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Smoking, childhood IQ, and cognitive function in old age

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ABSTRACT

OBJECTIVES: To examine the association between smoking history and cognitive function in old age, and whether it remains after controlling for childhood cognitive ability (IQ) and adult socioeconomic status (SES).

METHODS: In the Lothian Birth Cohort 1936 Study, 1080 men and women, who previously participated in a nationwide IQ-type test in childhood, were followed up at age 70. The associations between smoking history and age 70 IQ, general (*g*) cognitive ability, processing speed, memory, and verbal ability were assessed.

RESULTS: Lower childhood IQ was associated with a higher risk of becoming a smoker and continuing to smoke in late life, and with reduced lung function (FEV1) in late life. Current smokers scored significantly lower than ex-smokers and never smokers on tests of age 70 IQ, general cognitive ability, and processing speed, but not memory or verbal ability. After controlling for childhood IQ and SES, current smoking at age 70 (but not pack years of smoking) was associated with impairments in general cognitive ability and processing speed.

CONCLUSION: Smoking in old age makes a small, independent contribution to cognitive performance in old age.

Key words: aging, cognitive function, childhood IQ, socioeconomic status, smoking

INTRODUCTION

Many people continue to smoke into old age despite public health messages warning of the long-term effects on health. Those smokers who survive to old age have an increased risk of significant morbidity [1]. Moreover, a growing body of evidence suggests that smokers perform more poorly in late life on tests of global cognitive function [2,3] and across several cognitive domains, including memory [4-6], information processing speed [4,6], executive function [7] psychomotor speed [8,9] and cognitive flexibility [8], and especially on cognitively demanding tasks [10]. Numerous longitudinal studies have suggested a causal relationship between chronic smoking into late life and an increased risk of cognitive decline and dementia [5,11-14]. Some of these studies propose a dose-response effect of smoking, whereby the degree of cognitive decline experienced increases with the quantity of cigarettes smoked [12,13]. Typically, ex-smokers cognitively outperform those who continue to smoke into old age [4,15].

Several mechanisms have been proposed to explain this relationship. The most widely accepted suggest that cognitive impairment may be due to adverse effects of chronic smoking on cardiac, vascular [11,16] and pulmonary function [9,17,18]. However, recent findings point to alternative, non-physiological, underlying mechanisms. Firstly, population-based studies have identified an association between prior intelligence and smoking behaviour. People with lower IQ-type scores are more likely to become smokers [19-21]. Those who quit are more likely to have had a higher childhood intelligence than those who continue to smoke [22]. Secondly, there is a socioeconomic gradient in smoking prevalence [20,23]. Socioeconomic factors may also explain some of the IQ-smoking relationship [19,24]. A link between prior low IQ and/or low SES and smoking habits may partly explain, by confounding, why long-term smokers perform

more poorly on tests of cognition later in life than never-smokers, and why ex-smokers perform better than those who continue to smoke.

Many studies have been unable to rule out prior intelligence (before initiation of smoking) as a potential confounder as this is rarely available when examining cognitive function in older people. Those studies with this data have found that smoking was associated with a small relative decline in cognition after controlling for childhood IQ. However, in one study, the results were based on a single cognitive outcome (verbal reasoning at age 80) and did not control for SES [25]. In the other, of the five cognitive outcomes tested, only psychomotor speed was found to be adversely affected by smoking history (after adjusting for childhood IQ) in a sample of 64–year olds [9]. A 2-year follow-up of the same sample identified a small additional deficit in memory with a positive smoking history [6].

The present study addresses the gaps in the current knowledge of the smoking-cognition relationship in a large sample of older individuals (aged 70), for whom childhood intelligence, later life intelligence and extensive other cognitive data, adult SES, health measures (including lung function), and smoking history are all available. We examine: (1) the association between early life intelligence and smoking history; (2) the association between smoking and cognitive function in old age across a wide range of cognitive domains; and (3) the roles of prior intelligence and SES (independently and in combination) in order to evaluate more fully the underlying mechanisms in the smoking-cognition relationship which are as yet, unclear.

METHODS

Participants

We examined individuals in the Lothian Birth Cohort 1936 Study (LBC1936) which comprises 1091 men and women. Almost all participants were resident in Edinburgh and the surrounding Lothian region. Early life (mean age 11 years) intelligence test data are available for this sample, because they are surviving participants of the Scottish Mental Survey of 1947 (SMS1947) [26]. Assessment in later life took place between 2004 and 2007 when participants were about 70 years old. Full recruitment and testing procedures are reported in an open-access protocol paper [27]. In brief, the assessment involved an interview, extensive cognitive testing, a physical examination, and questionnaires. Smoking data were available for all 1091 participants in the LBC1936. Eleven participants scored less than 24 on the MMSE; this cut-off is often used to indicate possible dementia. These participants were excluded from statistical analyses. The sample comprised 1080 individuals (539 men). Ethics permission for the Lothian Birth Cohort 1936 (LBC1936) study protocol was obtained from the Multi-Centre Research Ethics Committee for Scotland and from Lothian Research Ethics Committee for Scotland. The research was carried out in compliance with the Helsinki Declaration. All participants gave their written, informed consent.

Measurements

Smoking history. Data on smoking history were collected during the interview, including: current smoking status (never, ex or current smokers) ; the age at which the participants started smoking; age at quitting (for ex-smokers) and; the average number of cigarettes smoked per day. Pack years were calculated as the average number of cigarettes per day times years as a smoker, divided by 20. Pack years expresses lifelong exposure to cigarettes.

Moray House Test (age 11 and 70 MHT IQ). On June 4th 1947, about 95% of schoolchildren born in 1936 and attending Scottish schools (70,805), took a version of the Moray House Test (MHT) No.12 (Scottish Council for Research in Education (SCRE) [28-29]. The MHT is a group-administered test of general intelligence. This test was concurrently validated against the Terman-Merill revision of the Binet scales (SCRE) [29]. SCRE recorded and archived these scores and made them available to the LBC1936 Study. Participants re-sat the MHT at a mean age of 70. MHT scores for the LBC1936 were corrected for age in days at time of testing and converted into IQ-type scores for the sample (M = 100, SD = 15).

Other cognitive testing. See Deary et al. (2007) for full details [27]. Composite cognitive function scores were used to represent three main cognitive domains. The extraction of these factors, using principal components analysis has been described in detail elsewhere [30,31]. A *g* factor score, representing general cognitive ability, was derived from scores on six Wechsler Adult Intelligence Scale-III^{UK} (WAIS-III) subtests [32], namely: Letter-Number Sequencing; Matrix Reasoning; Block Design; Digit Symbol; Digit Span Backwards; Symbol Search. A processing speed factor was derived from scores on a set of mental speed measures, namely: Symbol Search (WAIS-III); Digit Symbol (WAIS-III); Simple and Choice Reaction Time mean [33]; Inspection Time (a computer-based test of elementary visual processing speed) [34]. A memory factor was derived from scores on a set of memory measures from Wechsler Memory Scale-III^{UK} (WMS-III) [35], namely: Logical Memory I immediate and II delayed recall; Spatial Span Forwards and Spatial Span Backwards; Verbal Paired Associates I immediate recall and II delayed recall; and two WAIS-III subtests (Letter-Number Sequencing, Digit Span Backwards). Verbal ability was assessed using the National Adult Reading Test (NART) [36] and the

Wechsler Test of Adult Reading (WTAR) [37]. These tests are widely used to estimate prior cognitive ability and each requires the pronunciation of a list of 50 irregular words. The Mini-Mental State Examination (MMSE) is a standardised brief screening measure for cognitive pathology [38]. Scores range from 0-30 with a score of less than 24 often used to indicate possible dementia.

Covariates

Sociodemographic. Participants provided general demographic information including marital status and years of full-time education. Adult occupational social class was derived from each participant's highest reported occupation and classified into one of six categories ranging from I (professional occupations) to V (unskilled occupations), with III divided into IIIN (non-manual) and IIIM (manual) [39]. For data analysis, classes IV and V were combined due to the small number of participants in class V.

Health behaviors. Alcohol use was recorded using the Scottish Collaborative Group Food Frequency Questionnaire (FFQ) version 7.0 [40] and expressed as units of alcohol consumed in a typical week. Body mass index (BMI) was calculated as weight (kg)/height (m^2) from measurements recorded during the physical examination at time of testing. Level of physical activity was obtained from a self-report questionnaire. This measure was coded as follows: 1 – "household chores"; 2 – "walking etc. 1-2 times a week"; 3 – "walking etc. several times a week"; 4 – "exercise 1-2 times a week"; 5 – "exercise several times a week"; 6 – "keep-fit/heavy exercise/sport several times a week".

Health measures. Presence or history of hypertension, stroke, cardiovascular disease (CVD) and diabetes were recorded. Serum cholesterol (mmol/L) was measured as part of a blood

analysis profile. A measure of lung function (forced expiratory volume in 1 second, FEV1) was obtained using a spirometer. This measure was performed three times and the highest measure was used in the present study.

Statistical Analyses

Analyses were performed using IBM SPSS Version 19 (IBM, NY, USA). Lung function measures were converted into percentages of the value predicted from a linear regression of lung function on height for each sex, based on never-smokers. We identified 16 participants' 'pack years' scores as being outliers, which were capped at 100 prior to analysis. We used analysis of variance and chi-square tests to examine whether demographic and health variables differed between smoking categories. General linear models (GLMs) were used to investigate the associations between cognition at age 70 and smoking status (categorical variable), and cognition at age 70 and pack years (continuous variable), where never smokers were given a score of 0. Cognitive outcomes (dependent variables) were age 70 IQ, *g* factor, processing speed factor, memory factor, NART, and WTAR. Separate models were fitted adjusting for potential confounding variables: age and sex only (model 1); occupational social class (model 2); childhood IQ (model 3); occupational social class and childhood IQ (model 4); lung function (FEV1, model 5); health behaviors (alcohol use, BMI, physical activity, model 6); and health measures (diabetes, stroke, CVD, model 7). The final, 8th, model included all the covariates mentioned above. All models contained age and sex, except for models containing the outcome age 70 IQ, which is already adjusted for age. We present relevant estimates of effect size, reported here as partial eta-square (η_p^2), and *p* values (the 0.05 level of significance was used for all data analyses). In examining any attenuation by covariates, of the effect of smoking on

cognition at age 70, we draw the reader's attention to the change in effect size, not merely to any change in the significance level.

RESULTS

Descriptive results - participants

Participants' characteristics are summarized in **Table 1**. At time of testing, 46% reported that they had never smoked, 42% were ex-smokers, and 12% were current smokers. Compared to never smokers, smokers were more likely to be male, unmarried (single, widowed, or divorced), belong to a 'less professional' social class, have fewer years of full-time education, a lower MMSE score, a higher weekly alcohol intake, and a lower BMI. Lung function (FEV1) was lowest in current smokers. Current smokers were more likely to have a history of stroke, CVD or diabetes. Average age at smoking initiation was 18.3 years (*SD* = 5.43) for current smokers, and 18.5 (*SD* = 5.36) for ex-smokers. Average age at quitting, for ex-smokers, was 46.76 (*SD* = 13.24).

Current smoking status and cognitive function

Table 2 presents IQ-type scores (at age 11 and 70) and other cognitive function scores by smoking status. These unadjusted results showed a significant association between smoking status and IQ at age 11 ($p = .036$) and age 70 ($p < .001$). Age 11 IQ scores were significantly lower in current smokers compared to never smokers (by 3.2 points), with an intermediate score in ex-smokers. At age 70, current smokers had a 5.5 point IQ disadvantage compared to never-smokers. Smokers also performed significantly worse than never smokers on tests of general cognitive ability (*g* factor) ($p < .001$) and processing speed ($p < .001$), but not memory or verbal

ability (NART and WTAR). **Table 3** shows the results of GLMs used to examine the association between smoking status and cognitive outcomes at age 70. In the age- and sex-adjusted only model (model 1), smoking status was associated with significantly lower scores on tests of age 70 IQ ($\eta_p^2 = .012$), *g* factor ($\eta_p^2 = .016$), and processing speed ($\eta_p^2 = .015$). After adjusting for social class and age 11 IQ independently and in combination (models 2, 3 and 4), the association between smoking and age 70 IQ ceased to be significant. The associations between smoking status and general cognitive ability and processing speed remained significant after controlling for both age 11 IQ and social class (independently and in combination), although the effect sizes were slightly attenuated (see model 4: *g* factor $\eta_p^2 = .007$; processing speed $\eta_p^2 = .006$) . These associations persisted (with the exception of model 5 for processing speed, which narrowly missed significance after controlling for FEV1) until full adjustment for all covariates (model 8). Further GLMs (data available on request) confirmed that these results were driven by significant differences between current smokers and non-current smokers, rather than between ever- and never-smokers.

Lifetime smoking (pack years) and cognitive function

Pack years of smoking was negatively correlated with age 11 IQ (-.12, $p < .001$). A separate set of GLMs were used to examine the association between pack years and cognitive outcomes at age 70, and the effects of potential confounders on these associations (see **Table 4**). In the age- and sex-adjusted only model (model 1), increasing pack years was associated with significantly lower scores on tests of age 70 IQ ($\eta_p^2 = .017$), *g* factor ($\eta_p^2 = .018$), processing speed ($\eta_p^2 = .017$), and verbal ability (NART $\eta_p^2 = .008$; WTAR $\eta_p^2 = .007$). After adjusting for social class and age 11 IQ separately (models 2 and 3), the significant associations between pack

years and age 70 IQ (model 2; $\eta_p^2 = .006$, model 3; $\eta_p^2 = .004$) *g* factor (model 2; $\eta_p^2 = .005$, model 3; $\eta_p^2 = .007$) and processing speed (model 2; $\eta_p^2 = .004$, model 3; $\eta_p^2 = .007$) remained, but the effect sizes were markedly attenuated. After adjustment for social class and age 11 IQ in combination (model 4), there were no significant associations between lifetime smoking and cognitive abilities. Controlling for FEV1 (model 5) also removed significant associations between smoking and cognitive abilities, with the exception of age 70 IQ ($p = .030$). It should be noted, however, that FEV1 correlated significantly with age 11 IQ ($r = .159$, $p < .001$). Adjustment for health behaviors (model 6) or health measures (model 7) caused no attenuation of the effect of smoking on cognitive functions that was found in the sex and age-adjusted model (model 1). With full adjustment (model 8), there were no significant effects of lifetime smoking on cognitive abilities.

We also examined the effect of pack years in current smokers only and found no dose-response effect of smoking for any of the cognitive outcomes tested at age 70. The correlation between pack years of smoking in current smokers and age 70 IQ is $r = .038$ ($p = .668$) and between pack years of smoking and general (g) cognitive ability is $r = .093$ ($p = .296$). We also ranked smokers into quintiles of pack years; as before, there were no significant associations between pack years and any of the cognitive outcomes. On examination of the means plots, there was no trend to suggest that such a relationship exists.

DISCUSSION

We examined a sample of individuals, aged about 70, for whom childhood intelligence scores are available. We replicated previous findings that lower IQ at age 11 was associated with a higher risk of becoming a smoker and continuing to smoke into old age, and with the amount of

cigarettes smoked over the lifespan. We found an adverse effect of current smoking on general cognitive ability (*g*) and information processing speed—but not memory or verbal ability—even after controlling for childhood IQ and SES. We found an effect of pack years of smoking on cognitive function of those who had smoked more over their lifetime, which was non-significant after controlling for their (lower) IQ scores and SES. In this sample, current smoking makes a small, negative contribution to cognitive ability level in old age. However, a novel finding was that the observed associations between cumulative smoking (pack years) and poorer cognitive function scores in old age were accounted for, largely, by variations in prior cognitive ability and SES, not independently, but in combination.

Childhood IQ and adult smoking behaviour

The first objective of the study was to examine the relationship between childhood IQ and smoking behavior in adulthood. Our results are consistent with previous studies showing that IQ predicts onset and cessation of smoking. In a cohort born in 1921, Taylor et al. (2003) reported that age 11 IQ predicted cessation, but not uptake of smoking [22], perhaps reflecting social attitudes to tobacco over a particular historical period. Batty et al. (2007) found that a higher IQ at age 10 was associated with a reduced prevalence of current smoking and an increased likelihood of having given up smoking by midlife [19]. The conclusion often drawn from the body of literature supporting a link between IQ and smoking behavior, is that IQ influences differential valuations of the health consequences of smoking [41] as well as access to smoking cessation programmes, via social background [42].

Interestingly, we identified a link between lower childhood IQ and poorer adult lung function, consistent with the work of Richards et al. (2005) [17] and Deary et al. (2006) [43].

They reported a link between childhood intelligence and FEV1 in later life, even after controlling for lifetime smoking. They proposed that respiration and mental function may be subject to the same physiological processes and are a function of what has been termed general bodily system integrity [44].

Smoking and cognitive function at age 70

Our second objective was to examine the association between smoking and cognitive function across a range of cognitive domains. General cognitive ability (*g*) and processing speed were lower in current smokers at age 70, than never- and ex-smokers. These results are in keeping with other studies reporting cognitive impairment in old age with long-term smoking [11]. The relationship between current smoking and poorer cognitive function remained after controlling for markers of ill-health, health behaviors and lung function (FEV1). However, despite having made an especially thorough assessment of memory, we found no effect of smoking on memory function, in contrast to some other studies [4,6,12,14].

Past smoking was not associated with significantly poorer performance than never smokers in any cognitive domain. Deary et al. (2003) found that current smokers, but not ex-smokers, had lower IQ scores at age 80 (after adjusting for childhood IQ) when compared with lifelong never-smokers [25]. This suggests that cognitive detriments, albeit small, may only occur after prolonged exposure to cigarettes over the course of a lifetime. However, a 24-month smoking cessation trial using an old age sample (>68 years), showed that unsuccessful quitters over this 2-year period scored significantly worse on cognitive tests than successful quitters and non-smokers [15]. Therefore, quitting smoking, at any stage in adulthood, may be beneficial to cognitive health in later years. Given that pack years of smoking ceased to be associated with

cognitive performance in the present study, after controlling for childhood IQ and SES, it may be that smoking in old age, irrespective of previous smoking history, affects cognitive health at a time when the body is most vulnerable to its adverse, systemic, and physiological effects.

Childhood IQ and SES as confounders in the smoking-cognitive function relationship

Our final objective was to investigate the roles of childhood IQ and SES in the smoking-cognition relationship. There is a high level of stability in cognitive function across most of the lifespan; childhood IQ accounts for about half the variance observed in later life [45]. In this sample, the raw correlation (r) between age 11 and age 70 IQ is .69, $p < .001$. Controlling for age 11 IQ and SES attenuated the relationship between smoking status and cognitive function and reduces to non-significance the association between lifetime smoking and cognitive function. A contribution of SES was predicted given that 1) it is a correlate of IQ, and 2) smoking was less common among participants from higher (more professional) social classes, a finding documented by other studies in the area [19,23]. Controlling for sociodemographic variables was found to completely attenuate the observed association between lung function and cognitive function in a large sample of men and women in early old age [46]. It has been proposed that lower intelligence among lower socioeconomic groups leads to a poorer understanding of the negative health consequences of health-risk behaviors such as smoking [47]. The inability to give up smoking may also be related to poorer SES as a result of lower job attainment and financial strains [48].

Strengths and limitations

The major strength of this study was the availability of IQ data at two distant time points (age 11 and age 70) derived from scores on a validated test of general intelligence (the MHT). We were able to examine the contribution of smoking to relative cognitive change on this measure from childhood to late adulthood. There was a further advantage of examining childhood cognitive ability before the majority (98.5%) had begun smoking. We studied a cohort who share the same year of birth. The narrow age cohort is an important design advantage given that smoking patterns change over time, and because chronological age itself makes a large contribution to cognitive functions and health in old age [49]. We had comprehensive cognitive data with multiple tests of important cognitive domains at age 70. We had data on quantity and duration of smoking in addition to current smoking status. The LBC1936 sample has data on a wide variety of sociodemographic and health factors. This made it possible to adjust for a range of potential confounders.

The study has some limitations. Although the use of a narrow age group is advantageous, we note that this limits the generalizability of results. Second, self-reported smoking history is open to recall bias, especially among elderly participants. Under-reporting of smoking history may be a potential confounder of the data. It should be noted that, if the direction of this effect was such that people with greater cognitive declines were more likely to under-report smoking history, then the effect sizes in this report would be under-estimates. However, self-reported smoking has high agreement with serum cotinine measures [50]. Third, the LBC1936 are a self-selecting, volunteer sample and, as such, and are typically more cognitively able and healthier than the general population. The LBC1936 achieved higher mean age 11 cognitive scores (m = 49.0, sd = 11.8) than both the nationwide and Edinburgh SMS1947 average (m = 36.7, sd = 16.1, m = 40.3, sd = 15.5, respectively) and this may have restricted the range of cognitive outcome

scores in the study. That said, the range of age 11 cognitive scores in the current sample was still large (m = 49.1, sd = 11.7). The most likely result of this is some small underestimation of effects sizes of the associations reported here. Smoking is associated with higher morbidity and mortality rates. Of the smokers who survive to old age, those with significant health problems are less likely to participate in such a study, possibly leading to an under-representation of smokers in the current sample.

In conclusion, our results suggest that smoking in old age may contribute to age-related cognitive impairments in general cognitive ability and speed of information processing. Some of the apparent cognitive impairment due to lifetime smoking can be accounted for by a lower childhood cognitive ability and socioeconomic status Cessation of smoking in adulthood appears to 'buffer' the cognitive aging experienced by those who continue to smoke. In terms of public health implications, the present findings suggest that there are cognitive benefits to quitting smoking, even for older adults who have been smoking for many years; intervention programmes should target smokers at all ages.

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Author Contributions

JC performed the analyses and led the writing of the article. JC and AG were involved in data collection. AG, JS and ID contributed to study design, interpretation of analysis and drafting the manuscript.

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Tables

Table 1

Characteristics of the study population grouped by smoking status (mean values and % prevalence)

	Total		Never smokers		Ex-smokers		Current smokers		
	n = 1080		*n* = 495		*n* = 455		*n* = 130		*p*
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	
Pack years[a], mean (sd)	31.38	(27.69)	0		27.85	(28.32)	43.84	(21.17)	
Sociodemographic measures									
Age, y, mean (sd)	69.53	(0.83)	69.51	(0.84)	69.55	(0.85)	69.54	(0.74)	
Men %	49.9		40.4		58.4		52.9		**<.001**
Married %	71.2		71.9		73.6		60.9		**.001**
Social class %									**<.001**
I	17.5		18.6		18.8		9.4		
II	36.9		38.3		38.6		26.8		
IIIN	22.8		26		16.8		31.9		
IIIM	17.0		13.5		18.8		23.2		
IV+ V	3.8		2.5		4.2		6.5		

Education, yrs, mean (sd)	10.75	(1.13)	10.87	(1.16)	10.69	(1.13)	10.49	(0.95)	**.001**
MMSE, mean (sd)	28.86	(1.28)	28.95	(1.26)	28.84	(1.31)	28.62	(1.28)	**.029**
Health behaviors									
Alcohol use, U/wk, mean (sd)	10.56	(14.21)	8.24	(12.31)	12.63	(14.40)	12.17	(18.46)	**<.001**
Body Mass Index, mean (sd)	27.81	(4.43)	27.61	(4.24)	28.51	(4.39)	26.10	(4.73)	**<.001**
Physical activity (level)	7.63	(8.11)	7.77	(7.75)	7.98	(8.39)	5.61	(8.28)	**.027**
Health measures									
Diabetes (%)	8.1		5.7		10.7		8		**.026**
Stroke (%)	4.9		3.8		4.7		9.4		**.014**
Cardiovascular disease (%)	24.6		20.7		28.6		24.6		**.046**
Cholesterol mmol/l, mean (sd)	5.46	(1.16)	5.57	(1.17)	5.34	(1.15)	5.47	(1.15)	**.011**
Lung function measure									
FEV1	2.36	(0.69)	2.44	(0.65)	2.38	(0.69)	1.96	(0.70)	**<.001**
%FEV1[b]	94.36		100.06		92.64		78.58		

Note.

MMSE - Mini-Mental State Examination; FEV1 – forced expiratory volume in 1 second

p values are from t-tests, ANOVA and chi-square tests as appropriate

[a]Pack years was calculated by dividing the average number of cigarettes smoked daily by 20 and then multiplying that by the number of years smoked

[b]% values refer to percentage of values predicted by sex and height in never-smokers

Table 2

Cognitive outcomes by smoking status, with reported mean and p-values (ANOVA)

	Never smokers n = 495		Ex-smokers n = 455		Current smokers n = 130		
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*p*
Age 11 IQ[a]	101.4	(14.9)	99.3	(14.8)	98.2	(14.7)	**.036**
Age 70 IQ[a]	101.5	(13.4)	100.5	(14.7)	96.0	(16.0)	**<.001**
g factor	0.11	(0.97)	0.02	(0.99)	-0.32	(0.93)	**<.001**
Processing speed	0.10	(0.97)	0.03	(0.98)	-0.34	(0.94)	**<.001**
Memory	0.04	(0.97)	0.04	(0.97)	-0.18	(1.04)	.063
NART	34.9	(8.0)	34.6	(8.1)	33.2	(8.2)	.120
WTAR	41.4	(6.9)	41.2	(7.2)	40.0	(6.9)	.136

Note.

[a]Age 11 IQ and age 70 IQ are derived from scores on the Moray House Test and converted to standard IQ type scores (*M* = 100, *SD* = 15)

Table 3

General linear models of associations between smoking status and cognitive outcomes at age 70 (p values and associated partial eta squared values)

	*Age 70 IQ		*g* factor		Processing speed		Memory		NART		WTAR	
	n = 1069		*n* = 1062		*n* = 1028		*n* = 1037		*n* = 1078		*n* = 1078	
Models	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2
1. A+S	**<.001**	.012	**<.001**	.016	**.009**	.015	.202	.002	.120	.002	.125	.002
2. A+S+SC	**.029**	.005	**.004**	.008	**.003**	.009	.852	.000	.987	.000	.973	.000
3. A+S+IQ11	.057	.004	**.002**	.010	**.006**	.008	.964	.000	.488	.000	.474	.001
4. A+S+SC+IQ11	.205	.002	**.011**	.007	**.019**	.006	.763	.000	.179	.002	.198	.002
5. A+S+FEV1	.068	.003	**.041**	.004	.056	.004	.852	.000	.943	.000	.597	.000
6. A+S+HB	**<.001**	.015	**<.001**	.017	**<.001**	.015	.133	.002	**.043**	.004	.062	.004
7. A+S+HM	**<.001**	.011	**<.001**	.014	**<.001**	.012	.208	.002	.133	.002	.148	.002
8. ALL	.550	.000	.329	.001	.379	.001	.626	.000	.285	.001	.329	.001

Note.

pack years of smoking were capped at 100 (n = 16 outliers were recoded as 100)

A – age; S – sex; SC – social class; IQ11 – age 11 IQ; FEV1 – forced expiratory volume in 1 second; HB – health behaviors (alcohol units/wk, BMI, physical activity level); HM – health measures (diabetes, stroke, CVD); ALL – all covariates

*Age 70 IQ is already age-adjusted (age not included in the models for this outcome variable)

p values in bold represent significant negative associations between smoking score and cognitive outcome

Table 4

General linear models of associations between pack years of smoking (lifetime smoking) and cognitive outcomes at age 70 (p values and associated partial eta squared values)

	*Age 70 IQ		*g* factor		Processing speed		Memory		NART		WTAR	
	n = 1069		*n* = 1062		*n* = 1028		*n* = 1037		*n* = 1078		*n* = 1078	
Models	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2
1. A+S	**<.001**	.017	**<.001**	.018	**<.001**	.017	.104	.003	**.004**	.008	**.006**	.007
2. A+S+SC	**.015**	.006	**.022**	.005	**.038**	.004	.931	.000	.439	.001	.450	.001
3. A+S+IQ11	**.035**	.004	**.007**	.007	**.008**	.007	.765	.000	.829	.000	.829	.000
4. A+S+SC+IQ11	.163	.002	.122	.002	.214	.002	.315	.001	.554	.000	.628	.000
5. A+S+FEV1	**.030**	.004	.067	.003	.091	.003	.712	.000	.402	.001	.495	.000
6. A+S+HB	**<.001**	.013	**.002**	.010	**.003**	.010	.317	.001	**.**084	.003	.138	.002
7. A+S+HM	**<.001**	.015	**<.001**	.014	**<.001**	.012	.113	.002	**.006**	.007	**.009**	.006
8. ALL	.231	.002	.895	.000	.713	.000	.536	.000	.481	.000	.602	.000

Note.

pack years of smoking were capped at 100 (n = 16 outliers were recoded as 100)

A – age; S – sex; SC – social class; IQ11 – age 11 IQ; FEV1 – forced expiratory volume in 1 second; HB – health behaviors (alcohol units/wk, BMI, physical activity level); HM – health measures (diabetes, stroke, CVD); ALL – all covariates

*Age 70 IQ is already age-adjusted (age not included in the models for this outcome variable)

p values in bold represent significant negative associations between smoking score and cognitive outcome