



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

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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.

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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

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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

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