Childhood IQ and social factors on smoking behaviour, lung function and smoking-related outcomes in adulthood: linking the Scottish Mental Survey 1932 and the Midspan studies.

Michelle D. Taylor[1], Carole L. Hart[2], George Davey Smith[3], John M. Starr[4], David J. Hole[2], Lawrence J. Whalley[5], Valerie Wilson[6], Ian J. Deary[1*]

[1]Department of Psychology, University of Edinburgh, UK

[2]Public Health and Health Policy, Division of Community Based Sciences, University of Glasgow, UK

[3]Department of Social Medicine, University of Bristol, UK

[4]Royal Victoria Hospital, Edinburgh, UK

[5]Department of Mental Health, University of Aberdeen, UK

[6]The Scottish Council for Research in Education Centre, University of Glasgow, UK

Running title: Childhood IQ, smoking and health

Word count: 4150

*Requests for reprints should be addressed to Professor Ian J Deary, Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh, EH8 9JZ (e-mail: I.Deary@ed.ac.uk)

Childhood IQ and social factors on smoking behaviour, lung function and smoking-related outcomes in adulthood: linking the Scottish Mental Survey 1932 and the Midspan studies.

Objective: To investigate the associations of childhood IQ and adult social factors, and smoking behaviour, lung function (FEV_1), and smoking-related outcomes in adulthood.

Design: Retrospective cohort study.

Method: Participants were from the Midspan prospective studies conducted on Scottish adults in the 1970s. The sample consisted of 938 Midspan participants born in 1921 who were successfully matched with their cognitive ability test results on the Scottish Mental Survey 1932.

Results: Structural equation modelling showed that age 11 IQ was not associated with smoking consumption directly but IQ and adult social class had indirect effects on smoking consumption via deprivation category. The influence of IQ on FEV_1 was partly indirect via social class. Sex influenced smoking consumption and also IQ and social class. There was a 21% higher risk of having a smoking-related hospital admission, cancer, or death during 25 years of follow-up for each standard deviation disadvantage in IQ. Adjustment for adult social class, deprivation category and smoking reduced the association to 10%.

Conclusion: Childhood IQ was associated with social factors which influenced lung function in adulthood but was not associated directly with smoking consumption. In future studies, it is important to consider other pathways which may account for variance in the link between childhood IQ and health in later life.

Introduction

The prevalence of smoking has declined since the 1950s following the publication of studies linking cigarette smoking with the development of lung cancer (Doll & Hill, 1950; Levin, Goldstein & Gerhardt, 1950; Schairer & Schöniger, 1943; Wynder & Graham, 1950), and health publicity effects have led to an increase in the number of smokers who want to stop (Townsend, Roderick & Cooper, 1994). Among people who continue to smoke in later life the risk of lung cancer may be greater than previous estimates, due to the number of cigarettes that these individuals have smoked throughout their life (Peto *et al*., 2000). Understanding the factors that influence smoking behaviour is important for health promotion and interventions to encourage smoking cessation.

Smoking is a major risk factor for disease and mortality. One of the ways that smoking affects a person's health is by causing a decline in lung function (Xu, Dockery, Ware, Speizer & Ferris, 1992). Poor lung function in adulthood has been associated with all-cause mortality (Hole *et al*., 1996; Schunemann, Dorn, Grant, Winkelstein & Trevisan, 2000), respiratory disease (Ebi-Kryston, 1988; Peto *et al*., 1983), lung cancer (Tockman, Anthonisen, Wright & Donithan, 1987; Van Den Eeden & Friedman, 1992), cardiovascular disease (Ebi-Kryston, 1988; Persson et al., 1986), and stroke (Strachan, 1991; Welin, Svardsudd, Wilhelmsen, Larsson & Tibblin, 1987). The association between lung function and mortality may be explained by smoking status (Marcus, Curb, MacLean, Reed & Yano, 1989), but others have found evidence for a relationship between impaired lung function and mortality risk in never-smokers which suggests that smoking cannot be the sole underlying cause (Hole *et al*., 1996).

Although the prevalence of smoking has declined, some studies suggest that socio-economic and educational differences in smoking have increased (Iribarren, Luepker, McGovern, Arnett & Blackburn, 1997; Peto *et al*., 2000; Pierce, Fiore, Novotny, Hatziandreu & Davis, 1989; Townsend *et al*., 1994). This may be a result of higher cessation rates among people from higher socio-economic groups. In previous studies, older age, higher educational attainment and higher socio-economic position were consistent predictors of successful smoking cessation (Coambs & Kozlowski, 1992; Freund, D'Agostino, Belanger, Kannel & Stokes, 1992; Osler & Prescott, 1998; Tillgren, Haglund, Lundberg & Romelsjö, 1996), whereas people in lower socio-economic groups (Chassin, Presson, Rose & Sherman, 1996; Clark, Kviz,

Crittenden & Warnecke, 1998; Osler & Prescott, 1998; Tillgren *et al*., 1996), and people who smoke more heavily (Nordstrom *et al*., 2000) were more likely to continue smoking. For example, in the Dutch GLOBE study, lower educated people were more likely to continue smoking during 6.5 years of follow-up (Droomers, Schrijvers & Mackenbach, 2002). Poor perceived health and earlier initiation of smoking were the main predictors of the educational differences in smoking in this group.

Previous analyses using the combined databases of the Midspan studies and the Scottish Mental Survey 1932 (SMS1932) found no difference in childhood IQ scores between those individuals who had ever-smoked and those who had never-smoked (Taylor *et al*., 2003). However, current cigarette smokers (at midlife in the 1970s) had significantly lower childhood mental ability scores than past smokers. In this sample there was a 19% advantage in giving-up smoking by midlife associated with a one standard deviation increase in childhood IQ after adjusting for adult social class and deprivation (Taylor *et al*., 2003). Our data have allowed us to examine smoking cessation over a considerably longer period than the GLOBE study and have the advantage of having mental ability data prior to the initiation of smoking for 99.3% of the ever-smokers. In the present study we extend our analyses to examine the associations of childhood IQ and social factors, and smoking behaviour, lung function and smoking-related outcomes in mid-life and later-life.

Higher mental ability is related to more favourable educational and occupational outcomes (Neisser *et al*., 1996). Childhood IQ predicts age at death, even after adjusting for socioeconomic factors (Hart *et al*., 2003; Whalley & Deary, 2001). One possible cause of these associations is via their impact on health behaviours such as smoking. However, no studies to our knowledge have been able to examine childhood intelligence and a range of socio-economic measures in relation to smoking behaviour and smoking-related outcomes in adulthood. Childhood IQ data has the advantage of measuring ability prior to the influence of formal tertiary and secondary education, and the initiation of health behaviours such as smoking. In addition, childhood IQ at age 11 has been shown to remain remarkably stable up to age 77 years (Deary, Whalley, Lemmon, Crawford & Starr, 2000).

The present study combines information on childhood intelligence (age 11) with self-reported mid-life smoking, objectively assessed mid-life lung function (at age about 48-55), and

subsequent smoking-related health outcomes (up to about age 80) in a cohort of Scottish people born in 1921. They took part in both the Midspan surveys of health in the west of Scotland in midlife in the early 1970s (Davey Smith, Hart & Hole *et al*., 1998; Hawthorne *et al*., 1995), and in the Scottish Mental Survey 1932 at age 11 (Deary *et al*., 2000). We investigate (i) whether childhood IQ is linked to smoking consumption and lung function and, if so, whether these links are mediated by social factors in adulthood, and (ii) whether childhood IQ is linked to smoking-related health events and, if so, whether these smoking-related health events can be explained by social factors and smoking in adulthood.

Method

Early life data

Data on childhood mental ability were obtained from the Scottish Mental Survey 1932 (SMS1932) which tested mental ability, using a version of the Moray House Test No. 12 (MHT), in almost all 1921-born children attending school in Scotland on the 1 June 1932 (N = 87 498) (Scottish Council for Research in Education [SCRE], 1933). The MHT has a maximum score of 76. It contains several general, spatial, and numerical reasoning items including directions, same-opposites, word classification, analogies, practical, reasoning, proverbs, arithmetic, spatial, mixed sentences and cipher decoding items. The validity of the MHT was established by individually retesting a representative sample of 1000 children on the Stanford Revision of the Binet-Simon Scale (SCRE, 1933).

Midlife data

The social, lifestyle and health data were from two of the Midspan prospective cohort studies—the Collaborative study and the Renfrew/Paisley study. The Collaborative study consisted of workers (6022 men, 1006 women) who were located in 27 workplaces in the west and central belt of Scotland between 1970 and 1973 (Davey Smith, Hart & Hole *et al*., 1998). The Renfrew/Paisley study was based on a population cohort of people (7052 women, 8354 men), aged between 45 and 64 years, who were resident in the towns of Renfrew and Paisley, close to the city of Glasgow, between 1972 and 1976 (Hawthorne *et al*., 1995). The response rate was 79% in Renfrew and 78% in Paisley. Participants completed a questionnaire and attended a physical examination.

Occupational social class was coded according to the Registrar General's Classification (General Register Office, 1966). The home address of the participants at the time of screening was retrospectively postcoded to assign deprivation category as defined by Carstairs and Morris (1991). Deprivation scores were converted to seven categories ranging from 1 (least deprived) to 7 (most deprived). Participants were classified as never, past or current smokers at midlife. Past smokers were ex-smokers of one year or more. Twelve pipe or cigar only smokers were excluded. The amount smoked was measured by asking the number of cigarettes smoked per day.

As part of the Midspan physical examination, forced expiratory volume in one second (FEV_1) was assessed and the better of two expirations was recorded. To provide an estimate of the participant's impairment, the expected FEV_1 was calculated from linear regression equations of age (years) and height (cm) from each survey and from each sex. The expected coefficients were obtained from a healthy subset of the study populations (Davey Smith, Hart & Hole *et al*., 1998; Hole *et al*., 1996). The adjusted FEV_1 was defined as the actual FEV_1 as a percentage of the expected FEV_1.

Later life data

Midspan participants were flagged at the National Health Service Register in Edinburgh and causes and dates of deaths were provided (Hart *et al*., 2003). A computerised linkage with acute hospital discharges in Scotland provided records of acute hospital admissions and cancer registrations (Hanlon *et al*., 1998). Smoking-related events were categorised as a smoking-related hospital admission, cancer or death during 25 years of follow-up after the Midspan screening examination for never smokers and past smokers as well as for current smokers. Smoking-related admissions or deaths were defined as chronic obstructive pulmonary disease and allied conditions (ICD9 490-496) and the equivalent in ICD10. Smoking-related cancers or deaths were mouth & pharynx (ICD9 140-149), oesophagus (ICD9 150), larynx (ICD9 161), lung (ICD9 162), pancreas (ICD9 157) and bladder (ICD9 188). These are regarded by the International Agency for Research on Cancer (IARC, 1986) as diseases that can be caused by smoking. Results of a study by Doll and colleagues (1994) found that cancers of the "upper respiratory sites", lung, and oesophagus were closely related to mortality among smokers; mortality among heavy smokers was at least 15 times that in non-smokers (Doll, Peto, Wheatley, Gray & Sutherland, 1994). Cancer of the bladder and pancreas were about three times more common in heavy smokers than non-smokers (Doll et al, 1994).

Sample

Permission to link the Midspan and SMS1932 studies was obtained from the Multi-Centre Research Ethics Committee for Scotland. The procedures used in the linking of the Midspan studies and the SMS1932 are described elsewhere (Hart *et al*., 2003). Participants who were

matched to an MHT score were between 48 and 55 years of age at the time of Midspan screening. Of the 1921-born Midspan participants, a total of 1032 out of 1251 people were finally matched with an SMS1932 record. Of those matched, 938 (91%) had an MHT score recorded (552 men, 386 women). The MHT scores were corrected for age (in days) at the time of testing and converted to IQ-type scores with a mean 100 and standard deviation 15 (Hart *et al*., 2003). The analysis was conducted on 905 participants, including non-smokers, who had complete data. The participants with missing data did not differ significantly from the sample with complete data.

Analyses

The data were analysed using SPSS for Windows (version 11.0) and the EQS Structural Equation Modelling Program (Bentler, 1995). Chi-square analysis and t-tests were used to compare the number (%) of people in each smoking status category and differences in smoking variables and FEV_1 between those participants who were matched and not matched to a MHT score. Pearson's product-moment correlation coefficients and, where appropriate, Spearman's correlations, were used to investigate the relationships between variables. Structural equation modelling (SEM) was performed using the EQS program (Bentler, 1995) to test the relationships among the variables. Sex, adult social class and deprivation category were categorical variables. This technique allowed us to examine the hypothesis that the effect of childhood IQ on smoking and FEV_1 was mediated through lifestyle and occupational factors. Robust methods were used for analysis. Chi-square, residual covariance and fit indices were used to indicate the goodness of fit of the model to the data (Bentler, 1995). Cox's models were used to calculate proportional hazard ratios of having a smoking-related event in 25 years of follow-up, associated with a one standard deviation decrease in IQ. The survival time was the time between Midspan screening and the first associated event.

Results

Matched versus non-matched participants

Participants who were matched did not differ from participants who were not matched to an MHT score in the number of cigarettes smoked per day and FEV_1. The proportion of never smokers, ex-smokers and current smokers at midlife was similar for the participants who were matched and not matched to a MHT score.

Correlations

Correlations between childhood IQ, adult social class, deprivation category, and the number of cigarettes smoked per day and FEV_1 at midlife were examined for men (n = 534) and women (n = 371) separately. Lower childhood IQ was associated with poorer adult social class (men r = -.43, p < .001; women r = -.35, p < .001) and worse deprivation category (men r = -.27, p < .001; women r = -.22, p < .001). There was no significant association between childhood IQ or adult social class and the number of cigarettes smoked per day. In women, worse deprivation category was associated with smoking a greater number of cigarettes per day (r = .18, p < .001) but this relationship was not significant in men. Lower childhood IQ scores were significantly associated with poorer lung function in adulthood with similar, small effect sizes in men and women (r = .16, p < .001 and r = .14, p < .01, respectively). After controlling for smoking in adulthood, the relationship between childhood IQ and FEV_1 remained significant (men: partial r = .17, p < .001; women: partial r = .14, p < .01). In women, smoking a greater number of cigarettes per day was associated with poorer lung function (r = -.31, p < .001). The association between the number of cigarettes smoked per day and lung function was smaller in men but tended towards significance (r = -.08, p = .06).

Structural equation modelling

Structural equation modelling (SEM) was used to examine the influence of childhood IQ and social and lifestyle factors in adulthood on the number of cigarettes smoked per day and FEV_1 at midlife. When specifying the theoretical model to be tested it was hypothesised that (i) early life variables influence later life variables, (ii) social and lifestyle factors influence health behaviours, (iii) adult social class is directly associated with deprivation category, (iv) smoking

influences lung function, and (v) there will be direct and indirect pathways. The model that resulted from the SEM exercise is shown in Figure 1. The arrows represent the hypothesised directions of influence. The values placed next to the arrows are the parameter weights estimated by the model fitting procedures; these values may be squared to give the percentage of variance shared by adjacent variables. The Wald test was used to indicate non-significant parameters that should be dropped from the model and the Lagrange multiplier suggested pathways that could be added to improve the goodness of fit. All of the parameters in the model were significant and the model had a good fit to the data. The chi-square value for the model is 3.05 (*d.f.* = 5, p = .69). Non-significant values indicate that the model has a good fit to the data. The average of the off-diagonal absolute standardised residuals is 0.009; values of less than 0.04 typically indicate good fit. These tests indicate that the residual covariance among the variables is low after the model's paths are taken into account. Three further indices reflect how well the model fits the variables' covariance matrix. A good fit is indicated by values equal to or greater than 0.9. For the present model, the Bentler-Bonnett (normed and non-normed) and comparative fit indices were 0.994, 1.012 and 1.0, respectively, indicating that the model had a good fit.

Insert Figure 1 about here

Childhood IQ had a direct influence on adult social class and deprivation category in adulthood. Childhood IQ and social class were not associated directly with smoking consumption at midlife. There was a significant pathway between deprivation category and smoking consumption, indicating that the influence of childhood IQ and adult social class on smoking consumption was mediated via deprivation in adulthood. Adult social class had a direct influence on FEV_1. There was a significant pathway between sex and smoking consumption in adulthood. A further significant pathway was found between the number of cigarettes smoked per day and lung function at midlife. The Lagrange multiplier indicated that adding a direct pathway between sex and adult social class, and a further direct pathway between childhood

IQ and FEV_1 would result in a superior model fit. To summarise, the model shows that age 11 IQ did not effect smoking consumption directly but childhood IQ and adult social class had indirect effects on smoking consumption via deprivation category. There is a small direct effect of IQ on lung function at midlife, and another effect that is indirect, via adult social class. Sex influenced smoking consumption and also IQ and social class.

Smoking-related outcomes over 25 years of follow-up

One hundred and ten people had a smoking-related admission, cancer registration, or death in the 25 year follow-up period (84 men, 26 women). 'Smoking-related' events happened to never smokers (n = 8) and past smokers (n = 9) as well as a larger number of current smokers (n = 93). Table 1 shows the hazard ratios of having a smoking-related event, during 25 years of follow-up, for each standard deviation disadvantage in childhood IQ. After controlling for sex, the risk of having a smoking-related event was 21% higher for each standard deviation disadvantage in childhood IQ. Adjustment for social class, deprivation category and smoking separately reduced the risk to 14%, 18% and 16%, respectively, and the hazard ratios were no longer statistically significant. The fully adjusted model controlling for sex, social class, deprivation category and smoking reduced the risk to 10%.

Insert Table 1 about here

Discussion

In both men and women, a lower childhood IQ at age 11 was associated with poorer adult social class, greater deprivation and poorer lung function at midlife, but there was no significant association between childhood IQ and smoking consumption. These correlations may oversimplify the relationships among the measured risk factors and smoking behaviour. The use of SEM allowed us to test hypotheses about how different risk factors associate with each other (Bentler, 1995). We were able to (i) explore whether social and lifestyle factors in adulthood had direct or indirect links with childhood IQ, and smoking consumption and lung function, and (ii) examine the effect of different predictors on smoking and lung function within the same analysis.

The substantial effect of social and lifestyle factors on smoking and health is well known (Carstairs & Morris, 1991; Whitehead, 1992). In structural equation modelling, deprivation category emerged as a predictor of smoking consumption. This finding complements previous studies which showed that people who live in more deprived areas were more likely to smoke and had poorer lung function even after social class had been taken into account (Davey Smith, Hart, Watt, Hole & Hawthorne, 1998). Childhood IQ and adult social class were not directly related to smoking consumption but there was evidence to suggest they may have a small indirect effect on smoking consumption via deprivation in adulthood. Childhood IQ, adult social class and smoking consumption had significant associations with FEV_1 in adulthood. These associations were partly indirectly influenced by sex. The association between childhood IQ and FEV_1 in adulthood remained significant after partialling out the effect of smoking but the effect size was small. This finding contrasts with the results from adults in the 1921 Aberdeen birth cohort (another SMS1932 sample) which found no association between childhood IQ and lung function at the later age of 77 years (Starr, Deary, Lemmon & Whalley, 2000).

Several studies have shown an association between lung function and specific diseases and mortality (Ebi-Kryston, 1988; Hole *et al*., 1996; Marcus *et al*., 1989; Persson *et al*., 1986; Peto *et al*., 1983; Schunemann *et al*., 2000; Strachan, 1991; Tockman *et al*., 1987; Van Den Eeden & Friedman, 1992; Welin *et al*., 1987). One's level of FEV_1 in adulthood is likely to be determined by genetic and biochemical factors, intra-uterine growth, social and lifestyle factors (e.g. diet, exercise), occupational exposure not fully captured by social class, as well as air

pollution, and health behaviours such as smoking. The mechanisms of the association between FEV_1 and morbidity and mortality have not been determined. The effect of poorer lung function on mortality is unlikely to be purely attributable to smoking as the same association was found in non-smokers (Hole *et al*., 1996). Our novel results show that IQ in childhood can explain a small amount of the variance in FEV_1 in adulthood which is not captured by smoking behaviour. Childhood IQ may be an indicator of other unmeasured factors. For example, people with lower IQs may experience higher levels of childhood respiratory illness as a result of damp housing and overcrowding.

We have shown previously that childhood IQ at age 11 was related to survival up to age 76 (Hart *et al*., 2003). Here we tested the hypothesis that the relationship between IQ and mortality involves mediation via smoking behaviour. There was a 21% increased hazard ratio of having a smoking-related illness event associated with a one standard deviation disadvantage in childhood IQ. After controlling for adult social class, deprivation and smoking consumption in adulthood the risk of having a smoking-related event associated with IQ was somewhat attenuated and became non-significant. We have shown previously that the more intelligent smokers in this cohort were more likely to give up smoking by the time they reached midlife (Taylor *et al*., 2003). It may be, therefore, that higher IQ in early life is associated with a tendency to make healthier lifestyle decisions, and responsiveness to health education material.

In this study information about the degree to which each cigarette is smoked down to the butt, the brand and strength of the cigarettes, and whether the cigarettes were filtered, was not available for all of the participants. The data are a poor surrogate for the 'dose' of the exposure and when in life it occurred. These and other smoking behaviours could differ between social groups and by childhood IQ and may influence the health consequences associated with smoking.

It is possible that current smoking was under-reported or concealed by some of the participants in our sample. However, in epidemiological studies, self reported smoking is found to be valid (Vartiainen, Seppala, Lillsunde, & Puska, 2002). Cotinine concentrations were not measured in the present study. Measurement of cotinine concentrations is one possible way to obtain an objective assessment of current smoking consumption (Heinrich, Holscher, Seiwert,

Carty, Merkel, & Schulz, 2004) but high performance liquid chromatography (HPLC)-based cotinine assessment does not provide a measure of the number of cigarettes smoked over the lifetime.

It is important to consider the sampling differences between the two Midspan cohorts when considering the generalisability of the findings. The Collaborative participants were workers whereas the Renfrew/Paisley participants were from the general population living in these towns. However, both samples were based in the same general area (mainly the west of Scotland) and born in the same year. It is likely, therefore, that both samples had similar experiences and lifestyles. As with all longitudinal cohort studies, our data are susceptible to cultural and temporal factors, such as the age of the participants and the area where they grew up. These factors should be acknowledged when interpreting the results. Unlike today, the participants in the present study would not have been aware of the health risks of smoking when many of them started smoking; otherwise, the more intelligent smokers might have chosen not to start. Replication of these findings in other samples will be necessary to evaluate whether the results are cohort-specific.

We have shown that age 11 IQ had a small effect on lung function in adulthood but was not associated directly with smoking consumption in this cohort. Childhood mental ability was associated with smoking-related outcomes during 25 years of follow-up, but this was attenuated once smoking had been controlled for. Future investigations should investigate further the possible pathways which may link childhood IQ to health in later life.

Acknowledgements

The study was funded by the Chief Scientists Office of the Scottish Executive. We thank the Scottish Council for Research in Education for making the data from the Scottish Mental Survey 1932 available to the authors. Victor Hawthorne was responsible for the original Midspan studies and Pauline MacKinnon updates the mortality information. IJD is the Recipient of a Royal Society-Wolfson Research Merit Award and LJW holds a Wellcome Trust Career Development Award.

References

Bentler, P.M. (1995). EQS structural equations program manual. Encino (CA): Multivariate Software INC.

Carstairs, V., & Morris, R. (1991). Deprivation and Health in Scotland. Aberdeen: Aberdeen University Press.

Chassin, L., Presson, C.C., Rose, J.S., & Sherman, J.J. (1996). The natural history of cigarette smoking from adolescence to adulthood: Demographic predictors of continuity and change. *Health Psychology*, *15*, 478-484.

Clark, M.A., Kviz, F.J., Crittenden, K.S., & Warnecke, R.B. (1998). Psychosocial factors and smoking cessation behaviours among smokers who have and have not ever tried to quit. *Health Education and Research*, *13*, 145-153.

Coambs, R.B., & Kozlowski, L.T. (1992). Age interacts with heaviness of smoking in predicting success in cessation of smoking. *American Journal of Epidemiology*, *135*, 240-246.

Davey Smith, G., Hart, C., Hole, D., MacKinnon, P., Gillis, C., Watt, G., Blane, D., & Hawthorne, V. (1998). Education and occupational social class: which is the more important indicator of mortality risk? *Journal of Epidemiology and Community Health*, *52*, 153-160.

Davey Smith, G., Hart, C., Watt, G., Hole, D., & Hawthorne, V. (1998). Individual social class, area-based deprivation, cardiovascular disease risk factors, and mortality: the Renfrew and Paisley study. *Journal of Epidemiology and Community Health*, *52*, 399-405.

Deary, I.J., Whalley, L.J., Lemmon, H., Crawford, J.R., & Starr, J.M. (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.

Doll, R., & Hill, A.B. (1999). Smoking and carcinoma of the lung. Preliminary report. 1950. *Bulletin of the World Health Organisation*, *77*, 84-93.

Doll, R., Peto, R., Wheatley, K., Gray, R., & Sutherland, I. (1994). Mortality in relation to smoking: 40 years' observations on male British doctors. *British Medical Journal, 309*, 901-911.

Droomers, M., Schrijvers, C.T.M., & Mackenbach, J.P. (2002). Why do lower educated people continue smoking? Explanations from the longitudinal GLOBE study. *Health Psychology*, *21*, 263-272.

Ebi-Kryston, K.L. (1988). Respiratory symptoms and pulmonary function as predictors of 10-year mortality from respiratory disease, cardiovascular disease, and all causes in the Whitehall study. *Journal of Clinical Epidemiology*, *41*, 251-260.

Freund, K.M., D'Agostino, R.B., Belanger, A.J., Kannel, W.B., & Stokes, J. (1992). Predictors of smoking in the Framingham study. *American Journal of Epidemiology*, *135*, 957-964.

General Register Office. (1966). *Classification of Occupations 1966*. London: HMSO.

Hanlon, P., Walsh, D., Whyte, B.W., Scott, S.N., Lightbody, P., & Gilhooly, M.L. (1998). Hospital use by an ageing cohort: an investigation into the association between biological, behavioural and social risk markers and subsequent hospital utilization. *Journal of Public Health Medicine*, *20*, 467-476.

Hart, C.L., Deary, I.J., Taylor, M.D., MacKinnon, P.L., Davey Smith, G., & Whalley, L.J., Wilson, V., Hole, D.J., & Starr, J.M. (2003). The Scottish Mental Survey 1932 linked to the Midspan studies: a prospective investigation of childhood intelligence and future health. *Public Health*, *117*, 187-195.

Hart, C.L., Taylor, M.D., Davey Smith, G., Whalley, L.J., Starr, J.M., Hole, D.J., Wilson, V., & Deary, I.J. (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.

Hawthorne, V.M., Watt, G.C., Hart, C.L., Hole, D.J., Davey Smith, G., & Gillis, C.R. (1995). Cardiorespiratory disease in men and women in urban Scotland: baseline characteristics of the Renfrew/Paisley (Midspan) study population. *Scottish Medical Journal*, 40, 102-107.

Hole, D.J., Watt, G.C.M., Davey Smith, G., Hart, C.L., Gillis, C.R., & Hawthorne, V.M. (1996). Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. *British Medical Journal*, *313*, 711-715.

Heinrich, J., Holscher, B., Seiwert, M., Carty, C.L., Merkel, G., & Schulz, C. (2004). Nicotine and cotinine in adults' urine: The German Environmental Survey 1998. *Journal of Exposure Analysis and Environmental Epidemiology* (In Press – Epub ahead of print).

International Agency for Research on Cancer. (IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans Lyons, IARC, 1986 (No 38.).

Iribarren, C., Luepker, R., McGovern, P.G., Arnett, D.K., & Blackburn, H. (1997). Twelve year trends in cardiovascular risk factors in the Minnesota Heart Survey: Are socio-economic differences widening? *Archives of Internal Medicine*, *157*, 873-881.

Levin, M.L., Goldstein, H., & Gerhardt, P.R. (1950). Cancer and tobacco smoking. *Journal of the American Medical Association, 143*, 336-338.

Marcus, E.B., Curb, J.D., MacLean, C.J., Reed, D.M., & Yano, K. (1989). Pulmonary function as a predictor of coronary heart disease. *American Journal of Epidemiology*, *129*, 97-104.

Neisser, U., Boodoo, G., Bouchard, T.J., Boykin, A.W., Brody, N., Ceci, S.J., Halpern, D.F., Loehlin, J.C., Perloff, R., Sternberg, R.J., & Urbina, S. (1996). Intelligence: knowns and unknowns. *American Psychologist*, *51*, 77-101.

Nordstrom, B.L., Kinnunen, T., Utman, C.H., Krall, E.A., Vokonas, P.S., & Garvey, A.J. (2000). Predictors of continued smoking over 25 years of follow-up in the normative aging study. *American Journal of Public Health*, *90*, 404-406.

Osler, M. & Prescott, E. (1998). Psychosocial, behavioural, and health determinants of successful smoking cessation: a longitudinal study of Danish adults. *Tobacco Control*, *7*, 262-267.

Persson, C., Bengtsson, C., Lapidus, L., Rybo, E., Thiringer, G., & Wedel, H. (1986). Peak expiratory flow and risk of cardiovascular disease and death. *American Journal of Epidemiology*, *124*, 942-948.

Peto, R., Darby, S., Deo, H., Silcocks, P., Whitley, E., & Doll, R. (2000). Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *British Medical Journal*, *321*, 323-329.

Peto, R., Speizer, F.E., Cochrane, A.L., Moore, F., Fletcher, C.M., Tinker, C.M., Higgins, I.T., Gray, R.G., Richards, S.M., Gilliland, J., & Norman-Smith, B. (1983). The relevance in adults of air-flow obstruction but not of mucus hypersecretion, to mortality from chronic lung disease. *American Review of Respiratory Disorders*, *128*, 491-500.

Pierce, J.P., Fiore, M.C., Novotny, T.E., Hatziandreu, E.J., & Davis, R.M. (1989). Trends in cigarette smoking in the United States. Educational differences are increasing. *Journal of the American Medical Association*, *261*, 56-60.

Schairer, E., Schöniger, E., Lungenkrebs, & Tabakverbrauch, Z. (1943). *Krebsforsch*, *54*, 261-269.

Schunemann, H.J., Dorn, J., Grant, B.J., Winkelstein, W., & Trevisan, M. (2000). Pulmonary function is a long-term predictor of mortality in the general population. 29-year follow-up of the Buffalo Health Study. *Chest*, *118*, 656-664.

Scottish Council for Research in Education. (1933). *The intelligence of Scottish children: a national survey of an age group*. London: University of London Press.

Starr, J.M., Deary, I.J., Lemmon, H., & Whalley, L.J. (2000). Mental ability age 11 years and health status age 77 years. *Age and Ageing*, *29*, 523-528.

Strachan, D.P. (1991). Ventilatory function as a predictor of fatal stroke. *British Medical Journal*, 302, 84-87.

Tillgren, P., Haglund, B.J., Lundberg, M., & Romelsjö, A. (1996). The sociodemographic pattern of tobacco cessation in the 1980s: results from a panel study of living condition surveys in Sweden. *Journal of Epidemiology and Community Health*, *50*, 625-630.

Tockman, M.S., Anthonisen, N.R., Wright, E.C., & Donithan, M.G. (1987). Airways obstruction and the risk of lung cancer. *Annals of Internal Medicine*, *106*, 512-518.

Townsend, J., Roderick, P., & Cooper, J. (1994). Cigarette smoking by socio-economic group, sex and age: effects of price, income, and health publicity. *British Medical Journal*, *309,* 923-927.

Van den Eeden, S.K., & Friedman, G.D. (1992). Forced expiratory volume (1 second) and lung cancer incidence and mortality. *Epidemiology*, *3*, 253-257.

Vartiainen, E., Seppala, T., Lillsunde, P., & Puska, P. (2002). Validation of self-reported smoking by serum cotinine measure in a community-based study. *Journal of Epidemiology and Community Health*, *56*, 163-164.

Welin, L., Svardsudd, K., Wilhelmsen, L., Larsson, B., & Tibblin, G. (1987). Analysis of risk factors for stroke in a cohort of men born in 1913. *New England Journal of Medicine, 317*, 521-526.

Whalley, L.J., & Deary, I.J. (2001). Longitudinal cohort study of childhood mental ability and survival up to age 76. *British Medical Journal*, *322,*1-5.

Whitehead, M. (1992). *Inequalities in health: the health divide.* London: Penguin.

Wynder, E.L., & Graham, E.A. (1950). Tobacco smoking as a possible etiologic factor in bronchogenic carcinoma. *Journal of the American Medical Association*, *143*, 329-336.

Xu, X., Dockery, D.W., Ware, J.H., Speizer, F.E., & Ferris, B.G. (1992). Effects of cigarette smoking on rate of loss of pulmonary function in adults: a longitudinal assessment. *American Review of Respiratory Disorders*, *146*, 1345-8.

Table 1: Hazard ratios (and 95% confidence intervals) of smoking-related hospital admissions, cancers or smoking related deaths associated with 1 SD decrease in IQ (N= 905; 110 people had a smoking-related event during 25 years of follow-up)

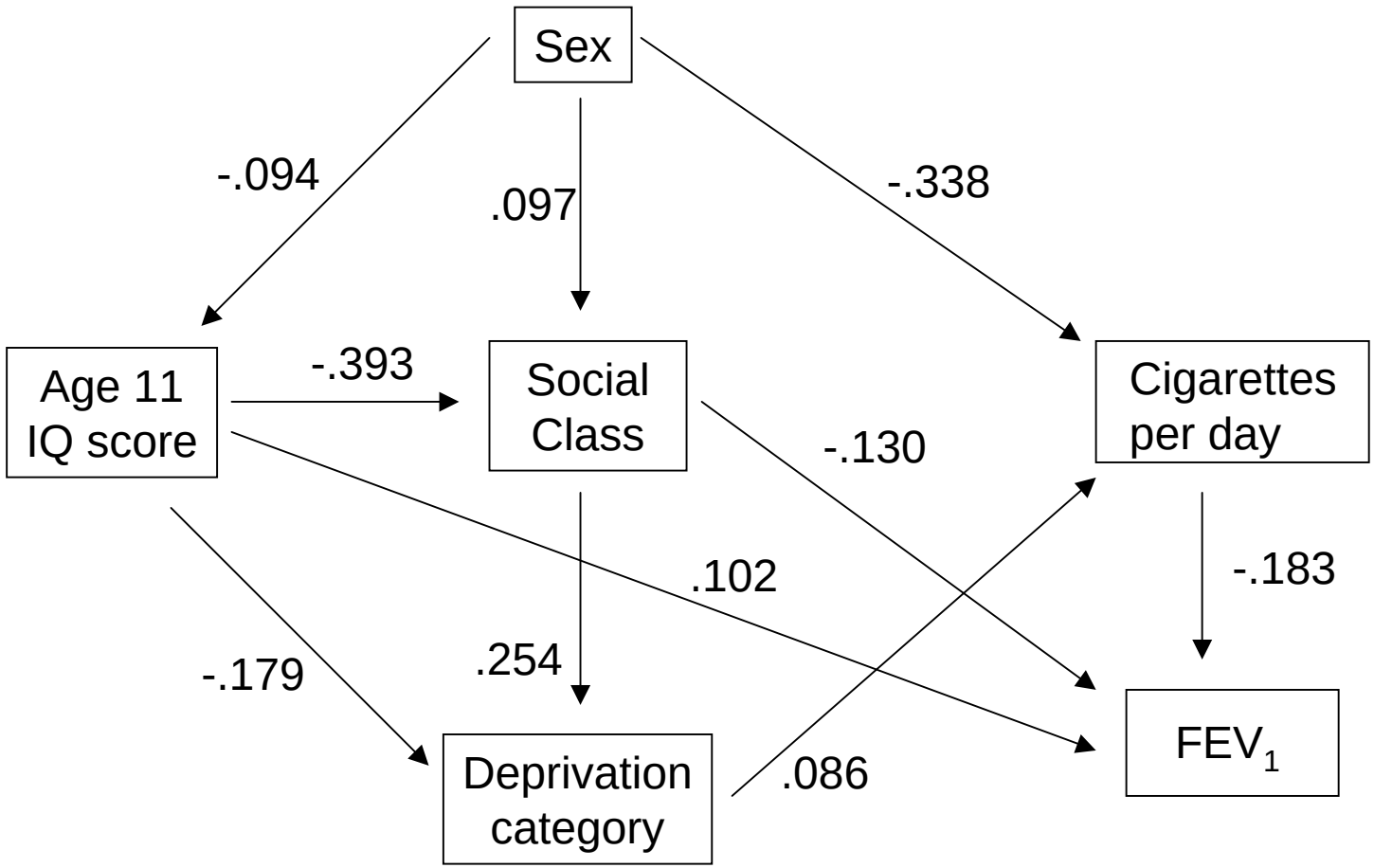
	Hazard ratios associated with 1 SD decrease in IQ
Adjusted for:	
Sex	1.21 (1.01 – 1.45)
Sex and social class	1.14 (0.93 – 1.39)
Sex and deprivation category	1.18 (0.98 – 1.42)
Sex and smoking	1.16 (0.97 – 1.38)
Sex, social class, deprivation and smoking	1.10 (0.90 – 1.34)

Excludes pipe/cigar smokers (n = 12) and people with missing data.

Figure caption

Figure 1

Structural equation model of the associations among childhood IQ, sex (1 = male, 2 = female), social class (lower numbers = higher social class), deprivation (higher numbers = more deprivation), cigarettes per day, and FEV_1 for 905 participants with complete data. Coefficients placed beside arrows may be squared to give the variance shared by adjacent variables.


Sex
-.094
.097
-.338
Age 11
IQ score
-.393
Social
Class
Cigarettes
per day
-.130
.102
-.183
-.179
.254
Deprivation
category
.086
FEV1