and later adverse health risks in the offspring. To the extent that the association of childhood intelligence with mortality is causal it might be mediated by one or more adult characteristics such as educational attainment, occupation and socioeconomic position. Intelligence might also directly influence an individual's ability to interpret and effectively utilise information about health-related risk factors and health service use.

Despite progress in trying to understand the mechanism and implications of the inverse association between childhood intelligence and adult mortality, several issues remain to be resolved (Deary & Batty, 2006). These include the extent to which the associations of childhood intelligence with different causes of death really do vary, how far these associations are truly linear, and whether associations differ depending upon the age in early life that intelligence is tested. Moreover, few large studies have been able to examine the association of childhood IQ with later mortality in a complete population comprised of individuals ranging from those with major cognitive deficits (due to congenital anomalies or severe brain damage in early life) to the most intellectually gifted children.

The aim of this paper is to add to existing literature on the association of childhood intelligence with mortality in adulthood. It substantially extends an earlier report of from this study that was based on far fewer deaths (Batty, Clark, Morton, Macintyre, & Leon, 2002). In particular we will examine associations of childhood IQ at age 7 years with all-cause mortality, by sex and age at death, as well as investigating associations with mortality from major causes of death, taking account of a number of early-life potential confounders.

2. Methods

Data from the *Aberdeen Children of the 1950s* (ACONF) cohort study were used. This study was based on the Aberdeen Childhood Development Survey (ACDS) conducted in the early 1960s into the determinants of "mental subnormality" in a complete population (Birch, Richardson, Baird, Horobin, & Illsley, 1970). The ACONF cohort consists of the 12,150 members of the ACDS who were born in Aberdeen between 1950–1956 and for whom comprehensive information was abstracted from the Aberdeen Maternity and Neonatal Databank (AMND) (Samphier & Thompson, 1981) about the course of their mother's pregnancy and their own characteristics at birth. Routine school records provided the source of measurements of weight and height at school entry and childhood IQ at ages 7, 9 and 11. Full details of the cohort have been described elsewhere (Batty et al., 2004; Leon, Lawlor, Clark, & Macintyre, 2006).

2.1. Assessment of childhood intelligence

Throughout the 1950s in Scotland, tests of intelligence were routinely administered to children at 7, 9 and 11 years of age and results for members of the *ACDS* cohort were linked to their 1962 survey data (Batty et al., 2004). The tests used at age 7 were the Moray House Picture Intelligence test numbers 1 or 2. These were 100 and 98 item tests respectively where children were asked to variously identify which one of a series of pictures did not belong, were absurd, were reflections (reversed similarities) or analogous to a reference picture, or to put them in sequence or to complete sequences. To our knowledge these tests have not been validated directly against any standard measure of intelligence such as WAIS or WISC. At age 9, the Schonell and Adams Essential Intelligence tests form A or B were used. The tests at age 11 included a battery of Moray House tests: two ability tests (verbal reasoning 1 and 2) and two attainment tests (Arithmetic, English). All intelligence tests were taken within 6 months of the child's 7th, 9th and 11th birthday respectively. Tests were age standardised, for Scotland as a whole, with means of 100 and standard deviations of 15. In this paper results of cognitive tests are referred to as Intelligence Quotients (IQs).

At the time of the initial survey in 1962 members of the cohort were being educated in 46 schools ($N = 11{,}967$), 7 special institutions for children with special needs ($N = 178$) or at home ($N = 5$). Four private schools and four state schools did not provide the research team with results of intelligence tests at age 7 year for any of their pupils ($N = 226$). Interestingly, the majority of children educated in one of the special schools (143/178) did have intelligence test scores at age 7, although the mean score was low (67 vs 108 for children from ordinary schools), reflecting that these institutions mainly dealt with children with moderate or severe learning difficulties. In contrast, intelligence test scores at ages 9 and 11 years were only available respectively for 14 and 16 children attending these special institutions.

In this paper, we therefore focus entirely upon intelligence test scores at age 7 years as we wish to look at the associations with mortality in a fully representative sample of the population of school-age children in Aberdeen in 1962.

2.2. Potential alternative explanatory factors

Data on pregnancy induced hypertension, antepartum haemorrhage, birth weight, gestational age, father's occupational

social class at the subject's birth and maternal age at birth were abstracted from the Aberdeen Maternity and Neonatal Databank (Batty et al., 2004). Family size in 1962 (number of siblings) was obtained from the participant's responses to questions in the Aberdeen Child Development Survey.(Leon, Lawlor, Clark, & Macintyre, 2006) Height and weight in childhood were abstracted from school medical records at the time of the original survey. Birth weight *z*-scores were used in the analysis standardised by sex and completed week of gestation.(Wilcox & Skjaerven, 1992) This is so that it is possible to separate out the two quite separate determinants of size at birth: fetal growth rate in utero (measured by the *z*-score) and length of gestation. Height and weight at school entry were included as height and weight for age *z*-scores, stratifying by sex and age in 3 month intervals. The rationale for this is similar, in that in this study a child's height and weight are in part a reflection of their growth rate and in part a function of the age at which they are measured.

2.3. Mortality data

The vast majority (99%) of study members were successfully traced through the General Register Office (GRO) (Scotland) (Batty et al., 2004; Leon, Lawlor, Clark, & Macintyre, 2006). Traced participants have been linked to the National Health Service Central Register (NHSCR), which provides death certificate details (date and cause of death). Underlying causes of death were coded to the International Classification of Disease (ICD) revisions 7 to 10 depending upon when they occurred. Deaths from malignant neoplasms were defined as 140–207 in ICD revisions 7–9, and all those prefixed with "C" in ICD-10. Cardiovascular disease was 400–469 (ICD-7), 390–459 (ICD8-9) and all those with prefix "I" in ICD-10. External causes (injuries, poisonings and violence) was 800–999 (ICD-7 to ICD-9) and deaths prefixed with "V" to "Y" in ICD-10. Revision-specific ranges were used to define directly alcohol attributable deaths following standard practice (Baker & Rooney, 2003). These included deaths from alcoholic psychosis, alcoholism or alcohol dependence, liver cirrhosis, alcoholic cardiomyopathy and accidental poisoning by alcohol. Full details of codes are available from the authors.

2.4. Statistical methods

Data were analysed using Cox proportional hazards regression models, with participants' age as the time axis. At the time of the original 1962 survey children were between 6 and 13 years of age. The focus of this paper is on mortality in adulthood, so individuals aged less than 15 years of age did not contribute to risk. Contributions to risk were censored at the earliest of the following dates: (i) death; (ii) (first) emigration out of the UK; (iii) 31 December 2007.

In our main analyses the results are presented for categorical variables (as defined in Table 3). This avoided making assumptions about linearity, as well as enabling a direct inspection of the shape of associations, which was particularly crucial with respect to the IQ-mortality association. We also conducted analyses using IQ as a continuous variable examining the possibility of non-linear associations first by inspection of the hazard ratios across 8 categories of increasing intelligence test score. More formally we then modelled the association of IQ at 7 as a continuous variable (per standard deviation i.e. an increase of 15 points on the IQ scale) on the mortality hazard ratio. Using a likelihood ratio test we then compared the fit of this model with one including IQ at 7 years expressed as first, second, third or fourth order terms. A significant LR test was regarded as evidence of non-linearity, but in no case was this found to be the case.

The cohort contained siblings, with the 12,150 initial members being drawn from 9423 families. Because of this all regression models were estimated taking account of family clustering by using robust standard errors to calculate confidence intervals and *p*-values.

To determine whether the effect of intelligence on mortality varied by gender, gender-specific effects were examined and Wald-tests of interaction were computed. The use of Cox models in these analyses assumes that any association between childhood IQ and death does not vary by

Table 1
Baseline characteristics of participants in the Aberdeen Children of the 1950s cohort $N = 12{,}150$.

Characteristic		Mean (SD), Median (IQR) or number (%) [a]	Number (%) missing data
Female		5868 (48)	0
IQ score age 7	Mean (SD)	107.1 (16.4)	471 (4)
Father's occupational social class at birth	I and II	1163 (10)	681 (6)
	III non manual	1335 (12)	
	III manual	5319 (46)	
	IV	1689 (15)	
	V	1963 (17)	
Mother experienced gestational hypertension during pregnancy with participant		2062 (17)	1 (0.01)
Mother experienced antepartum haemorrhage during pregnancy with participant		274 (2)	1 (0.01)
Assisted delivery		1431 (12)	2 (0.02)
Maternal age at birth (years)	15–19	567 (5)	6 (0.05)
	20–24	3798 (31)	
	25–29	3777 (32)	
	30–34	2546 (21)	
	35–39	1108 (9)	
	>=40	348 (3)	
Twin or triplet		286 (3)	0
Gestational age (weeks)	<37	688 (7)	1148 (10)
	37–40	4433 (44)	
	>40	4856 (49)	
Birth weight (g)	Mean (SD)	3299 (513)	22 (0.2)
Height (cm) [b]	Mean (SD)	107.9 (6.4)	514 (4.2)
Weight (kg) [b]	Mean (SD)	19.1 (2.9)	511(4.1)
Family size (number of siblings) at time of 1962 primary school survey [c]	1	1122 (9)	94 (0.8)
	2	3677 (30)	
	3	3061 (25)	
	4	2048 (17)	
	5	1108 (9)	
	6 or more	1040 (9)	

[a] Percentages for categorical variables have been calculated with the denominator being the number with complete data (i.e. they are the % of those with complete data for each particular variable).
[b] Height and weight measured at first school entry, mean age 5 years.
[c] This is the total number of brothers and sisters (plus the participant themselves) that the study participant reported having at the time of the primary school survey.

age at death (since our models used age as the time axes). The validity of this assumption was assessed by inspection of survival plots and by testing for evidence of a statistical interaction with the time scale (age) of the models using a likelihood ratio test and also by using the Schoenfeld test.

Analyses were conducted using Stata/IC versions 9 and 10.

3. Results

Table 1 shows the baseline characteristics of participants and provides details about the extent of missing data by variable. Most variables were complete or almost complete. The variables with the highest percentage of missing values were IQ score at age 7 years (4%), height and weight at school entry (4%), father's occupational social class at birth of study member (6%) and gestational age at birth (10%). The subset of individuals with complete data on all variables shown in Table 1 (used in the multivariable analyses presented below) constituted 84% of the total study population (10,131/12,150). Among those with an IQ score at age 7, the proportion with missing data in one or more of the other variables in Table 1 declined as IQ increased. For those with an IQ of <70, 29% had data missing in one or more of the variables compared with 10% for those with an IQ of 130+. However, the all cause mortality of those with missing data in any of the Table 1 variables did not differ markedly from that of those with complete data, those with complete data having a hazard ratio of 0.89 (95% CI 0.73, 1.09) relative to those with missing data.

The associations of early life and childhood characteristics with IQ at age 7 are shown in Table 2. All of the variables are associated with IQ at age 7 years. Particularly strong associations were seen for father's social class, preterm birth, birth weight, height and weight at first entry to school and number of children in family in 1962.

From age 15 years study members contributed 465 thousand person years at risk, with 687 deaths occurring up to the end of 2007. The associations of mortality with key variables are shown in Table 3, adjusted for age and sex. IQ at age 7 was inversely associated with mortality, with a 20% decline in risk for every 1SD increase. Of the perinatal variables, only assisted delivery was associated with mortality. Father's social class showed an inverse association with mortality, while number of siblings showed a positive association. Childhood height, but not weight, showed a weak inverse association with mortality.

Although we do not report in this paper the association of IQ at ages 9 and 11 with mortality, these were very similar in magnitude to those shown for IQ at age 7 in Table 3. This is not surprising as IQ at age 7 years is correlated with those at IQ at 9 ($r=0.73$, $P<0.0001$) and at 11 years (0.75, $P<0.0001$).

The inverse association of all cause mortality with IQ at age 7 is shown in more detail in Table 4. This association was seen across the entire IQ range, and was not driven solely by high mortality among those with IQ scores of <80. However, it is notable that mortality HRs were particularly extreme at both ends of the IQ scale. Mortality among those with an IQ of 130+ was half that of those with an IQ of 90–109 and one quarter of that among those with an IQ of <70. Very similar patterns of association were evident among males and females and at ages less than 40 years and 40+ years. Formal tests of interaction by sex and age were both non-significant ($P>0.05$).

The inverse association of mortality with IQ remained substantial and apparent across the entire range of scores on adjustment for perinatal factors, childhood socio-economic circumstances and height and weight (Table 5). All models shown in this Table are for the consistent subset of 10,704 individuals (562 deaths) with complete data on all variables considered. Adjustment for father's social class and number of siblings in the family in 1962 resulted in some slight attenuation of the association. However, adjustment for perinatal factors and childhood height and weight had almost no impact on the association.

There was a small degree of variation in the strength of association of IQ at age 7 with different causes of death. In the same subset of subjects with complete data analysed in Table 5, there were 161 deaths from cancer, 115 from cardiovascular disease, 134 from external causes and 152 deaths from the aggregate of all other causes. When adjusted for age and sex only, the HR for cancer was 0.82 (0.71, 0.94),

Table 2
Percentage distribution or mean of early life characteristics by category of intelligence test scores at age 7.[a]

	N with data (maximum available)	Intelligence test score at age 7 years							*P* trend[c]
		<70	70–79	80–89	90–109	110–119	120–129	130+	
N with IQ at 7 years	11,679	176	399	1058	4854	2512	1655	1025	
Female	11,679	87 (49)	182 (46)	515 (49)	2224 (46)	1229 (49)	817 (49)	514 (50)	0.02
Manual social class at birth	11,080	146 (90)	348 (95)	920 (93)	3880 (85)	1851 (77)	1060 (67)	543 (55)	<0.001
Gestational hypertension	11,679	25 (14)	60 (15)	135 (13)	778 (16)	436 (17)	327 (20)	216 (21)	<0.001
Antepartum haemorrhage	11,679	5 (3)	17 (4)	33 (3)	11 (2)	39 (2)	37 (2)	21 (2)	0.008
Assisted delivery	11,678	17 (10)	44 (11)	93 (9)	512 (11)	317 (13)	227 (14)	148 (14)	<0.001
Mother <20 at birth	11,675	11 (6)	30 (8)	60 (6)	262 (5)	94 (4)	56 (3)	17 (2)	<0.001
Twin/triplet	11,679	5 (3)	24 (6)	55 (5)	120 (2)	48 (2)	31 (2)	16 (2)	<0.001
Preterm[b]	10,489	21 (15)	42 (13)	90 (10)	321 (7)	140 (6)	79 (5)	44 (5)	<0.001
Birth weight *z*-score	11,658	176 (−0.30)	397 (−0.26)	1055 (−0.17)	4843 (−0.03)	2509 (0.05)	1653 (0.12)	1025 (0.16)	<0.001
Height *z*-score	11,331	156 (−0.76)	387 (−0.57)	1034 (−0.40)	4717 (−0.10)	2435 (0.16)	1609 (0.28)	993 (0.31)	<0.001
Weight *z*-score	11,335	156 (−0.58)	387 (−0.39)	1035 (−0.32)	4717 (−0.07)	2437 (0.09)	1610 (0.22)	993 (0.26)	<0.001
More than 5 children in family	11,663	34 (20)	87 (22)	174 (16)	517 (11)	136 (5)	54 (3)	24 (2)	<0.001

[a] Results are Number (%) except for birth weight, height and weight *z*-score where they are Number (mean *z*-score).
[b] Gestational age <37 weeks.
[c] *P*-value for trend after adjustment for sex (other than for sex itself).

Table 3
Age and sex adjusted associations of childhood intelligence, and other early life characteristics, with all-cause mortality aged 15 ± years.

Exposure	Category	N with data (n deaths)	Sex adjusted HR for all cause mortality (95%CI)		P
Sex	Male	6234 (435)	1.00	[ref]	<0.001
	Female	5834 (252)	0.61	(0.52, 0.71)	
IQ 7	Per SD	11,603 (661)	0.80	(0.75, 0.86)	<0.001
Social class birth	I and II	1156 (54)	0.90	(0.67, 1.20)	0.001
	III NM	1327 (67)	0.96	(0.74, 1.25)	
	III M	5270 (277)	1.00	[ref]	
	IV	1685 (103)	1.15	(0.92, 1.44)	
	V	1951 (143)	1.38	(1.13, 1.69)	
	Other [a]	635 (43)	1.21	(0.88, 1.67)	
Gestational hypertension	No	10,023 (574)	1.00	[ref]	0.87
	Yes	2044 (113)	0.98	(0.80, 1.20)	
Antepartum haemorrhage	No	11,793 (670)	1.00	[ref]	
	Yes	274 (17)	1.08	(0.67, 1.75)	
Assisted delivery	No	10,645 (627)	1.00	[ref]	0.007
	Yes	1421 (60)	0.69	(0.53, 0.90)	
Maternal age at birth	15–19	561 (37)	1.16	(0.83, 1.63)	0.53
	20–34	10,053 (569)	1.00	[ref]	
	35+	1448 (81)	0.98	(0.78, 1.24)	
Twin or triplet	No	11,753 (669)	1.00	[ref]	0.97
	Yes	315 (18)	0.99	(0.63, 1.56)	
Gestational Age in weeks	<37	751 (50)	1.15	(0.86, 1.56)	0.57
	37–39	4807 (271)	1.00	(0.84, 1.18)	
	40+	5254 (292)	1.00	[ref]	
Birth weight for gestational age *z*-score	Per SD	12,046 (687)	0.99	(0.91, 1.07)	0.76
Childhood height *z*-score	Per SD	11,563 (652)	0.91	(0.85, 0.99)	0.019
Childhood weight *z*-score	Per SD	11,566 (654)	1.00	(0.93, 1.09)	0.90
Family size (number of siblings in family) at time of 1962 survey [b]	1	1114 (54)	1.00	[ref]	<0.001
	2	3652 (189)	1.08	(0.80, 1.45)	
	3	3045 (155)	1.06	(0.78, 1.45)	
	4	2029 (128)	1.31	(0.96, 1.81)	
	5	1098 (76)	1.42	(1.00, 1.01)	
	6 or more	1036 (79)	1.55	(1.10, 2.19)	

HR: hazard ratio; CI: confidence interval; SD: standard deviation; ref: reference category.
All analyses (except sex) are adjusted for sex. Adjustment for age is undertaken by doing survival analyses with age as the time scale.
Analyses conducted on maximum number of participants with data for each exposure variable. N varies as indicated in third column of table.
[a] Unemployed, deceased, not known.
[b] This is the total number of brothers and sisters (plus the participant themselves) that the study participant reported having at the time of the primary school survey.

for cardiovascular disease it was 0.81 (0.68, 0.95), for external causes it was 0.75 (0.65, 0.87) and for the aggregate of all other causes it was 0.77 (0.66, 0.90). More detail of these cause-specific associations adjusted for all perinatal and childhood factors is shown in Table 6. The IQ categories used have been collapsed (relative to Table 5) because of the smaller numbers of cause-specific deaths available for analysis. The strongest inverse association of mortality with IQ at 7 years was seen for external causes. Inverse associations were also seen for cancer and the aggregate of all other causes (152 deaths). Among the cancer deaths, the two most frequent cancer sites were cancer of the lung and of the female breast. In adjusted analyses equivalent to those reported in Table 6, the hazard ratio per 1 SD increase in IQ at 7 years was 0.71 (95% CI 0.53, 0.96) for cancer of the lung (based on 31 deaths) and 0.69 (95% CI 0.46, 1.04) for cancer of the female breast (based on 23 deaths). For cardiovascular disease, the highest HR was for those in the <70 group and the lowest among those in the 130+ group. However, overall this inverse trend was not statistically significant ($P>0.05$).

The very high mortality for the residual category of "other causes" in the <70 IQ category deserves some further comment. In total there were 9 deaths in this category, 3 of which occurred among individuals with missing data in one or more of the other variables included in our analyses. Four of these 9 had a congenital problem assigned as an underlying cause (1 malformation of the nervous system, 1 muscular dystrophy and 2 Down's syndrome). This underlines the fact

Table 4
Age and sex adjusted associations of childhood intelligence aged 7 years with all-cause mortality aged 15+ years stratified by sex and age.

IQ at age 7 years	Total			Sex						Age in years					
				Males			Females			15–39			40+		
	HR*	(95% CI)	N deaths	HR	(95% CI)	Number deaths	HR	(95% CI)	Number deaths	HR [a]	(95% CI)	Number deaths	HR [a]	(95% CI)	Number deaths
<70	1.70	(1.06, 2.73)	19	1.71	(0.95, 3.07)	12	1.80	(0.83, 3.91)	7	1.51	(0.66, 3.46)	6	1.81	(1.03, 3.2)	13
70–79	1.22	(0.85, 1.77)	31	1.05	(0.65, 1.72)	18	1.57	(0.89, 2.78)	13	1.22	(0.66, 2.27)	11	1.22	(0.77, 1.94)	20
80–89	1.06	(0.82, 1.37)	72	0.94	(0.67, 1.31)	41	1.34	(0.90, 2.00)	31	0.87	(0.55, 1.39)	21	1.17	(0.86, 1.59)	51
90–109	1.00	[ref]	308	1.00	[ref]	208	1.00	[ref]	100	1.00	[ref]	110	1.00	[ref]	198
110–119	0.76	(0.61, 0.94)	121	0.81	(0.62, 1.05)	82	0.71	(0.49, 1.02)	39	0.76	(0.53, 1.08)	43	0.76	(0.58, 0.99)	78
120–129	0.76	(0.60, 0.98)	80	0.70	(0.51, 0.96)	47	0.91	(0.62, 1.35)	33	0.58	(0.37, 0.92)	22	0.86	(0.64, 1.15)	58
130+	0.46	(0.32, 0.67)	30	0.44	(0.27, 0.71)	18	0.53	(0.29, 0.96)	12	0.38	(0.19, 0.76)	9	0.50	(0.32, 0.79)	21
HR per SD	0.80	(0.75,0.86)		0.82	(0.75, 0.89)		0.78	(0.69, 0.88)		0.79	(0.71, 0.89)		0.81	(0.74, 0.88)	
P-value interaction		n.a.				$P=0.51$						$P=0.84$			

HR: hazard ratio; CI: confidence interval; ref: reference category.
All analyses (except sex) are adjusted for sex. Adjustment for age is undertaken by doing survival analyses with age as the time scale.
Analyses conducted on 11,603 participants at risk on their 15th birthday with complete data on IQ at 7 years and sex.
[a] Adjusted for sex.

that this group with very low IQ includes people who had serious medical conditions in early life of which low cognition was just one aspect. Another two deaths were classed as "unattended deaths" and another from epilepsy. If this residual category of "other causes" is excluded, then the aggregate of the remaining deaths from cancer, cardiovascular disease and external causes had a HR of 1.39 (0.67, 2.85) for the <70 IQ group relative to those with an IQ of 90–109, compared with 2.63 (1.11, 6.21) for the full aggregate of "other causes" (Table 6).

In the main analyses, based on the subset of individuals with complete data, there were 42 deaths assigned to the category of directly alcohol-attributable deaths. None of these deaths occurred among people with an IQ<70 or among those with an IQ of 130+. The HR per 1 SD increase in IQ score at age 7 for this class of alcohol attributable deaths was 0.86 (0.68, 1.09). This was a weaker association than seen for all causes of death combined, thus suggesting that alcohol attributable deaths were not an important component of the inverse association of IQ at 7 years with all cause mortality.

Finally, despite the fact that the strongest association was seen for external causes, the aggregate of all other causes together showed an almost identical strength of association with IQ at age 7, with an HR per 1 SD increase in score of 0.82 (0.75, 0.91).

4. Discussion

We have found that childhood cognition/IQ is inversely associated with mortality from all causes from age 15 years into middle age. This association is seen across the entire range of IQ scores from <70 to 130+ and is equally strong among men and women. Perinatal factors, childhood socioeconomic circumstances and growth appear to explain very little of this association. However, we have found evidence that the strength of this association varies to some degree by cause of death. After adjustment for perinatal and childhood factors cardiovascular disease shows the weakest association (that is in fact statistically non-significant), while the category of external causes shows the strongest association. However,

Table 5
Age and sex adjusted associations of childhood intelligence aged 7 years with all-cause mortality aged 15+ years adjusted for potential confounders, Aberdeen Children of the 1950s cohort.

IQ at age 7 years	N deaths	Model 1		Model 2		Model 3		Model 4		Model 5	
		HR	(95% CI)	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)
<70	14	1.82	(1.06, 3.13)	1.80	(1.04, 3.10)	1.75	(1.01, 3.02)	1.74	(1.01, 2.99)	1.74	(1.00, 3.01)
70–79	22	1.15	(0.74, 1.78)	1.13	(0.73, 1.76)	1.08	(0.70, 1.69)	1.12	(0.72, 1.73)	1.07	(0.68, 1.66)
80–89	66	1.18	(0.90, 1.55)	1.17	(0.89, 1.53)	1.14	(0.87, 1.50)	1.16	(0.88, 1.52)	1.13	(0.86, 1.49)
90–109	261	1.00	–	1.00	–	1.00	–	1.00	–	1.00	–
110–119	107	0.78	(0.62, 0.97)	0.78	(0.62, 0.99)	0.80	(0.64, 1.01)	0.77	(0.61, 0.97)	0.81	(0.64, 1.02)
120–129	67	0.73	(0.56, 0.95)	0.74	(0.56, 0.97)	0.76	(0.58, 1.00)	0.72	(0.55, 0.95)	0.76	(0.58, 1.00)
130+	25	0.44	(0.29, 0.66)	0.44	(0.29, 0.67)	0.46	(0.30, 0.70)	0.43	(0.28, 0.65)	0.46	(0.30, 0.70)
HR per SD		0.79	(0.73, 0.85)	0.79	(0.73, 0.86)	0.80	(0.74, 0.87)	0.79	(0.73, 0.85)	0.81	(0.74, 0.88)

HR: hazard ratio; CI: confidence interval; ref: reference category.
Note all hazard ratios adjusted for age as this defined as the time dimension in the Cox regression.
Analyses conducted on 10,704 participants at risk on their 15th birthday with complete data on IQ at 7 years and all other covariates considered in table.
Model 1: Adjusted for sex.
Model 2: Model 1 plus perinatal factors (maternal age at birth, gestational age, birth weight *z*-score, gestational hypertension, antepartum hemorrhage, assisted delivery, whether multiple birth or not).
Model 3: Model 1 plus familial factors (father's social class at birth, family size).
Model 4: Model 1 plus childhood height and weight *z*-scores (measured at mean age 6 years).
Model 5: Models 1–4 combined.

Table 6
Age and sex adjusted associations of childhood intelligence aged 7 years with cause-specific mortality aged 15+ years adjusted for potential confounders.

IQ at age 7 years	All causes			Cancer			Cardiovascular disease			External causes			Other causes		
	HR	(95% CI)	N deaths	HR	(95% CI)	N deaths	HR	(95% CI)	N deaths	HR	(95% CI)	N deaths	HR	(95% CI)	N deaths
<70	1.74	(1.00, 3.01)	14	0.91	(0.22, 3.73)	2	1.70	(0.52, 5.61)	3	1.69	(0.53, 5.38)	3	2.63	(1.11, 6.21)	6
70–89	1.11	(0.87, 1.43)	88	1.42	(0.93, 2.19)	30	0.83	(0.46, 1.51)	15	1.15	(0.69, 1.89)	21	1.02	(0.62, 1.68)	22
90–109	1.00	[ref]	261	1.00	[ref]	71	1.00	[ref]	54	1.00	[ref]	68	1.00	[ref]	68
110–129	0.79	(0.65, 0.96)	174	0.72	(0.50, 1.06)	47	0.94	(0.62, 1.43)	39	0.64	(0.42, 0.97)	37	0.92	(0.64, 1.33)	51
130+	0.46	(0.30, 0.70)	25	0.66	(0.34, 1.30)	11	0.40	(0.14, 1.17)	4	0.34	(0.13, 0.87)	5	0.36	(0.14, 0.91)	5
HR per SD	0.81	(0.74, 0.88)		0.81	(0.70, 0.94)		0.88	(0.73, 1.06)		0.74	(0.63, 0.87)		0.81	(0.68, 0.96)	

HR: hazard ratio; CI: confidence interval; ref: reference category.
All HRs adjusted for sex, perinatal factors (maternal age at birth, gestational age, birth weight for gestational age *z*-score, gestational hypertension, antepartum haemorrhage, assisted delivery, whether multiple birth or not), familial factors (father's social class at birth, family size) and childhood height and weight *z*-score (measured at around age 6 years). Note all models adjusted for age as this is the time dimension of the Cox regression.
Analyses conducted on 10,704 participants at risk on their 15th birthday with complete data on IQ at 7 years and all other covariates considered in table.

it should be noted that these differences in strength of association by cause could be explained by chance, as indicated by the width and overlap of the confidence intervals of the estimates.

These findings based on mortality are consistent with our previous findings that in this cohort childhood IQ is inversely associated with adult-life hospital admissions for unintentional injuries (Lawlor, Clark, & Leon, 2007) and coronary heart disease and stroke (Lawlor, Batty, Clark, Macintyre, & Leon, 2008). Moreover, the results are broadly consistent with what has been found in the few other studies that have looked at the association of childhood cognition/IQ with later life mortality (Batty, Deary, & Gottfredson, 2007).

Earlier analyses of questionnaire responses gathered in middle age in this cohort have suggested that lower childhood IQ is associated with indicators of hazardous patterns of alcohol consumption (Batty, Deary, & Macintyre, 2007; Batty, Deary, & Macintyre, 2006). However, analyses of other cohorts have found associations in the opposite direction (Hatch et al., 2007; Batty, Deary et al., 2008a) and only weak inverse associations of binge drinking with cognition measured at 7 years (Jefferis, Manor, & Power, 2008). The mortality analyses reported in this paper suggest that deaths directly attributable to alcohol are not notably associated with childhood IQ and explain little of the overall inverse mortality gradient observed.

There are a number of particular strengths of this study. First, the measure of IQ used in these analyses at age 7 was the Moray House Picture test. Compared to tests conducted at a later age that often involve numeracy or literacy skills, this has the advantage that it provides a purer measure of cognitive function per se that is less influenced by educational environment and attainment. Second, the study is based on a complete population cohort. The motivation for the original data collection in the early 1960s was to provide the first ever estimate of the prevalence and investigate the determinants of "mental subnormality" in a complete unselected population (Birch, Richardson, Baird, Horobin, & Illsley, 1970). Thus the cohort includes individuals who in 1962 were in special-needs schools or were being educated at home or were in other institutions as well as those in mainstream schools, resulting in a full range of IQ scores in childhood. Third, the recent work done to ascertain mortality to date in this cohort ascertained the vital status of 99% of the 12,150 subjects born in Aberdeen 1950–56 (Nishiwaki, Clark, Morton, & Leon, 2005).

However, the study and analyses presented here have a number of weaknesses. Although the cohort as a whole is fully representative of all school-aged children born in Aberdeen and living in the city in 1962, the subset used in the multivariable analysis is based on only 84% of cohort members because of missing data. Those with a low IQ are more likely to be excluded. However, several results suggest this may not have led to bias in our findings. Firstly, the mortality rate of those excluded because of missing data was not substantively different to those included in the multivariable analyses. Secondly, and more importantly, the hazard ratios for all cause mortality per 1 SD increase in IQ at age 7 years were almost identical when estimated on all subjects irrespective of whether they had complete data on other covariables (HR = 0.80) and when estimated on the subset with complete data on all covariables (HR = 0.79).

The main limitation of the study is that we have not been able to adjust for adult behavioural and socio-economic factors that would allow us to explore the extent to which these mediate the association between childhood IQ and later mortality. Although we conducted a postal questionnaire survey of cohort members in 2001–3 (Nishiwaki, Clark, Morton, & Leon, 2005), these data cannot yet be used in such an analysis because the numbers of deaths occurring since this survey is as yet too small.

We have previously investigated the association of childhood IQ with characteristics in middle age of the 64% of study members who did respond to the postal questionnaire survey. A wide range of adult risk factors were observed to be associated with childhood IQ including smoking, heavy alcohol consumption and obesity and overweight (Batty, Deary, & Macintyre, 2007; Lawlor et al., 2005). What is notable, however, is that when adjusted for adult socio-economic circumstances, and education in particular, these associations of childhood IQ with risk factors are either eliminated (Lawlor, Clark, Davey Smith, & Leon, 2006), or attenuated to a considerable degree (Batty, Deary, & Macintyre, 2007; Lawlor et al., 2005).

A few other studies have been able to look at the mediating role of educational attainment and adult socio-economic circumstances on the association between child or early adult IQ and later mortality. These have tended to show

that these factors are important mediators (Batty, Shipley et al., 2008c; Kuh, Richards, Hardy, Butterworth, & Wadsworth, 2004).

Some part of the inverse IQ-mortality association may be due to IQ being a marker of long-term general health problems or frailties. As we have shown above, in our study the very high mortality seen among people with a very low IQ in childhood (<70) is partly explained by the fact that these individuals were born with serious morbidities such as congenital anomalies and Down's syndrome. This is an obvious and extreme manifestation of IQ providing what has been described as a measure of "system integrity" (Whalley & Deary, 2001). However, aside from this extreme end of the cognition spectrum, adjustment for a range of physical signs and health indicators at birth and in childhood has little or no effect on the association of childhood IQ on later mortality. To the extent that the markers of frailty and general health that we have available are informative about "system integrity", these analyses provide no support for the hypothesis that it is this general factor that underlies the IQ-mortality association as has been previously hypothesised (Whalley & Deary, 2001).

Further work needs to be done to confirm the extent to which these inverse associations are mediated by adult circumstances. It should nevertheless be noted that, as always, identifying mediating pathways would not provide "alternative explanations" for this association. Instead identification of mediators enables an elaboration of the nature of the pathways through which factors might operate (Leon, 1993). There is good evidence that childhood IQ is very strongly related to circumstances and individual characteristics in childhood as well as adult life. For example, we have previously shown that response to a postal questionnaire sent out to members of the Aberdeen Children of the 1950s cohort increased almost monotonically for every 5-point increase in IQ score at 7 years, going from 50% in the bottom fifth of the distribution of IQ at age 7, to 74% among those in the top fifth (Nishiwaki, Clark, Morton, & Leon, 2005). Further research is required to better understand the mechanisms by which childhood IQ does affect later life circumstances and behaviours so powerfully. In particular, given that this association appears to be particularly strong for injuries, poisonings and violence (Lawlor, Clark, & Leon, 2007; Osler, Andersen, Laursen, & Lawlor, 2007), it would be useful to know what aspects of adult behaviour associated with childhood IQ lead to increased risk.

Finally, it is notable that there are two rather distinct literatures that both look at the association of intelligence with mortality, but these are generally not linked together (Sabia et al., 2008). There is the growing body of evidence that measures of cognition in childhood and early adult life are predictive of mortality in middle and old age that has been the focus of this paper. There is also the more substantial literature that also shows inverse associations between intelligence measured in middle age or older with mortality. There are very few studies linking measures of cognition/intelligence taken both in childhood as well as middle or older ages with subsequent mortality. However, despite the absence of such data, it is still surprising that there have been so few attempts to link up these literatures at least at a conceptual level. The logic is obvious. As has been reported by a number of researchers, there appears to be a strong relationship between measures of intelligence in childhood and those in later life, with a correlation of 0.63 being shown between cognitive test results age 11 and aged 77 in 97 individuals who took part in the 1932 Scottish Mental Survey (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). This is comparable to the correlation (Pearson r) observed in the UK 1946 birth cohort (National Study of Health and Development) between an aggregate measure of general cognitive ability at 8 years and the National Adult Reading Test (which correlates highly with full-scale IQ) at 53 years of 0.66 (M Richards, personal communication, 2008). To this extent associations between childhood cognitive ability and later health and mortality may operate through the adult correlates of cognitive ability measured in adulthood. These in turn are going to be easier to study, as they do not depend upon identifying special historic cohorts with childhood IQ measures with follow-up into middle age or later.

In conclusion, in a large cohort of a complete population of children born in the UK in the early 1950s we have provided further evidence that IQ in childhood is inversely related to mortality in adult life up to age 57 years from a range of causes including cancer and external causes. These associations are not explained by characteristics and circumstances in the perinatal period or in childhood up to age 7 years. However, whether the pattern of association will remain the same as the cohort moves into their 7th decade of life remains to be seen.

5. Funding

The Aberdeen Children of the 1950s Study was funded as a component project of a Medical Research Council Co-operative Group. The Scottish Chief Scientist Office provided funds for a study of relationships of childhood intelligence. D A Lawlor is funded by a Department of Health (UK) Career Scientist Award and works in an MRC centre. David Batty is a Wellcome Trust Fellow. The Medical Research Council (MRC) Social and Public Health Sciences Unit receives funding from the UK MRC and the Chief Scientist Office at the Scottish Government Health Directorates. The UK Medical Research Council (MRC) and University of Edinburgh provide core funding for the MRC Centre for Cognitive Ageing and Cognitive Epidemiology.

Acknowledgements

We are very grateful to Raymond Illsley for providing us with the data from the Aberdeen Child Development Survey and for his advice about the study. Graeme Ford played a crucial role in identifying individual cohort members and in helping us initiate the process of revitalisation. Doris Campbell, George Davey Smith, Bianca De Stavola, Marion Hall, David Godden, Diana Kuh, Glyn Lewis, Susan MB Morton and Viveca Östberg collaborated with the authors to revitalise the cohort. Margaret Beveridge assisted Heather Clark in the study management. We would also like to thank staff at the ISD (Edinburgh), GRO (Scotland) and NHSCR (Southport) for their substantial contributions and John Lemon who undertook the linkage to the Aberdeen Maternity and Neonatal Databank. Bianca De Stavola provided crucial and very helpful statistical advice on these analyses. Finally, we thank the study participants.

References

Baker, A., & Rooney, C. (2003). Recent trends in alcohol-related mortality, and the impact of ICD-10 on the monitoring of these deaths in England and Wales. *Health Statistics Quarterly*, *17*, 5—14.

Batty, G. D., Clark, H., Morton, S. M. B., Macintyre, S., & Leon, D. A. (2002). Intelligence in childhood and mortality, migration, questionnaire response rate, and self-reported morbidity and risk factor levels in adulthood — Preliminary findings from the Aberdeen Children of the 1950s' study.*J Epidemiol.Community Health*, *56*(Suppl 2) A1.

Batty, G. D., & Deary, I. J. (2004). Early life intelligence and adult health. *British Medical Journal*, *329*, 585—586.

Batty, G. D., Morton, S. M., Campbell, D., Clark, H., Davey Smith, G., Hall, M., et al. (2004). The Aberdeen children of the 1950s cohort study: Background, methods and follow-up information on a new resource for the study of life course and intergenerational influences on health. *Paediatric and Perinatal Epidemiology*, *18*, 221—239.

Batty, G. D., Deary, I. J., & Macintyre, S. (2006). Childhood IQ and life course socioeconomic position in relation to alcohol induced hangovers in adulthood: The Aberdeen Children of the 1950s study. *Journal of Epidemiology Community Health*, *60*, 872—874.

Batty, G. D., Mortensen, E. L., Nybo Andersen, A. M., & Osler, M. (2005). Childhood intelligence in relation to adult coronary heart disease and stroke risk: Evidence from a Danish birth cohort study. *Paediatric and Perinatal Epidemiology*, *19*, 452—459.

Batty, G. D., Deary, I. J., & Gottfredson, L. S. (2007). Premorbid (early life) IQ and later mortality risk: Systematic review. *Annals of Epidemiology*, *17*, 278—288.

Batty, G. D., Deary, I. J., & Macintyre, S. (2007). Childhood IQ in relation to risk factors for premature mortality in middle-aged persons: The Aberdeen Children of the 1950s study. *Journal of Epidemology Community Health*, *61*, 241—247.

Batty, G. D., Wennerstad, K. M., Davey Smith, G., Gunnell, D., Deary, I. J., Tynelius, P., et al. (2007). IQ in early adulthood and later cancer risk: Cohort study of one million Swedish men. *Annals of Oncology*, *18*, 21—28.

Batty, G. D., Deary, I. J., Schoon, I., Emslie, C., Hunt, K., & Gale, C. R. (2008). Childhood mental ability and adult alcohol intake and alcohol problems: The 1970 British cohort study. *American Journal of Public Health*, *98*(12), 2237—2243.

Batty, G. D., Gale, C. R., Mortensen, L. H., Langenberg, C., Shipley, M. J., & Deary, I. J. (2008). Pre-morbid intelligence, the metabolic syndrome and mortality: The Vietnam Experience Study. *Diabetologia*, *51*, 436—443.

Batty, G. D., Shipley, M. J., Mortensen, L. H., Boyle, S. H., Barefoot, J., Gronbaek, M., et al. (2008). IQ in late adolescence/early adulthood, risk factors in middle age and later all-cause mortality in men: the Vietnam Experience Study. *Journal of Epidemiology Community Health*, *62*, 522—531.

Birch, H. G., Richardson, S. A., Baird, D., Horobin, G., & Illsley, R. (1970). *Mental Subnormality in the Community: A Clinical and Epidemiologic Study.* Williams & Wilkins Co: Baltimore.

Chandola, T., Deary, I. J., Blane, D., & Batty, G. D. (2006). Childhood IQ in relation to obesity and weight gain in adult life: The National Child Development (1958) Study. *International Journal of Obesity (London)*, *30*, 1422—1432.

Deary, I. J., & Batty, G. D. (2006). Commentary: Pre-morbid IQ and later health — The rapidly evolving field of cognitive epidemiology. *International of Journal Epidemiology*, *35*, 670—672.

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

Hart, C. L., Taylor, M. D., Davey Smith, G., Whalley, L. J., Starr, J. M., Hole, D. J., et al. (2003). Childhood IQ, social class, deprivation, and their relationships with mortality and morbidity risk in later life: prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Psychosomatic Medicine*, *65*, 877—883.

Hart, C. L., Taylor, M. D., Davey Smith, G., Whalley, L. J., Starr, J. M., Hole, D. J., et al. (2004). Childhood IQ and cardiovascular disease in adulthood: Prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Social Science and Medicine*, *59*, 2131—2138.

Hatch, S. L., Jones, P. B., Kuh, D., Hardy, R., Wadsworth, M. E., & Richards, M. (2007). Childhood cognitive ability and adult mental health in the British 1946 birth cohort. *Social Science & Medicine*, *64*, 2285—2296.

Jefferis, B. J., Manor, O., & Power, C. (2008). Cognitive development in childhood and drinking behaviour over two decades in adulthood. *Journal of Epidemiology Community Health*, *62*, 506—512.

Kuh, D., Richards, M., Hardy, R., Butterworth, S., & Wadsworth, M. E. (2004). Childhood cognitive ability and deaths up until middle age: A post-war birth cohort study. *International Journal of Epidemiology*, *33*, 408—413.

Lawlor, D. A., Batty, G. D., Morton, S. M., Clark, H., Macintyre, S., & Leon, D. A. (2005). Childhood socioeconomic position, educational attainment, and adult cardiovascular risk factors: The Aberdeen Children of the 1950s cohort study. *American Journal of Public Health*, *95*, 1245—1251.

Lawlor, D. A., Clark, H., Davey Smith, G., & Leon, D. A. (2006). Childhood intelligence, educational attainment and adult body mass index: Findings from a prospective cohort and within sibling-pairs analysis. *International Journal of Obesity(London)*, *30*, 1758—1765.

Lawlor, D. A., Clark, H., & Leon, D. A. (2007). Associations between childhood intelligence and hospital admissions for unintentional injuries in adulthood: The Aberdeen Children of the 1950s cohort study. *American Journal of Public Health*, *97*, 291—297.

Lawlor, D. A., Batty, G. D., Clark, H., Macintyre, S., & Leon, D. A. (2008). The association of childhood intelligence with risk of coronary heart disease and stroke: Findings from the Aberdeen Children of the 1950s cohort study. *European Journal of Epidemiology*.

Leon, D. A. (1993). Failed or misleading adjustment for confounding. *Lancet*, *342*, 479—481.

Leon, D. A., Lawlor, D. A., Clark, H., & Macintyre, S. (2006). Cohort profile: the Aberdeen Children of the 1950s study. *International Journal of Epidemiology*, *35*, 549—552.

Martin, L. T., & Kubzansky, L. D. (2005). Childhood cognitive performance and risk of mortality: a prospective cohort study of gifted individuals. *American Journal of Epidemiology*, *162*, 887—890.

Nishiwaki, Y., Clark, H., Morton, S. M., & Leon, D. A. (2005). Early life factors, childhood cognition and postal questionnaire response rate in middle age: The Aberdeen Children of the 1950s study. *BMC Medical Research Methodology*, *5*, 16.

Osler, M., Andersen, A. M., Due, P., Lund, R., Damsgaard, M. T., & Holstein, B. E. (2003). Socioeconomic position in early life, birth weight, childhood cognitive function, and adult mortality. A longitudinal study of Danish men born in 1953. *Journal of Epidemiology and Community Health*, *57*, 681—686.

Osler, M., Andersen, A. M., Laursen, B., & Lawlor, D. A. (2007). Cognitive function in childhood and early adulthood and injuries later in life: the Metropolit 1953 male birth cohort. *International Journal of Epidemiology*, *36*, 212—219.

Patja, K., Iivanainen, M., Vesala, H., Oksanen, H., & Ruoppila, I. (2000). Life expectancy of people with intellectual disability: A 35-year follow-up study. *Journal of Intellectual Disability Research*, *44*(Pt 5), 591—599.

Patja, K., Molsa, P., & Iivanainen, M. (2001). Cause-specific mortality of people with intellectual disability in a population-based, 35-year follow-up study. *Journal of Intellectual Disability Research*, *45*, 30—40.

Pearce, M. S., Deary, I. J., Young, A. H., & Parker, L. (2006). Childhood IQ and deaths up to middle age: The Newcastle thousand families study. *Public Health*, *120*, 1020—1026.

Sabia, S., Gueguen, A., Marmot, M. G., Shipley, M. J., Ankri, J., & Singh-Manoux, A. (2008). Does cognition predict mortality in midlife? Results from the Whitehall II cohort study. *Neurobiology Aging*.

Samphier, M. L., & Thompson, B. (1981). Longitudinal studies in Aberdeen, Scotland. B. The Aberdeen maternity and neonatal databank. In S. Mednick, A. Baert, & B. Bachmann (Eds.), Prospective Longitudinal Research. *An Empirical Basis for the Primary Prevention of Psychosocial Disorders* (pp. 61—65). Oxford: OUP.

Simila, S., von Wendt, L., & Rantakallio, P. (1986). Mortality of mentally retarded children to 17 years of age assessed in a prospective one-year birth cohort. *American Journal of Hypertension*, *30*(Pt 4), 401—405.

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

Wilcox, A. J., & Skjaerven, R. (1992). Birth weight and perinatal mortality: The effect of gestational age. *American Journal of Public Health*, *82*, 378—382.