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245 Smoking and blood pressure in 15 year olds in Dunedin, New Zealand //

Ian M St George, Sheila Williams,
Warren R Stanton, Phil A Silva

exercise. After adjustment for height, weight, and sex the difference in blood pressure between the non-smoking group and the group who smoked daily was 4.3 (95% confidence interval 1.8 to 6.8) mm Hg for resting systolic blood pressure and 4.8 (2.1 to 7.5) mm Hg for systolic blood pressure before exercise.

The differences among systolic blood pressures during exercise were not significant, but there were differences for blood pressure after recovery: these were smaller but remained significant after adjustment, suggesting differences in recovery rate among the groups.

The differences in diastolic blood pressure among the groups were not significant. The differences for the blood pressures at age 11 among the 15 year olds who smoked were not significant.

Comment

Explanations suggested for lower interval blood pressure in adult smokers have included lower weight, differences in alcohol consumption, relief of stress by smoking, or a lower "sympathetic tone." Some researchers have even suggested that smokers with hypertension die younger than non-smokers, leaving a population of smokers with lower than average blood pressures.

Our results were similar in direction and magnitude to those found in adult smokers and suggest an effect at least on systolic blood pressure, evident even in young occasional smokers. This effect was not apparent during exercise, suggesting it can be overcome by increased catecholamine release. That the differences had not been evident four years earlier suggests that they were caused by smoking.

Selective mortality does not apply at age 15 and the effect of body size has been adjusted for in the present study. A reduction in sympathetic activity evident in the intervals between smoking seems the only tenable explanation.

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Otago Medical School, PO Box 913, Dunedin, New Zealand
Ian M St George, FRNZCGP, senior lecturer, general practice department
Sheila Williams, BSC, biostatistician, preventive and social medicine department
Warren R Stanton, PHD, research fellow, Dunedin multidisciplinary health and development research unit
Phil A Silva, PHD, director, Dunedin multidisciplinary health and development research unit

Correspondence to: Dr St George.

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Among the immediate effects of inhaling cigarette smoke is a rise in heart rate and blood pressure as a result of the action of nicotine at a number of sites effecting neurocardiovascular control. Many studies—for example, that by Simpson *et al*¹—have found lower average systolic and diastolic blood pressures in adults who smoked than in non-smokers.

Methods and results

The sample and techniques of measurement used in this study have been detailed elsewhere.² Children born between 1 April 1972 and 31 March 1973 in Dunedin were seen at intervals of two years. Sample selection, attrition, and changes in characteristics with time have been described by Silva.³

Measures of smoking, cardiovascular fitness, exercise tolerance, and body size were available for 839 children at age 15.

Blood pressures were obtained at rest and during a standard six minute submaximal cycle ergometer test: before exercise, while seated on the cycle at rest; immediately after the sixth minute of exercise; and after five minutes' recovery.

Smoking histories were obtained at ages 11 and 15.⁴ Blood pressures at age 15 were compared for five groups, defined in terms of their smoking history at age 15: those who had never smoked, had smoked infrequently but not in the past two years, had smoked infrequently in the past two years, had smoked occasionally in the past four weeks, and smoked daily. Analyses of variance and covariance were used.

The table shows means for various systolic blood pressure measures. There was a significant difference among the smoking groups for both unadjusted and adjusted systolic blood pressure at rest and before

Means and adjusted means for systolic blood pressure (mm Hg) measured at age 15 in terms of smoking history at age 15

Systolic blood pressure	Smokers					Error root mean square	p Value
	Never smokers (n=167)	Not in past 2 years (n=210)	Infrequently in past 2 years (n=241)	Occasionally in past 4 weeks (n=110)	Daily (n=103)		
Resting	127.1	125.7	124.2	124.2	122.3	10.87	0.004
Adjusted for sex, height, and weight	124.9	123.6	122.6	123.3	120.6	10.05	0.024
Before exercise	114.7	112.5	111.0	111.0	109.3	11.71	0.002
Adjusted for sex, height, and weight	114.2	112.3	111.2	111.8	109.4	10.87	0.008
During exercise	158.9	157.6	156.7	156.1	155.0	14.93	0.261
Adjusted for systolic blood pressure before exercise, sex, height, and weight	157.4	157.0	157.1	156.8	156.6	13.78	0.990
Recovery	112.2	109.7	108.7	106.5	108.0	10.29	0.000
Adjusted for pre-exercise systolic, sex, height and weight	110.6	109.3	109.1	107.2	109.4	7.64	0.014

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Serum cholesterol concentration and risk of primary brain tumours

Paul Knekt, Antti Reunanen, Lyly Teppo

Research Institute for Social Security, Social Insurance Institution, 00380 Helsinki, Finland)

Paul Knekt, PHD, research officer

Antti Reunanen, MD, research officer

Finnish Cancer Registry, 00170 Helsinki, Finland
Lyly Teppo, MD, director

Correspondence to:
Dr Knekt.

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Several studies have reported raised serum cholesterol concentrations in patients with brain tumours.¹⁻⁴ On the other hand, a large number of studies have reported an inverse association between serum cholesterol concentration and risk of cancer at various other sites.⁵ The only cohort study of the relation between cholesterol and brain tumours examined relatively few patients.⁴ We examined the possibility of an association in a larger cohort.

Subjects, methods, and results

During 1966-72, 48 325 people (26 001 men and 22 324 women) aged 20-75 years and apparently free of cancer participated in the Finnish Social Insurance Institution's mobile clinic health survey. Serum cholesterol concentration was determined at the initial visit and during follow up until 31 December 1984. In all, 72 people were diagnosed as having a brain tumour according to the Finnish Cancer Registry.⁶ Of these 44 had malignant tumours and 28 non-malignant tumours.

The mean age of those with brain tumours was 45.1 years, that of the rest of the subjects being 41.7 years. The age adjusted mean cholesterol concentration in those with malignant brain tumours was 6.54 mmol/l in men and 6.70 mmol/l in women. The corresponding values in those without brain tumours were both 0.03 mmol/l lower (differences were not significant). In men with non-malignant tumours the mean concentration was 0.31 mmol/l lower and in women with non-malignant tumours it was 0.21 mmol/l higher than in those without tumours. These differences were also not significant.

Morbidity* from brain tumours per million person years by tertiles of serum cholesterol concentration and relative risk* of brain tumours between highest and lowest tertiles

	No of cases	Serum cholesterol (mmol/l)			Relative risk	95% Confidence interval
		<5.95	5.95-7.09	≥7.10		
Men:						
All tumours	42	110	157	112	1.02	0.45 to 2.31
Malignant tumours	30	58	130	78	1.36	0.47 to 3.88
Non-malignant tumours	12	54	27	35	0.64	0.17 to 2.41
Women:						
All tumours	30	104	73	101	0.97	0.39 to 2.41
Malignant tumours	14	52	27	49	0.95	0.26 to 3.56
Non-malignant tumours	16	51	49	52	1.02	0.28 to 3.66
Men and women:						
All tumours	72	110	117	101	0.93	0.50 to 1.69
Malignant tumours	44	57	81	60	1.05	0.47 to 2.34
Non-malignant tumours	28	53	37	41	0.78	0.31 to 1.94

*Adjusted for age based on Cox's life table regression model.

The table gives the age adjusted morbidity associated with brain tumours by tertiles of serum cholesterol concentration and the relative risks of brain tumours in the highest compared with the lowest tertile. The relative risk of a malignant or non-malignant brain tumour was 1.02 in men and 0.97 in women, which suggests there was no association between serum cholesterol concentration and the occurrence of brain tumours. The relative risk of a malignant tumour was higher (1.36) among men, but it did not differ significantly from unity. Further adjustment for body mass index and occupation did not notably alter the results; neither did exclusion of tumours occurring during the first two years of follow up, except in women with non-malignant tumours, in whom the association was strengthened, with the relative risk rising from 1.02 to 1.44 (95% confidence interval 0.37 to 5.64).

Comment

We investigated the possibility of an association between serum cholesterol concentration and the incidence of malignant and non-malignant brain tumours in a large cohort with relatively high serum cholesterol concentrations. No notable association was found. Thus our study does not confirm the findings of previous studies.¹⁻⁴

In the present cohort the mean serum cholesterol concentration was approximately 1.5 mmol/l higher than in the Whitehall population.⁴ A possible association between the presence of brain tumours and serum cholesterol concentration might, therefore, have been missed in our study if it existed only at low serum cholesterol concentrations, but this is unlikely. The possibility cannot be excluded that the results of the previous case-control studies may be artefactual because an existing brain tumour may have caused a spurious increase in the serum cholesterol concentration.¹⁻³

In conclusion, our results, which are based on the largest population reported so far, do not support the conclusion that a high serum cholesterol concentration is a risk factor for brain tumours.

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