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Childhood IQ and all cause mortality before and after age 65: prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies

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Running title: Childhood IQ and all cause mortality

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Abstract

Objectives The objective was to investigate how childhood IQ related to all cause mortality before and after age 65.

Design The Midspan prospective cohort studies, followed-up for mortality for 25 years, were linked to individuals’ childhood IQ from the Scottish Mental Survey 1932.

Methods The Midspan studies collected data on risk factors for cardiorespiratory disease from a questionnaire and at a screening examination, and were conducted on adults in Scotland in the 1970s. Age 11 IQ from the Scottish Mental Survey 1932, a cognitive ability test conducted on 1921-born children attending schools in Scotland, was found for 938 Midspan participants. The relationship between childhood IQ and mortality risk, adjusting for adulthood socioeconomic confounders, was analysed. The effect of adjustment for childhood IQ on the relationship between established risk factors (blood pressure, smoking, height and respiratory function) and mortality was also investigated.

Results For deaths occurring up to age 65, there was a 36% increased risk per standard deviation decrease (15 points) in childhood IQ which was reduced to 29% after adjusting for social class and deprivation category. There was no statistically significant relationship between childhood IQ and deaths occurring after the age of 65. Adjustment for childhood IQ attenuated the risk factor-mortality relationship in deaths occurring up to age 65, but had no effect in deaths occurring after age 65.

Conclusions Childhood IQ was significantly related to deaths occurring up to age 65, but not to deaths occurring after age 65.
Introduction

Few studies have attempted to apply personal psychological variables, such as cognitive ability and personality, to the study of morbidity and mortality across the human lifespan. Many studies which contain data on psychological factors and disease have been cross-sectional or have concentrated on middle-aged samples with relatively low rates of mortality. In the present study we aim to investigate (1) whether mental ability in childhood contributes to all cause mortality in later adulthood, specifically to deaths before and after age 65; and (2) how childhood mental ability contributes to the relationship between risk factors and all cause mortality.

It is known that a person’s social circumstances, for example, education and socio-economic status, are related to mortality risk (Davey Smith et al., 1998a; Department of Health and Social Security, 1980; Kunst & Mackenbach, 1994). In addition, people who live in more deprived areas have poorer lung function (Davey Smith, Hart, Watt, Hole & Hawthorne, 1998b), higher rates of illnesses such as cardiovascular diseases and cancers, and tend to die younger (Duijkers, Kromhout, Spruit & Doombos, 1989; Eames, Ben Shlomo & Marmot, 1993). Social circumstances in childhood have been associated with several major illnesses in adulthood including coronary heart disease, stroke, lung cancer, stomach cancer, and respiratory diseases (Davey Smith, Hart, Blane & Hole, 1998; Joseph & Kramer, 1996). The age at which people leave full-time education has also been linked to death from cancer and cardiovascular disease and to deaths from all causes (Davey Smith et al., 1998a). However, these studies lacked information on personal factors, such as cognitive function and personality traits, which may be important determinants of disease and mortality.

One exception to the lack of personal factors in lifespan studies of mortality is the “Terman Life Cycle of Children with High Ability” sample (N=1258), which showed that conscientiousness, lack of cheerfulness, and permanence of mood were associated with living longer among people with very high ability (Schwartz et al., 1995). Part of the influence of personality on mortality may be explained by a person’s health-related behaviours.
Friedman (2000) refers to personality influences on health choices over time as ‘health trajectories’ (Friedman, 2000). Researchers examining such trajectories found the trait neuroticism may be associated with health damaging behaviours such as smoking or alcohol consumption (Aldwin, Spiro, Levenson & Cupertino, 2000; Whiteman, Fowkes, Deary & Lee, 1997). However, whether the associations between psychological factors and mortality are attributable to these lifestyle factors remains unclear. It is therefore important to include information about a person’s health behaviours in any investigation of the risk factors for mortality to identify potential intervening variables (for example, cigarette smoking and alcohol consumption) which may account for variance in the link between personal psychological factors and subsequent health outcomes.

Higher cognitive ability is associated with more education and higher socio-economic position (Neisser et al., 1996). There are consistent reports of significant associations between cognitive ability and mortality: among old people, cognitive status and the amount of cognitive decline in old age is associated with mortality (Anstey, Luszcz, Giles & Andrews, 2001; Deeg, Hofman & van Zonneveld, 1990; Korten et al., 1999; Neale, Brayne & Johnson, 2001), an association that remains even after demographic and health variables are taken into account (Korten et al., 1999). Cognitive ability in younger adults also predicts mortality: one study of Australian Vietnam veterans found that intelligence scores taken at army recruitment predicted death between age 22 and 40 (O'Toole & Stankov, 1992). Educational attainment and mental ability in young adulthood were also predictive of mortality in old age in the Nun study (Snowdon, Greiner, Kemper, Nanayakkara & Mortimer, 1999; Snowdon, Ostwald & Kane, 1989).

There are now well-known factors associated with mortality risk. Both systolic and diastolic blood pressure are positively related to cardiovascular disease and mortality (Prospective Studies Collaboration, 2002). Cigarette smoking is also a notorious risk factor (Doll, Peto, Wheatley, Gray & Sutherland, 1994). Achieved adult height is inversely associated with mortality: shorter people tend to die earlier on average (Davey Smith et al., 2000). Respiratory function is a good indicator of mortality, even in never smokers (Hole et al., 1996). We previously investigated the relationships
between childhood IQ and these classical risk factors (Hart et al., 2003b; Starr et al., 2003; Taylor et al., 2003). Correlation coefficients between childhood IQ and diastolic blood pressure, systolic blood pressure, adult height and respiratory function were -0.12, -0.16, 0.24 and 0.15 respectively (Hart et al., 2003b). This means that people with higher childhood IQ had lower blood pressure in adulthood, were taller and had better respiratory function. Mean IQ was average for never smokers (100.5 [standard deviation (SD) 14.4]), lower for current smokers (98.5 [SD 15.3]) and higher for former smokers (103.7 [SD 14.3]) (Taylor et al., 2003).

Childhood IQ was inversely related to all cause mortality in two Scottish populations (Hart et al., 2003c; Whalley & Deary, 2001). Whalley and Deary (2001) ascertained the survival status of over 2,000 men and women who had taken part in the Scottish Mental Survey 1932 when living in the Aberdeen area. They found that IQ at age 11 was significantly related to survival from age 11 to age 76. People with lower IQ were less likely to be alive at age 76. The chances of those with lower IQ being alive at age 76 were 79% per standard deviation lower childhood IQ (95% confidence interval 75% to 84%). We previously showed in over 900 men and women from the Midspan studies, in the central belt of Scotland, that childhood IQ, as measured in the Scottish Mental Survey 1932, was significantly related to all cause mortality in the 25 years after they had been screened when aged about 50 years in the 1970s (Hart et al., 2003c). There was a 17% increased risk of all cause mortality per standard deviation disadvantage in IQ (95% confidence interval 7% to 29%). This was similar to the results from the Aberdeen study above: when converted to survival rather than death, the risk was 85% (95% confidence interval 75% to 84%). We have also investigated the relationship of childhood IQ with cause specific events in the Midspan population, defined as hospital admissions for, or deaths from certain diseases (Hart et al., 2003b). We found that childhood IQ was related to cardiovascular disease, coronary heart disease and stroke events occurring before the age of 65, but not with those events occurring after the age of 65. Additionally we looked at the relationship between classical risk factors and cardiovascular events, and investigated any additional role of childhood IQ. We found that although childhood IQ was associated with the risk factors, it contributed a small amount to the risk factor-cardiovascular disease event relationship. In this paper, we expand on all of these findings, by examining
whether the effects of childhood IQ are different in all cause mortality occurring before or after the age of 65.

We then investigate how childhood IQ contributes to the relationship between risk factors and all cause mortality and see whether any contributions of childhood IQ to the risk factor-mortality relationship are different in these two periods.

Methods

The study combines data from two large Scottish studies – the Scottish Mental Survey 1932 (SMS1932) and the Midspan prospective cohort studies. The SMS1932, conducted under the auspices of the Scottish Council for Research in Education (SCRE), obtained data about the intelligence of Scottish school children (Scottish Council for Research in Education, 1933). On 1\textsuperscript{st} June 1932, almost all children born in the year 1921 and at school in Scotland on that day, were given the same mental ability test, administered in the classroom by the class teacher. In total, 87,498 children (44,210 boys and 43,288 girls) sat the Moray House Test. The test had 71 numbered items and a maximum score of 76. It contained several general, spatial, and numerical reasoning items which included: following directions, same-opposites, word classification, analogies, practical, reasoning, proverbs, arithmetic, spatial, mixed sentences and cipher decoding. The test was closely related to the Moray House Test No.12, which was used in ‘eleven-plus’ examinations in England. Validation was carried out by a representative sample of 1,000 of the children also taking the Stanford Revision of the Binet-Simon scale, resulting in a correlation of 0.81 for boys (n = 500) and 0.78 for girls (n = 500).

The Midspan studies were large prospective cardiorespiratory studies of adults carried out in Scotland in the 1960s and 1970s. Two Midspan studies are included in this analysis. The Collaborative study was conducted between 1970 and 1973 in 27 workplaces in the west and central belt of Scotland (Davey Smith et al., 1998a). The full sample consisted of 6,022 men and 1,006 women of working age. The Renfrew/Paisley general population study was carried out between 1972 and 1976. It included 7,052 men and 8,354 women aged 45-64 years who were resident in the towns of Renfrew and Paisley near Glasgow (Hawthorne et al., 1995).
Both studies included some participants who were born in 1921. Ethical permission was obtained from the Multi-Centre Research Ethics Committee for Scotland to link the SMS1932 data set with the 1921-born participants of the Midspan data sets.

There were 1,251 Midspan participants born in 1921 and of these, 938 (75%) were matched to their mental ability score from the SMS1932. Full details of the matching procedures have been reported elsewhere (Hart et al., 2003a). The test scores were corrected for age in days, since the children’s ages varied between 10½ and 11½ years at the time of testing. The scores were then converted to the more usual IQ scores with mean 100 and standard deviation 15. The IQ distribution was also divided into quarters for some analyses.

The Midspan studies were both structured similarly, with participants completing a questionnaire and attending a physical examination (Davey Smith et al., 1998a; Hawthorne et al., 1995). The questionnaire included questions about smoking habit, home address and occupation. Social class was coded according to the Registrar General's Classification (General Register Office, 1966) for occupation at the time of screening. The social class of women was allocated according to their own occupation, except for those women in the Renfrew/Paisley study who gave their occupation as housewives. For these women, the social class allocated was that of their husband. The home address at the time of screening was retrospectively postcoded, enabling deprivation category as defined by Carstairs and Morris to be ascertained (Carstairs & Morris, 1991). This is a well-used area-based measure of deprivation, obtained from four census variables - male unemployment, overcrowding, car ownership and the proportion of heads of households in social classes IV and V (the two lowest social classes). A deprivation score for each postcode sector is obtained which is converted to seven categories ranging from 1 (least deprived) to 7 (most deprived). The few missing values for social class (n=13) and deprivation category (n=3) were imputed as social class III manual and deprivation category 5 as these were the most commonly occurring. The physical examination included the measurement of blood pressure, height and forced expiratory function in one second (FEV1), a measure of respiratory function. Blood pressure was measured with the subject seated, and diastolic pressure was recorded at the
disappearance of the fifth Korotkoff sound. The adjusted FEV1 was defined as the actual FEV1 as a percentage of the expected FEV1, derived from sex-specific linear regressions of age and height from healthy subsets of the study populations (Davey Smith et al., 1998a; Hole et al., 1996).

Midspan study participants were flagged at the National Health Service Central Register in Edinburgh. Causes and dates of death in a 25 year follow-up period for each individual were provided. Since the Collaborative study was conducted between 1970 and 1973, the follow-up period covered the years from 1970 to 1998, during which time participants were aged between 49 and 77. The follow-up period for the Renfrew/Paisley study covered the years from 1972 to 2001, with participants being aged from 51 to 80. Cox’s models (Cox, 1972) were used to calculate proportional hazards regression coefficients for one standard deviation change in risk factor (including childhood IQ), and for current and ex-smokers compared to never smokers. Relative rates are the exponentiated proportional hazards regression coefficients. The method takes account of the time to an event (in this case death) and addresses the fact that some people do not experience the event during the follow-up period. Survival time in 25 years was taken from the date of screening until the date of death. One participant had embarked from the UK during the follow-up period and survival time was taken until the date of embarkation. Deaths for the whole follow-up period were divided into those occurring up to and including age 65 (the early period) and those occurring after age 65 (the later period).

**Results**

More deaths occurred after age 65 than up to and including age 65 (table 1). Of the 432 deaths in the follow-up period, 259 (60%) occurred after age 65. Coronary heart disease and cancer were the most common causes of death in both periods, each being about a third of all deaths. A higher percentage of deaths in the early period compared with the later period were from coronary heart disease. A greater percentage of deaths in the later period were from stroke and respiratory disease.
The relative rate of dying in 25 years was inversely associated with childhood IQ (table 2). The relative rate per standard deviation decrease in childhood IQ was 1.17 with a confidence interval of 1.07 to 1.29. As the confidence interval did not include one, this indicates that the result was statistically significant, with a $P$ value less than the 0.05 level. The relative rate of 1.17 means that there was a 17% increased risk of death per standard deviation lower childhood IQ. To take account of socioeconomic factors, which may be connected with childhood IQ and act as confounders in the IQ-mortality relationship, additional adjustment was made for social class and deprivation category in adulthood and this reduced the risk to 12%. For deaths occurring up to age 65, there was a 36% increased risk per standard deviation decrease in childhood IQ which was reduced to 29% after adjusting for social class and deprivation category. There was no statistically significant relationship between childhood IQ and deaths occurring after the age of 65. That is, the confidence interval included 1.

Survival curves of the highest and lowest quarters of the IQ distribution showed a widening after about five years of follow-up, with the highest IQ quarter faring better than the lowest (Figure). Patterns were similar for men and women separately (not shown). After 15 years of follow-up (when the participants were aged about 65), the curves became parallel. This suggests that participants in the highest and lowest quarters had similar mortality experiences after age 65, although starting from a different level. Survival curves for the middle two quarters lay between the highest and lowest (not shown). The survival function for the lowest IQ quarter was significantly different from the survival functions of the other three quarters (Breslow test: lowest versus 2$^{nd}$ lowest $p=0.05$, lowest versus 2$^{nd}$ highest $p=0.048$, lowest versus highest $p=0.012$). The other survival function pairs were not significantly different from each other.
The next analyses investigated the relationship between classical risk factors and all cause mortality. Both diastolic and systolic blood pressure were positively related to mortality over 25 years, with a 22% increased risk per standard deviation increase in blood pressure, when adjusted for sex (table 3). Additional adjustment for childhood IQ attenuated the risk slightly. Adjustment for social class and deprivation category had no effect on the diastolic blood pressure-mortality relationship and little effect for systolic blood pressure. Both diastolic and systolic blood pressure were not significantly associated with mortality for deaths occurring up to age 65, but adjustment for childhood IQ reduced the increased risk from 14% to 9%, whereas adjustment for social factors had virtually no effect. Both diastolic and systolic blood pressure were positively related to deaths occurring after age 65. Adjustment for childhood IQ had no effect on the relative rates and adjustment for social factors had only a very minor effect on the systolic blood pressure-mortality relationship.

(Table 3 about here)

There was an inverse relationship between adult height and all cause mortality (table 3). For each standard deviation lower height, there was an increased risk of mortality over the 25 year period. Adjustment for childhood IQ or for social factors attenuated the relationship, with a cumulative effect when both IQ and social factors were adjusted for. In deaths occurring up to age 65, the relative rate was higher than for the whole 25 year period, and adjustment for either IQ or social factors attenuated the relative rate (from 1.32 to 1.21). Again, adjustment for both IQ and social factors had a cumulative effect. For deaths occurring after age 65, there was a non significant inverse relationship between height and mortality, and adjustment for IQ or social factors had only a small effect. Adjusted FEV1 was inversely related to mortality over 25 years. For each standard deviation lower adjusted FEV1, there was a 24% increased risk of death. Adjustment for either childhood IQ or social factors attenuated the risk. An increased relative rate per standard deviation lower adjusted FEV1 was seen for deaths occurring up to age 65. This was attenuated when adjusting for childhood IQ or social factors. There was also an increased relative rate of mortality for deaths occurring after age 65, although it was smaller, and it was not affected by further adjustment.
Smoking was related to mortality in all three follow-up periods (table 4). Current smokers had more than double the risk of death compared with never smokers. Ex-smokers’ risk was not significantly different from never smokers. Adjustment for childhood IQ attenuated the risk in deaths occurring up to age 65 from 2.01 to 1.91, but had no effect in deaths occurring after age 65. Adjustment for social factors also attenuated the risk of deaths up to age 65 and had virtually no effect on deaths occurring after age 65.

(Table 4 about here)

Discussion
We have shown that childhood IQ was significantly related to deaths occurring up to and including age 65, but not to deaths occurring after age 65. Age 11 IQ has substantial stability to IQ at age 77 (Deary, Whalley, Lemmon, Crawford & Starr, 2000) so may be expected to predict events throughout life. It is possible that the lack of effect of lower childhood IQ at older ages is due to the higher risk people being removed from the population at risk in the later period, since they are more likely to have died in the period up to age 65. The survival curves up to age 65 suggest that a subset of the lowest IQ group died early. After age 65 the survival curves were parallel indicating that the mortality experience was the same for each IQ group. This study differs from the Aberdeen study into childhood IQ and survival to age 76 (Whalley & Deary, 2001), since the current study investigates mortality from age about 50, when the participants took part in the Midspan screening, whereas the Aberdeen study considered survival from age 11. The survival curves from that study were different for men and women, mainly due to the effect on male mortality in the second world war. The current study is one of “survivors”, since all participants had survived to the Midspan screening, so it excludes any deaths occurring before age 50 due to the war, accidents and poor health.

There are several reasons why childhood IQ may be associated with mortality before the age of 65. Higher IQ may be associated with better care of health and lifestyle, which in turn influence mortality risk (Gottfredson & Deary, 2004). Examples here include ability to
understand and comply with correct medication routines, exercise, healthy eating, sensible alcohol consumption and smoking cessation (Taylor et al., 2003). Additionally, there may be shared genes between lower cognition and susceptibility to earlier death from various causes. However, investigation of this will require larger samples and more detailed analyses of the association between specific causes of death and childhood IQ.

We found that risk factors were related to all cause mortality in different ways in the early and late follow-up periods. Relationships were stronger in the later compared to the early period for blood pressure and smoking, and conversely were stronger in the early compared to the later period for height and FEV1. However since mortality rates are higher in the elderly, the absolute risks are higher in older than in younger age groups, even though the relative rates may be lower in the older groups. Our study only found significant relationships in the later period for both diastolic and systolic blood pressure. In the city of Bergen study in Norway, blood pressure in men and women in the age group 45-59 at screening was related to all cause mortality throughout the 20 year follow-up period (Selmer & Tverdal, 1994). In the current study, the relative rates for current smokers compared to never smokers were slightly stronger in the later period compared to the early period. The male British doctors' study found that all cause mortality was double that in continuing cigarette smokers compared to never smokers in age of death groups up to 75 years, similar to our findings (Doll et al., 1994).

Our finding of a stronger relationship of height with all cause mortality in the earlier period compared with the later period confirms results from the Whitehall study (Leon, Davey Smith, Shipley & Strachan, 1995). In that study of male civil servants, the relative rates of all cause mortality for a six inch (15.2cm) lower height were 1.36 (95% confidence interval 1.23 – 1.51) in the first nine years of follow-up, 1.15 (1.03 – 1.30) for 10-14 years of follow-up and 1.04 (0.91 – 1.19) for 15 or more years of follow-up. Adjustment for civil service grade, which is used instead of social class in that study, attenuated the relative rates, as seen in the current study. We found that FEV1 was related to all cause mortality in both the early and late periods. FEV1 was found to be inversely related to all cause mortality for a 29 year follow-up
period divided into five year intervals in the Buffalo Health Study (Schunemann, Dorn, Grant, Winkelstein & Trevisan, 2000). In a study of Swedish men, effort-related breathlessness predicted all cause mortality in the 16 year follow-up period and also in the second half of this period (Rosengren & Wilhelmsen, 1998).

We found that adjustment for childhood IQ had an attenuating effect on the risk factor-mortality relationship in the early follow-up period and no effect in the later follow-up period. The effect in the early period was fairly sizeable. The lack of effect in the later period is consistent with the absence of any significant relationship between childhood IQ and mortality in that later period. It was of interest to see if adjustment for childhood IQ was similar to adjustment for social factors, as these factors are more usually available in cohort studies. Similar to childhood IQ, there was no effect of adjustment for social factors in the later period. In the early period, adjustment for social factors had virtually no effect on the blood pressure-mortality relationship. Blood pressure is not strongly related to socio-economic position in this population (Davey Smith et al., 1998b; Hawthorne et al., 1995), therefore it is unsurprising that adjustment for social factors had little influence on the blood pressure-mortality association. In the early period, adjustment for social factors had a similar effect to adjustment for IQ on the height-mortality and FEV1-mortality relationship. Social factors had a slightly larger effect than IQ on the smoking-mortality relationship in the early period. These observations are consistent with Austin’s ‘ageing well’ hypothesis (Austin, 1991). This hypothesis suggests that there is a portion of any population that fails to reach old age in a state of good health because of social factors over the life course (e.g., poverty, rural residence, poor nutrition, substandard housing, limited educational opportunities etc). Lower childhood IQ may be an important marker for this group who have limited access to a successful ageing lifestyle, but may not relate to outcome in those who reach old age still in relative good health.

It is somewhat unusual to have a measure of mental ability available at a young age and relate this to mortality occurring after age 50. Other studies have related mental ability in later life to mortality risk. Cognitive function in middle age, as measured by the Delayed Word
Recall Test and the Digit Symbol Substitution Test, was inversely related to all cause mortality in the Atherosclerosis Risk in Communities Study after adjustment for potential confounders (Pavlik et al., 2003). The participants were aged between 48 and 67 years when tested and follow-up was measured after an average of 6.3 years. Studies in the USA (Bassuk, Wypij & Berkman, 2000), Australia (Korten et al., 1999) and the UK (Neale et al., 2001) have shown that cognitive function measured in old age also predicts mortality. One interpretation is that, for these studies, the association may be between cognitive change and mortality. That is, cognitive function in old age is reflective of the original IQ level in childhood and the change since then. It might be the degree to which the cognitive score reflects change from a previous level that relates to mortality within old age. We do not have information on later life cognitive function for the present study to test this hypothesis, but future studies could incorporate measures of cognitive function across the lifecourse.

To summarise, we have shown that childhood IQ was related to deaths occurring up to the age of 65, but not after 65. This corresponds to our previous findings on childhood IQ and cause-specific events (Hart et al., 2003b). Social factors acted only to a modest extent as confounders in the IQ-mortality relationship, more strongly in the earlier than the later period. There was an additional effect of childhood IQ on the risk factor-mortality relationship in the early period, which worked in a similar manner to social factors. Childhood IQ can be considered an early life risk factor for adult premature mortality, but further research may uncover the role of cognitive function in later life in this pathway.
Acknowledgements

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Figure Caption

Survival in years by highest and lowest quarter of childhood IQ
Table 1. Number (percentage) of deaths by causes, occurring up to age 65 and after age 65 in 938 participants of the Midspan and SMS1932 studies

<table>
<thead>
<tr>
<th>Cause</th>
<th>Up to age 65</th>
<th>After age 65</th>
<th>All ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td>54 (31.2%)</td>
<td>87 (33.6%)</td>
<td>141 (32.6%)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>68 (39.3%)</td>
<td>83 (32.0%)</td>
<td>151 (35.0%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>17 (9.8%)</td>
<td>32 (12.4%)</td>
<td>49 (11.3%)</td>
</tr>
<tr>
<td>Other cardiovascular disease</td>
<td>6 (3.5%)</td>
<td>7 (2.7%)</td>
<td>13 (3.0%)</td>
</tr>
<tr>
<td>Respiratory disease</td>
<td>4 (2.3%)</td>
<td>32 (12.4%)</td>
<td>36 (8.3%)</td>
</tr>
<tr>
<td>Other causes</td>
<td>24 (13.9%)</td>
<td>18 (6.9%)</td>
<td>42 (9.7%)</td>
</tr>
<tr>
<td>All causes</td>
<td>173</td>
<td>259</td>
<td>432</td>
</tr>
</tbody>
</table>
Table 2. Relative rate of all cause mortality associated with 1 SD decrease in childhood IQ

<table>
<thead>
<tr>
<th></th>
<th>Over 25 years of follow up</th>
<th>Up to age 65</th>
<th>After age 65</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>432</td>
<td>173</td>
<td>259</td>
</tr>
<tr>
<td>Relative rate&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.17 (1.07 - 1.29)</td>
<td>1.36 (1.18 - 1.57)</td>
<td>1.05 (0.93 - 1.19)</td>
</tr>
<tr>
<td>Relative rate&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.12 (1.01 - 1.24)</td>
<td>1.29 (1.10 - 1.51)</td>
<td>1.01 (0.88 - 1.16)</td>
</tr>
</tbody>
</table>

<sup>a</sup> adjusted for sex

<sup>b</sup> adjusted for sex, social class and deprivation category
Table 3. Relative rate of all cause mortality associated with 1 SD change$^a$ in risk factor

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Relative rate adjusted for</th>
<th>Sex</th>
<th>Sex &amp; childhood IQ</th>
<th>Sex, social class &amp; deprivation</th>
<th>Sex, childhood IQ, social class &amp; deprivation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td><strong>Over 25 years of follow up</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>1.22 (1.11 - 1.34)</td>
<td>1.20 (1.09 - 1.32)</td>
<td>1.22 (1.11 - 1.34)</td>
<td>1.21 (1.10 - 1.32)</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1.22 (1.11 - 1.34)</td>
<td>1.20 (1.09 - 1.31)</td>
<td>1.21 (1.10 - 1.33)</td>
<td>1.20 (1.09 - 1.31)</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>1.21 (1.07 - 1.38)</td>
<td>1.17 (1.02 - 1.33)</td>
<td>1.16 (1.02 - 1.32)</td>
<td>1.14 (1.00 - 1.30)</td>
<td></td>
</tr>
<tr>
<td>Adjusted FEV1 (%)</td>
<td>1.24 (1.13 - 1.36)</td>
<td>1.21 (1.11 - 1.33)</td>
<td>1.21 (1.11 - 1.33)</td>
<td>1.20 (1.10 - 1.32)</td>
<td></td>
</tr>
<tr>
<td><strong>Up to age 65</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>1.14 (0.98 - 1.32)</td>
<td>1.09 (0.94 - 1.26)</td>
<td>1.13 (0.98 - 1.32)</td>
<td>1.10 (0.95 - 1.27)</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1.14 (0.99 - 1.33)</td>
<td>1.09 (0.94 - 1.27)</td>
<td>1.14 (0.98 - 1.31)</td>
<td>1.09 (0.94 - 1.27)</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>1.32 (1.08 - 1.61)</td>
<td>1.21 (0.99 - 1.49)</td>
<td>1.21 (0.98 - 1.49)</td>
<td>1.16 (0.94 - 1.43)</td>
<td></td>
</tr>
<tr>
<td>Adjusted FEV1 (%)</td>
<td>1.29 (1.12 - 1.49)</td>
<td>1.24 (1.07 - 1.43)</td>
<td>1.25 (1.08 - 1.44)</td>
<td>1.23 (1.06 - 1.42)</td>
<td></td>
</tr>
<tr>
<td><strong>After age 65</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>1.28 (1.13 - 1.44)</td>
<td>1.28 (1.13 - 1.44)</td>
<td>1.28 (1.13 - 1.44)</td>
<td>1.28 (1.13 - 1.45)</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1.27 (1.13 - 1.43)</td>
<td>1.27 (1.13 - 1.43)</td>
<td>1.26 (1.12 - 1.42)</td>
<td>1.26 (1.12 - 1.42)</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>1.14 (0.97 - 1.35)</td>
<td>1.13 (0.95 - 1.34)</td>
<td>1.12 (0.94 - 1.33)</td>
<td>1.12 (0.94 - 1.33)</td>
<td></td>
</tr>
<tr>
<td>Adjusted FEV1 (%)</td>
<td>1.20 (1.07 - 1.35)</td>
<td>1.20 (1.07 - 1.36)</td>
<td>1.19 (1.05 - 1.34)</td>
<td>1.19 (1.06 - 1.34)</td>
<td></td>
</tr>
</tbody>
</table>

$^a$increase for diastolic blood pressure (SD=12.4) and systolic blood pressure (SD=21.5), decrease for height (SD=9.1) and adjusted FEV1 (SD=21.2)
Table 4. Relative rate of all cause mortality by smoking category

<table>
<thead>
<tr>
<th>Smoking category</th>
<th>Relative rate adjusted for</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sex</td>
</tr>
<tr>
<td>Over 25 years of follow up</td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>1</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2.06 (1.59 - 2.67)</td>
</tr>
<tr>
<td>Ex smoker</td>
<td>0.94 (0.65 - 1.36)</td>
</tr>
<tr>
<td>Up to age 65</td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>1</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2.01 (1.30 - 3.12)</td>
</tr>
<tr>
<td>Ex smoker</td>
<td>1.05 (0.58 - 1.89)</td>
</tr>
<tr>
<td>After age 65</td>
<td></td>
</tr>
<tr>
<td>Never smoker</td>
<td>1</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2.09 (1.51 - 2.90)</td>
</tr>
<tr>
<td>Ex smoker</td>
<td>0.88 (0.55 - 1.41)</td>
</tr>
</tbody>
</table>


