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Childhood IQ and cardiovascular disease in adulthood: prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies

C.L. Hart*[1], M.D. Taylor[2], G. Davey Smith[3], L.J. Whalley[4], J.M. Starr[5], D.J. Hole[1], V. Wilson[6], I.J. Deary[2]

[1]Public Health and Health Policy, Division of Community Based Sciences, University of Glasgow, 1 Lilybank Gardens, Glasgow G12 8RZ

[2]Psychology, School of Philosophy, Psychology and Language Sciences, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ

[3]Department of Social Medicine, University of Bristol, Canynge Hall, Whiteladies Road, Bristol BS8 2PR

[4]Department of Mental Health, University of Aberdeen, Clinical Research Centre, Royal Cornhill Hospital, Aberdeen AB25 2ZH

[5]Royal Victoria Hospital, Craigleith Road, Edinburgh EH4 2DN

[6]The Scottish Council for Research in Education Centre, University of Glasgow, 61 Dublin St, Edinburgh EH3 6NL

* Corresponding author:

Dr Carole Hart
Public Health and Health Policy
Division of Community Based Sciences
University of Glasgow
1, Lilybank Gardens
Glasgow G12 8RZ
Tel. 0141 330 4072
Fax. 0141 330 5018
Email c.l.hart@udcf.gla.ac.uk

Abstract

This study investigated the influence of childhood IQ on the relationships between risk factors and cardiovascular disease (CVD), coronary heart disease (CHD) and stroke in adulthood. Participants were from the Midspan prospective cohort studies which were conducted on adults in Scotland in the 1970s. Data on risk factors were collected from a questionnaire and at a screening examination, and participants were followed up for 25 years for hospital admissions and mortality. 938 Midspan participants were successfully matched with their age 11 IQ from the Scottish Mental Survey 1932, in which 1921-born children attending schools in Scotland took a cognitive ability test. Childhood IQ was negatively correlated with diastolic and systolic blood pressure, and positively correlated with height and respiratory function in adulthood. For each of CVD, CHD and stroke, defined as either a hospital admission or death, there was an increased relative rate per standard deviation decrease (15 points) in childhood IQ of 1.11 (95% confidence interval 1.01-1.23), 1.16 (1.03-1.32) and 1.10 (0.88-1.36) respectively. With events divided into those first occurring before and those first occurring after the age of 65, the relationships between childhood IQ and CVD, CHD and stroke were only seen before age 65 and not after age 65. Blood pressure, height, respiratory function and smoking were associated with CVD events. Relationships were stronger in the early compared to the later period for smoking and FEV1, and stronger in the later compared to the earlier period for blood pressure. Adjustment for childhood IQ had small attenuating effects on the risk factor-CVD relationship before age 65 and no effects after age 65. Adjustment for risk factors attenuated the childhood IQ-CVD relationship by a small amount before age 65. Childhood IQ was associated with CVD risk factors and events and can be considered an important new risk factor.

Keywords

cardiovascular disease, cohort, mental ability, mortality, risk factors, Scotland.

Wordcount

Abstract (299), Main text & references etc (5658)

Introduction

Although rates of coronary heart disease (CHD) and stroke are declining in the west (Office for National Statistics, 1997; Warlow, 1998), they were the cause of 21% and 12% of deaths, respectively, in Scotland in 2000 (Registrar General for Scotland, 2001). Cardiovascular disease (CVD), which includes CHD and stroke, was the underlying cause in 41% of deaths (Registrar General for Scotland, 2001). In addition, CVD is a major contributor to the burden of illness, hospital admissions and disability.

There are well established risk factors for CVD, such as tobacco smoking and raised blood pressure. People with poorer socioeconomic profiles, determined by education, social class and deprivation are known to be at higher risk of CVD (Davey Smith, Hart, Hole, MacKinnon, Gillis, Watt et al., 1998; Davey Smith, Bartley & Blane, 1990; Carstairs & Morris, 1991). Personal traits, such as hostility, submissiveness and the type A personality more generally, have a link to some CVD outcomes (Whiteman, Deary & Fowkes, 2000; Hemingway & Marmot, 1999). Childhood IQ, an intriguing new risk factor, has been found to be inversely related to all cause mortality, CVD and CHD (Whalley & Deary, 2001; Hart, Taylor, Davey Smith, Whalley, Starr, Hole et al., 2003; Osler, Andersen, Due, Lund, Damsgaard & Holstein, 2003). In this paper, we expand our previous findings on mortality to investigate how childhood IQ contributes to CVD events (hospital admissions and deaths). We next examine the associations between childhood IQ and specific CVDs, viz., CHD and stroke. Given the suggestion that childhood IQ influences health behaviours throughout life (Deary, Whalley & Starr, 2003), and the fact that childhood IQ is associated with birthweight (Shenkin, Starr, Pattie, Rush, Whalley & Deary, 2001), which is also linked to CVD in adulthood (Barker, 1994), it is possible that childhood IQ is associated with CVD outcomes relatively early in adult life. We therefore also explore whether the effects of childhood IQ are different in CVD, CHD and stroke occurring before or after the age of 65. Lastly we hypothesise that the effect of childhood IQ on CVD may be mediated by traditional risk factors for CVD.

Methods

The Scottish Mental Survey 1932 (SMS1932), conducted under the auspices of the Scottish Council for Research in Education (SCRE), obtained data about the whole distribution of the intelligence of Scottish pupils (Scottish Council for Research in Education, 1933). On June 1 1932, children born in the calendar year 1921 and attending schools in Scotland were given the same, well-validated mental ability test. The total number of children who sat the Moray House Test was 87,498 (44,210 boys and 43,288 girls). As far as we are aware, no other country collected information about the childhood mental ability differences of an entire year-of-birth cohort. The Moray House test is described in detail elsewhere (Deary, Whalley, Lemmon, Crawford & Starr, 2000). Briefly, it has 71 items with a maximum possible score of 76. The time limit was 45 minutes for completion. It contains a preponderance of verbal reasoning items but also some numerical, spatial and abstract reasoning items. The score is expressed as a total score; there are no subscores.

The Midspan studies were large cardiorespiratory studies of adults carried out in Scotland in the 1960s and 1970s. Two studies are included in this analysis, the Collaborative study and the Renfrew/Paisley study. The Collaborative study was conducted between 1970 and 1973 in 27 workplaces in the west and central belt of Scotland (Davey Smith, Hart, Hole et al., 1998). 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 and involved 7,052 men and 8,354 women aged 45-64 years who were resident in Renfrew and Paisley (Hawthorne, Watt, Hart, Hole, Davey Smith & Gillis, 1995). The age ranges of the studies were such that some participants 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 who were born in 1921 and of these, 938 (75%) were matched to a mental ability score from the SMS1932. Full details of the matching procedures are reported elsewhere (Hart, Deary, Taylor, MacKinnon, Davey Smith, Whalley et al., 2003).

Since the children's ages varied between 10½ and 11½ years at the time of testing, the test scores were corrected for age in days and converted to usual IQ-type scores with mean 100 and standard deviation 15.

In both Midspan studies, participants completed a questionnaire and attended a physical examination (Davey Smith, Hart, Hole et al., 1998; Hawthorne, Watt, Hart 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. In these cases, the social class 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 measure is an 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. 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 measurement of blood pressure, height, weight, respiratory function, plasma cholesterol and blood glucose (in the Renfrew/Paisley study only). Blood pressure was measured with the subject seated, using the London School of Hygiene sphygmomanometer, with a cuff of 12 x 22 cm. Diastolic pressure was recorded at the disappearance of the fifth Korotkoff sound. Two blood pressure readings were taken and the average diastolic and systolic pressure were recorded. 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, Hart, Hole et al., 1998; Hole, Watt, Davey Smith, Hart, Gillis & Hawthorne, 1996). Body mass index in kg/m^2 was calculated from the weight and height.

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 were provided. Underlying causes of death were identified and grouped as all cardiovascular disease (CVD) (ICD9 codes 390-459), coronary heart disease (CHD) (ICD9 410-414) and stroke (ICD9 430-438). The few more recent deaths were coded in ICD10 and equivalent codes were used for each underlying cause. In addition, a computerised linkage with acute hospital discharges in Scotland provided records of acute hospital admissions in up to 25 years of follow-up, although hospital admissions were only provided to the end of 1995 for Renfrew/Paisley study participants (Hanlon, Walsh, Whyte, Scott, Lightbody & Gilhooly, 1998). First occurrence of each cause of hospital admission from the discharge diagnosis was coded in the same way as the deaths.

Cox's models (Cox, 1972) were used to calculate proportional hazards regression coefficients for one standard deviation change in risk factor and for current and ex smokers compared to never smokers. The exponentiated proportional hazards regression coefficients are referred to as relative rates. Survival time in 25 years was taken from the date of screening until either the date of hospital admission, or the date of death if no hospital admission was found. One participant had embarked from the UK during the follow-up period and survival time was taken until the date of embarkation. Hospital admissions and deaths for the whole follow-up period were divided into those first occurring up to and including age 65 (the early period) and those first occurring after age 65 (the later period).

Results

There were significant negative correlations between childhood IQ and both adulthood diastolic blood pressure and systolic blood pressure, meaning that people with higher childhood IQ had lower blood pressure in adulthood (table 1). We have reported this relationship elsewhere (Starr, Taylor, Hart, Davey Smith, Whalley, Hole et al., 2003). There were significant positive correlations between childhood IQ and height and respiratory function, meaning that people with higher childhood IQ were taller and had better respiratory function in adulthood. There were no significant correlations between childhood IQ and blood sugar or cholesterol in adulthood. Partial correlation coefficients controlling for sex were nearly identical to the full correlation coefficients (not shown). The relationship between childhood IQ and smoking has been reported elsewhere (Taylor, Hart, Davey Smith, Starr, Hole, Whalley et al., 2003). 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]).

Of the 938 men and women who matched to a childhood IQ score (59% men, 41% women), 400 had a hospital admission for or died from CVD, 238 from CHD and 82 from stroke in the 25 year follow-up period (table 2). There was an 11% increased relative rate of CVD per 15 points lower childhood IQ over the 25 year follow-up period. In the early period (up to age 65), there was a 22% increased relative rate of CVD. There was no effect of childhood IQ on CVD occurring in the later period (after age 65). Additional adjustment for social class and deprivation category in mid-life only slightly attenuated the relative rate in the early follow-up period, to 19%. Adjustment was then made separately for the risk factors which were significantly correlated with childhood IQ. Adjusted FEV1, diastolic and systolic blood pressure very slightly attenuated the relative rates associated with IQ in the early period. Adjustment for height and smoking also had very small attenuating effects. There were similar findings for the relationships between childhood IQ and CHD. There was a 16% increased relative rate for CHD per 15 points lower childhood IQ over the 25 year period and a 29% increased relative rate for CHD in the early period - with no effect of childhood IQ for CHD in the later period. Adjustment for social factors had little effect on the relative rate in the early period. There were small attenuating effects after adjustment for diastolic and systolic blood pressure and smoking, with no effect for height and a very small effect for adjusted FEV1. For stroke, there was a 47% increased relative rate per 15 points lower childhood IQ in the early period and no effect in the later period. Small attenuating effects were seen after adjustment for social factors, diastolic blood pressure, adjusted FEV1 and smoking.

The next analyses examined the contributions of the risk factors to CVD and investigated whether their effects were attenuated after adjusting for childhood IQ. There was an increased relative rate of CVD for a standard deviation increase in diastolic blood pressure for the whole follow-up period and the early and later periods (table 3). The stronger effect was seen for CVD occurring after age 65. Adjusting for childhood IQ slightly reduced the relative rate in the early period but had no effect on the later period. Results for systolic blood pressure were similar, although adjusting for childhood IQ slightly increased the relative rate in the later period. The relative rate of CVD for a standard deviation decrease in height was similar in all three follow-up periods and was about 10% higher for each standard deviation decrease in height, although not statistically significant at conventional levels. Adjusting for childhood IQ had a small attenuating effect in the early follow-up period and no effect in the later period. There was an increased relative rate per standard deviation decrease in adjusted FEV1 for each follow-up period. The effect was stronger in the early period. Adjusting for childhood IQ had an small attenuating effect in the early period and no effect in the later period.

Current smokers had a higher relative rate of CVD compared to never smokers in the 25 year follow-up period. Adjustment for childhood IQ had a small attenuating effect. The relative rate for ex-smokers was not different from the never smokers. In the early follow-up period, current smokers had an 84% increased relative rate which was reduced to 78% after adjusting for childhood IQ. In the later follow-up period, the increased relative rate for current smokers was smaller and was not altered by adjustment for childhood IQ. Similar results were found for CHD and stroke (not shown).

Discussion

We have shown that childhood IQ was significantly related to CVD, CHD and stroke events occurring up to and including age 65, but not to these events occurring after age 65. Age 11 IQ has been shown to have substantial stability to IQ at age 77 (Deary, Whalley, Lemmon et al., 2000; Crawford, Deary, Starr & Whalley, 2001) 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 had an event in the period up to age 65. It is also possible that these medical events have differences in their mechanisms before and after age 65, and that childhood IQ partakes only in the former. It is not likely that the effect of IQ at age 11 is only related to events occurring between ages 50 and 65 and not to events occurring after age 65 due to the greater time difference between the measurement of IQ and the event. This is more likely to be the case when the variable of interest was measured nearer the time of the event, such as at the Midspan screening when the participants were about 50 years of age.

We found that diastolic blood pressure, systolic blood pressure, adjusted FEV1, smoking and, to a lesser extent height, acted in addition to childhood IQ in the relationships with CVD, CHD and stroke occurring up to age 65. The relative rates based on IQ score remained statistically significant after adjusting for these factors. This would suggest that childhood IQ is an important factor which is only slightly mediated by classical CVD risk factors measured in middle age. When looking the other way round, at the effect of adjusting for childhood IQ on the risk factor-CVD relationship, we found that adjustment for childhood IQ had a small attenuating effect in the early follow-up period and no effect in the later follow-up period. The lack of effect in the later period is consistent with the absence of any significant relationship between childhood IQ and events in that later period.

Risk factors were related to CVD in different ways in the early and late follow-up periods. Relationships were stronger in the early compared to the later period for smoking and FEV1, and conversely were stronger in the later compared to the earlier period for blood pressure. In the male British doctors study, there was a progressive reduction in the ratio of CVD mortality in continuing cigarette smokers to that in non-smokers, as the age at death increased (from 2.1 at ages under 65, to 1.7 at age 65-74, 1.4 at age 75-84 and 1.2 at ages 85 and over) (Doll, Peto, Wheatley, Gray & Sutherland, 1994). In a Swedish study of 50 year old men, smoking was significantly related to myocardial infarction or fatal CHD in the first 15 years, but not in the last 10 years of follow-up (Welin, Eriksson, Larsson, Svardsudd, Wilhelmsen & Tibblin, 1993). In cohort studies, since smokers die on average at younger ages than non smokers, they are removed from the population at risk (Marang-van de Mheen, Shipley, Witteman, Marmot & Gunning-Schepers, 2001). This attrition may be the reason behind the decrease in relative risk in later periods. FEV1 was found to be inversely related to CHD 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 mortality from CVD in the 16 year follow-up period and also in the second half of this period (Rosengren & Wilhelmsen, 1998). Studies on blood pressure and ageing have been less consistent (Tate, Manfreda & Cuddy, 1998). In the Swedish study of 50 year old men, systolic blood pressure was predictive of myocardial infarction or fatal CHD in the first 15 years and more weakly in the following 10 year follow-up period (Welin, Eriksson, Larsson et al., 1993). A recent meta-analysis of 61 prospective studies found lower proportional differences with increasing age of death, for either systolic or diastolic blood pressure and CHD, stroke or other vascular deaths (Prospective Studies Collaboration, 2002).

In the Midspan cohorts we have the availability of mid-life risk factors for later CVD, CHD and stroke. Childhood IQ is associated, because of the stability of IQ over the lifespan (Deary, Whalley, Lemmon et al., 2000), with life style – personal behaviours and environmental exposures – that, together with the individual's genetic predisposition, lead to these specific risks. By middle age, childhood IQ will already be associated with risk modification (for example cessation of cigarette smoking (Taylor, Hart, Davey Smith et al., 2003)), but its effect may not be completely worked out. For example, more people with higher childhood IQs may continue to quit smoking more often than those with lower childhood IQs. This effect will diminish as the 'pool' of smokers with higher IQs is reduced. On the other hand, hypertension is more commonly diagnosed and treated as people enter old age. Any differential effect of childhood IQ on behaviours such as on attending health care services for blood pressure checks, obtaining and compliance with antihypertensive medication, would be expected to be seen as the cohort ages. However, our data do not provide any evidence for this later effect of childhood IQ. For stroke, lower childhood IQs may reflect relatively retarded intra-uterine growth (Shenkin, Starr, Pattie et al., 2001), that in itself predisposes to premature stroke in ways not directly mediated by elevated blood pressure. Perhaps, in view of the minimal effect of social factors seen once childhood IQ was adjusted for, childhood IQ is a proxy that captures socio-economic disadvantage, health behaviours and environmental exposures more completely than other more conventional socio-economic indices in a situation where it is the combination of many factors, rather than any individual factor conferring major risk, that is important.

A possible data limitation concerns the hospital admissions data. Although the mortality information is available for deaths occurring anywhere in the UK, the hospital admissions data is only provided for hospital admissions occurring in Scotland. There may be some participants who moved from Scotland to the rest of the UK in the 25 year follow up period. Any hospital admissions between screening and leaving Scotland would be provided, but no hospital admissions would be available if they occurred in England for example. However we do not expect that this is a major limitation as 86% of the RP participants successfully linked to a hospital admission or death occurring in Scotland (Hanlon, Walsh, Whyte et al., 1998). Findings were not likely to have been biased by our not being able to match 25% of the 1921-born participants to a mental ability score (Hart, Deary, Taylor et al., 2003). There were 95 participants who matched to a SMS1932 record, but had no mental ability score recorded as they were probably absent from school on that day. The remaining participants who did not match to a SMS1932 record would have included migrants to the Midspan areas after age 11. Characteristics of these unmatched participants were similar to matched participants, but those not matched had fewer siblings, were heavier and their fathers were less likely to have had a manual occupation than those who were matched; also unmatched men were taller. These differences were unlikely to either over or under estimate the results to any sizeable extent.

To summarise, we have shown that childhood IQ was related to CVD, CHD and stroke occurring up to the age of 65, but not after 65. Childhood IQ was associated with CVD risk factors in adulthood (smoking, blood pressure, height and respiratory function) and it contributed a small amount to the risk factor-CVD relationship. Each risk factor explained a small amount of the IQ-CVD relationship. Childhood IQ is therefore an important factor, independent of the measures we had of traditional risk factors, in future CVD events occurring up to age 65. Future work will investigate the effect of childhood IQ on all cause mortality before and after age 65 and its relationship with risk factors.

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TABLE 1 *Correlation coefficients between childhood IQ and Midspan risk factors for cardiovascular disease*

Risk factor	N	Mean	Standard deviation	Correlation coefficient
Diastolic blood pressure (mmHg)	937	84.4	12.4	-0.12**
Systolic blood pressure (mmHg)	937	142.8	21.5	-0.16**
Height (cm)	937	165.6	9.1	0.24**
Adjusted FEV1 (%)	937	93.0	21.2	0.15**
Blood sugar[a] (mmol/L)	443	5.0	1.3	-0.03
Plasma cholesterol (mmol/L)	930	6.2	1.1	0.04
Body mass index (kg/m^2)	937	25.6	3.8	-0.04

[a] Only available for Renfrew/Paisley study participants
** significant at 0.01 level

TABLE 2 *Relative rate (and 95% confidence interval) of CVD, CHD and stroke hospital admission or death associated with 1 SD decrease in childhood IQ*

	Over 25 years of follow up	Up to age 65	After age 65
CVD			
Number	400	203	197
Relative Rate adjusted for:			
Sex	1.11 (1.01 - 1.23)	1.22 (1.06 - 1.39)	0.99 (0.85 - 1.14)
Sex, social class & deprivation category	1.08 (0.97 - 1.21)	1.19 (1.03 - 1.38)	0.98 (0.83 - 1.15)
Sex, diastolic blood pressure	1.08 (0.98 - 1.19)	1.19 (1.04 - 1.36)	0.97 (0.84 - 1.12)
Sex, systolic blood pressure	1.06 (0.96 - 1.17)	1.18 (1.03 - 1.36)	0.93 (0.81 - 1.08)
Sex, height	1.10 (1.00 - 1.22)	1.21 (1.05 - 1.39)	1.00 (0.86 - 1.15)
Sex, adjusted FEV1	1.09 (0.99 - 1.20)	1.18 (1.03 - 1.36)	1.00 (0.86 - 1.15)
Sex, smoking	1.09 (0.99 - 1.21)	1.20 (1.04 - 1.37)	0.99 (0.86 - 1.15)
CHD			
Number	238	108	130
Relative Rate adjusted for:			
Sex	1.16 (1.03 - 1.32)	1.29 (1.08 - 1.55)	1.06 (0.89 - 1.26)
Sex, social class & deprivation category	1.13 (0.98 - 1.31)	1.30 (1.06 - 1.60)	1.00 (0.82 - 1.21)
Sex, diastolic blood pressure	1.12 (0.99 - 1.28)	1.26 (1.05 - 1.51)	1.02 (0.85 - 1.21)
Sex, systolic blood pressure	1.11 (0.98 - 1.26)	1.25 (1.04 - 1.50)	1.00 (0.84 - 1.19)
Sex, height	1.16 (1.02 - 1.32)	1.29 (1.07 - 1.56)	1.06 (0.88 - 1.26)
Sex, adjusted FEV1	1.16 (1.02 - 1.31)	1.28 (1.07 - 1.54)	1.05 (0.88 - 1.26)
Sex, smoking	1.14 (1.00 - 1.29)	1.26 (1.05 - 1.51)	1.04 (0.88 - 1.24)
Stroke			
Number	82	28	54
Relative Rate adjusted for:			
Sex	1.10 (0.88 - 1.36)	1.47 (1.03 - 2.09)	0.92 (0.70 - 1.22)
Sex, social class & deprivation category	1.06 (0.83 - 1.35)	1.40 (0.95 - 2.08)	0.89 (0.66 - 1.22)
Sex, diastolic blood pressure	1.07 (0.86 - 1.33)	1.44 (1.01 - 2.06)	0.90 (0.68 - 1.19)
Sex, systolic blood pressure	1.06 (0.85 - 1.32)	1.46 (1.02 - 2.10)	0.88 (0.67 - 1.17)
Sex, height	1.09 (0.87 - 1.36)	1.47 (1.02 - 2.11)	0.92 (0.69 - 1.22)
Sex, adjusted FEV1	1.04 (0.83 - 1.30)	1.41 (0.98 - 2.03)	0.87 (0.65 - 1.16)
Sex, smoking	1.06 (0.85 - 1.32)	1.41 (0.98 - 2.01)	0.90 (0.68 - 1.19)

TABLE 3 *Relative rate (and 95% confidence interval) of CVD hospital admission or death associated with 1 SD change[a] in risk factor and by smoking category*

	Relative rate adjusted for	
Risk factor	Sex	Sex & childhood IQ
Over 25 years of follow up		
Diastolic blood pressure	1.27 (1.15 - 1.40)	1.25 (1.13 - 1.38)
Systolic blood pressure	1.34 (1.22 - 1.48)	1.33 (1.21 - 1.46)
Height	1.10 (0.96 - 1.25)	1.06 (0.93 - 1.22)
Adjusted FEV1	1.15 (1.04 - 1.26)	1.13 (1.03 - 1.24)
Smoking category		
Never smoker	1	1
Current smoker	1.49 (1.16 - 1.93)	1.47 (1.14 - 1.90)
Ex smoker	1.03 (0.74 - 1.45)	1.05 (0.74 - 1.47)
Up to age 65		
Diastolic blood pressure	1.22 (1.06 - 1.39)	1.18 (1.03 - 1.36)
Systolic blood pressure	1.24 (1.08 - 1.42)	1.20 (1.05 - 1.38)
Height	1.09 (0.90 - 1.31)	1.02 (0.84 - 1.24)
Adjusted FEV1	1.21 (1.06 - 1.38)	1.18 (1.03 - 1.35)
Smoking category		
Never smoker	1	1
Current smoker	1.84 (1.23 - 2.75)	1.78 (1.19 - 2.66)
Ex smoker	1.43 (0.87 - 2.35)	1.45 (0.88 - 2.39)
After age 65		
Diastolic blood pressure	1.33 (1.15 - 1.53)	1.33 (1.15 - 1.54)
Systolic blood pressure	1.46 (1.27 - 1.68)	1.49 (1.29 - 1.71)
Height	1.10 (0.91 - 1.33)	1.10 (0.91 - 1.34)
Adjusted FEV1	1.09 (0.95 - 1.25)	1.09 (0.95 - 1.26)
Smoking category		
Never smoker	1	1
Current smoker	1.29 (0.93 - 1.81)	1.29 (0.93 - 1.81)
Ex smoker	0.79 (0.49 - 1.28)	0.79 (0.49 - 1.27)

[a] increase for diastolic blood pressure and systolic blood pressure, decrease for height and adjusted FEV1