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Passive smoking as well as active smoking increases the risk of acute stroke

Ruth Bonita, John Duncan, Thomas Truelsen, Rodney T Jackson, Robert Beaglehole

Abstract

***Objective*—To estimate the relative risk of stroke associated with exposure to environmental tobacco smoke (ETS, passive smoking) and to estimate the risk of stroke associated with current smoking (active smoking) using the traditional baseline group (never-smokers) and a baseline group that includes lifelong non-smokers and long-term (>10 years) ex-smokers who have not been exposed to ETS.**

***Design and setting*—Population-based case-control study in residents of Auckland, New Zealand.**

***Subjects*—Cases were obtained from the Auckland stroke study, a population-based register of acute stroke. Controls were obtained from a cross-sectional survey of major cardiovascular risk factors measured in the same population. A standard questionaire was administered to patients and controls by trained nurse interviewers.**

***Results*—Information was available for 521 patients with first-ever acute stroke and 1851 community controls aged 35–74 years. After adjusting for potential confounders (age, sex, history of hypertension, heart disease, and diabetes) using logistic regression, exposure to ETS among non-smokers and long-term ex-smokers was associated with a significantly increased risk of stroke (odds ratio (OR) = 1.82; 95% confidence interval (95% CI) = 1.34 to 2.49). The risk was significant in men (OR = 2.10; 95% CI = 1.33 to 3.32) and women (OR = 1.66; 95% CI = 1.07 to 2.57). Active smokers had a fourfold risk of stroke compared with people who reported they had never smoked cigarettes (OR = 4.14; 95% CI = 3.04 to 5.63); the risk increased when active smokers were compared with people who had never smoked or had quit smoking more than 10 years earlier and who were not exposed to ETS (OR = 6.33; 95% CI = 4.50 to 8.91).**

***Conclusions*—This study is one of the few to investigate the association between passive smoking and the risk of acute stroke. We found a significantly increased risk of stroke in men and in women. This study also confirms the higher risk of stroke in men and women who smoke cigarettes compared with non-smokers. The stroke risk increases further when those who have been exposed to ETS are excluded from the non-smoking reference group. These findings also suggest that studies investigating the adverse effects of smoking will underestimate the risk if exposure to ETS is not taken into account.**

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Keywords: environmental tobacco smoke; stroke; smoking-attributable diseases

Department of Medicine, Faculty of Medicine and Health Science, University of Auckland, Auckland, New Zealand
R Bonita
J Duncan
T Truelsen

Department of Community Health
R T Jackson
R Beaglehole

Correspondence to:
Ruth Bonita, University Geriatric Unit, North Shore Hospital, Private Bag 93-503, Takapuna, Auckland 9, New Zealand;
r.bonita@auckland.ac.nz

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Introduction

There is now strong evidence of an independent causal association between cigarette smoking and ischaemic stroke and haemorrhagic stroke.[1–5] Few studies have examined the association of exposure to environmental tobacco smoke (ETS, passive smoking) and the subsequent risk of stroke[6] although a recent meta-analyses of over 20 epidemiological studies has found an adverse effect of passive smoking on the subsequent risk of coronary heart disease.[7]

This study examines the association between stroke and current (active) smoking using the traditional baseline of non-smokers as the reference group. In addition, we estimated the relative risk of stroke associated with exposure to ETS. A separate analysis using non-smokers not exposed to ETS as the reference group was used to investigate whether the stroke risk associated with exposure to cigarette smoke is underestimated using traditional definitions of non-smokers.

Methods

The patients for this study were taken from the Auckland stroke study, which documented all stroke events in residents of the Auckland population aged 15 years and over (total population 952 000 in the 1991 census) in 1991–92. The diagnostic criteria and methods were based on the World Health Organisation guidelines.[8] Stroke was defined as the rapid onset of focal neurological deficit lasting 24 hours or longer, or leading to death, and presumably of vascular origin. Multiple case finding methods were used to ensure that all residents in the Auckland region who experienced a stroke were identified. Details of these methods have been published previously.[9 10] Information was obtained by trained nurse interviewers as soon after the event as possible, either from the patient or from the main caregiver if the patient had speech or communication problems. If the patient had died, the same questionnaire was administered to a close relative or caregiver after six weeks.

Controls were participants in a 1993–94 major cross-sectional survey of the prevalence

Table 1 Age and sex distribution of patients with stroke and community controls and mean age within age bands

Age group (years)	Patients with stroke (n = 521)			Community controls (n = 1851)		
	n	(%)	Mean age	n	(%)	Mean age
Men						
35–44	18	6.5	41.6	226	24.2	40.3
45–54	23	8.2	50.5	226	24.2	49.7
55–64	82	29.4	60.5	251	26.9	59.6
65–74	156	55.9	69.4	231	24.7	69.7
Total	279	53.6		934	50.5	
Women						
35–44	13	5.4	39.6	211	23.0	39.8
45–54	28	11.6	51.1	236	25.7	49.5
55–64	51	21.0	60.0	232	25.3	59.7
65–74	150	62.0	69.9	238	26.0	69.8
Total	242	46.4		917	49.5	

of cardiovascular risk factors in the Auckland population, the methods of which have been published.[11] The study population included men and women aged 35–74 years resident within the Auckland statistical area. Samples stratified by age and sex were chosen at random from the Auckland general electoral rolls with 250 people randomly selected for each 10-year age and sex band to ensure time trend differences would be detected. Maori and Pacific people were not included in the survey because the general electoral rolls do not provide a representative sample of Maori, as approximately half of all Maori are registered on a separate Maori electoral roll. Altogether 104 Maori and Pacific patients with stroke and 80 Maori and Pacific community controls were omitted from the analyses. Participants completed a standard interviewer-administered questionnaire at the survey centre.

Questions about smoking history were the same for patients and controls. An active smoker was defined as a person who currently smoked at least one cigarette a day. An ex-smoker was defined as a person who had regularly smoked but who had not smoked in the previous month. Participants were included in the passive smoking analyses if they had never smoked cigarettes or had stopped smoking cigarettes for at least 10 years before the date of the interview and were not current smokers of pipes, cigars, or cigarillos. Exposure to ETS was ascertained using data derived from the same questions asked of patients and controls. A person was deemed to have been exposed to ETS if a household member had regularly smoked cigarettes in their presence or if a co-worker smoked in the same indoor room in their presence for more than one year during the past 10 years. These definitions are in accord with similar analyses investigating the association of passive smoking with coronary heart disease.[12]

Participants were categorised as having hypertension if they had a history of hypertension or treatment for hypertension. A history of diabetes was determined by self report in the patients and the controls. Information on past history of stroke or heart disease was obtained by self report for patients and controls. For the purposes of these analyses, only first-ever strokes were included; 230 patients with stroke and 41 community controls who had reported a previous stroke were excluded from the analyses.

Odds ratios (ORs) and 95% confidence intervals (95% CIs) for stroke associated with smoking, ex-smoking, and passive smoking, history of hypertension, ischaemic heart disease, and diabetes were calculated using the Cochran-Mantel-Haenszel method controlling for age and sex. The extended Mantel-Haenszel test was used to test for a dose response across active smoking categories. Variables representing these exposures were entered into an unconditional logistic regression model to provide summary odds ratios adjusted for potential confounders. An offset parameter was fitted to the logistic regression model to control for the different sampling fractions used between the case and control studies.[13 14] The cigarettes smoked per day were then fitted as a continuous measure in the logistic regression model to test for a dose response across active smoking categories. The precision of odds ratios was estimated using 95% CIs calculated by conditional maximum likelihood using the statistical software SAS v.6.12.[15] Odds ratios are described as statistically significant if the 95% CIs exclude an OR of 1.0.

All analyses were conducted separately for men and women. As there appeared to be no significant differences with respect to the prevalence of risk factors or odds ratios, the data for men and women have been combined in the tables.

Results

There were 521 patients with first-ever stroke (279 men and 242 women) and 1851 community controls (934 men and 917 women). The age and sex distributions of the patients and controls are shown in table 1. The difference in the proportions within age bands between the patients and controls reflects the sampling frame used in the cardiovascular risk factor survey; calculation of the overall mean age was therefore inappropriate. Within age bands the mean age was similar for patients and controls.

The prevalence of selected risk factors and their association (odds ratios adjusted for age and sex) with stroke is shown in table 2. Almost a third (31.5%) of the patients with stroke were active smokers, compared with 13.8% of the community controls. After adjusting for age and sex, current smokers had more than four times the risk of stroke (OR = 4.27; 95% CI = 3.23 to 5.65) compared with people who had never smoked. Although not shown in the table, the risk of stroke associated with active smoking, adjusted for age, was similar in men (OR = 4.07; 95% CI = 2.74 to 6.04) and in women (OR = 4.50; 95% CI = 3.03 to 6.69). A dose response with the number of cigarettes smoked was observed ($p<0.001$). The higher risk in ex-smokers compared with people who have never smoked reached statistical significance in recent ex-smokers (those who had quit within the previous two years) (OR = 2.49; 95% CI = 1.40 to 4.45). People who had given up smoking between two and 10 years

previously also had an increased risk (OR = 1.48; 95% CI = 1.01 to 2.17) but among ex-smokers who had quit more than 10 years previously, no increased risk was observed.

Table 2 Distribution of risk factors in patients with first-ever stroke and community controls and odds ratio associated with the risk of stroke

	Patients with stroke (n = 521)		*Community controls (n = 1851)*			
	n	*(%)*	*n*	*(%)*	*OR**	*95% CI*
Smoking status						
Never smoked	162	31.1	901	48.7	1.00	Referent
Active smokers	164	31.5	255	13.8	4.27	3.23–5.65
⩾15 cigarettes/day	95	18.2	154	8.3	4.65	3.32–6.53
6–14 cigarettes/day	42	8.1	62	3.4	3.90	2.50–6.08
⩽5 cigarettes/day	27	5.2	39	2.1	3.57	2.07–6.15
Never smoked	162	31.1	901	48.7	1.00	Referent
Ex-smokers	174	33.4	671	36.2	1.18	0.91–1.53
<2 years	22	4.2	46	2.4	2.49	1.40–4.45
2–10 years	49	9.4	190	10.3	1.48	1.01–2.17
>10 years	103	19.8	435	23.5	0.97	0.72–1.32
Unknown years	5	1.0	1	0.1	ND	ND
Passive smokers†	265	50.9	1336	72.2	ND	ND
Not exposed	110	21.1	660	35.7	1.00	Referent
Exposed	155	29.8	676	36.5	1.74	1.31–2.32
Unknown exposure	16	3.0	23	1.2	ND	ND
Hypertensive						
No	196	37.6	1241	67.0	1.00	Referent
Yes	325	62.4	610	33.0	2.59	2.10–3.20
History of heart disease						
No	429	82.3	1796	97.0	1.00	Referent
Yes	92	17.7	55	3.0	4.21	3.00–5.93
Diabetes						
No	445	85.4	1782	96.3	1.00	Referent
Yes	76	14.6	69	3.7	3.27	2.32–4.61

*Odds ratio adjusted for age and sex using the Cochran-Mantel-Haenszel method.
†Includes ex-smokers who quit more than 10 years previously.
95% CI = 95% confidence intervals; ND = no data.

Table 3 Risk of first-ever stroke associated with active smokers and ex-smokers, men and women aged 35–74 years

	Reference group 1: non-smokers (exposed and not exposed to ETS)					
	Patients with stroke (n = 521)		*Community controls (n = 1851)*			
Smoking status	*n*	*(%)*	*n*	*(%)*	*OR**	*95% CI*
Never smoked	162	31.1	901	48.7	1.00	Referent
Active smokers	164	31.5	255	13.8	4.14	3.04–5.63
⩾15 cigarettes/day	95	18.2	154	8.3	4.59	3.17–6.63
6–14 cigarettes/day	42	8.1	62	3.4	4.37	2.61–7.32
⩽5 cigarettes/day	27	5.2	39	2.1	2.56	1.35–4.88
Ex-smokers	174	33.4	671	36.2	1.00	0.75–1.32
<2 years	22	4.2	46	2.4	2.30	1.24–4.27
2–10 years	49	9.4	190	10.3	1.23	0.80–1.88
>10 years	103	19.8	435	23.5	0.79	0.57–2.51

*Adjusted for age, sex, diabetes, hypertension, and history of heart disease in logistic regression analysis.
ETS = environmental tobacco smoke; OR = odds ratios; CI = confidence intervals.

Table 4 Risk of first-ever stroke associated with active smoking, ex-smoking, and passive smoking, men and women aged 35–74 years

	Reference group 2: non-smokers (not exposed to ETS)					
	Patients with stroke (n = 521)		*Community controls (n = 1851)*			
Smoking status	*n*	*(%)*	*n*	*(%)*	*OR**	*95% CI*
Passive smokers†						
Not exposed	110	21.1	660	35.7	1.00	Referent
Exposed	155	29.8	676	36.5	1.82	1.34–2.49
Active smokers	164	31.5	255	13.8	6.33	4.50–8.91
⩾15 cigarettes/day	95	18.2	154	8.3	7.06	4.75–10.49
6–14 cigarettes/day	42	8.1	62	3.4	6.63	3.89–11.30
⩽5 cigarettes/day	27	5.2	39	2.1	3.89	2.03–7.47
Ex-smokers	71	13.6	236	12.7	2.21	1.50–3.27
<2 years	22	4.2	46	2.4	3.45	1.84–6.46
2–10 years	49	9.4	190	10.3	1.89	1.21–2.93

*Odds ratios adjusted for age, sex, diabetes, hypertension, and history of heart disease in logisitic regression analysis. †Includes ex-smokers who had quit more than 10 years previously. ETS = environmental tobacco smoke; CI = confidence intervals.

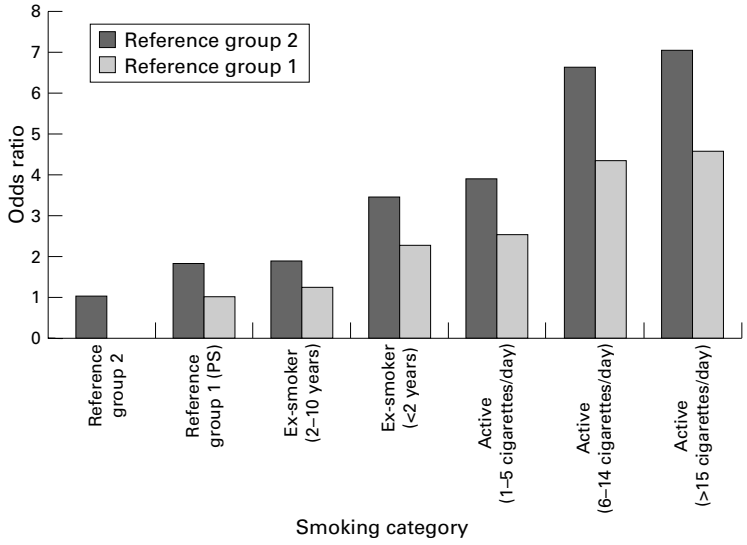
Information on exposure to ETS was available for 97.0% of patients with stroke and 98.8% of community controls. Exposure was associated with an increased risk in men (OR = 2.06; 95% CI = 1.34 to 3.17) and women (OR = 1.50; 95% CI = 1.01 to 2.21). After adjustment for age and sex, exposure to ETS was associated with an overall increased risk of stroke (OR = 1.74; 95% CI = 1.31 to 2.32).

A statistically significant association with the risk of stroke was seen for hypertension (OR = 2.59; 95% CI = 2.10 to 3.20), history of heart disease (OR = 4.21; 95% CI = 3.00 to 5.93), and diabetes (OR = 3.27; 95% CI = 2.32 to 4.61) and the associations were significant in men and women.

When the above risk factors were entered into a multivariate model, the risk of stroke in active smokers compared with people who have never smoked remained statistically significant (OR = 4.14; 95% CI = 3.04 to 5.63) as shown in table 3. Although not shown in the table, the risk in men was over three and a half times greater (OR = 3.79; 95% CI = 2.44 to 5.89) and in women was four and a half times greater (OR = 4.48; 95% CI = 3.10 to 7.55). A dose response was observed for the three different categories of active smokers, ranging from 2.56 (⩽5 cigarettes/day) to 4.59 (⩾15 cigarettes/day). The test for a dose response using logistic regression was significant ($p<0.001$). Recent ex-smokers (less than two years) had a significantly increased risk of stroke (OR = 2.30; 95% CI = 1.24 to 4.27). In comparison, there was no increased risk of stroke in people who had quit smoking more than two years ago.

Table 4 presents data on the effect of passive smoking on stroke and in addition, examines the extent to which the choice of a baseline reference group makes a difference to the overall outcome. When the reference group is restricted to include only those non-smokers who had not been exposed to ETS, the risk of stroke in active smokers is increased even further—to sixfold (OR = 6.33; 95% CI = 4.50 to 8.91) and twofold in ex-smokers (OR = 2.21; 95% CI = 1.50 to 3.27). Again, a dose response with increasing numbers of cigarettes smoked was evident ($p<0.001$) and a higher risk of stroke was found in recent (less than two years) ex-smokers (OR = 3.45; 95% CI = 1.84 to 6.46). In comparison with the results found in table 3, an increased risk of stroke in smokers who had quit between two and 10 years previously reached statistical significance (OR = 1.89; 95% CI = 1.21 to 2.93).

Of particular interest, our results suggest that non-smokers who have been exposed to ETS have an increased risk of stroke of approximately 82% (OR = 1.82; 95% CI = 1.34 to 2.49). The risk is statistically significant in men (OR = 2.10; 95% CI = 1.33 to 3.32), and in women (OR = 1.66; 95% CI = 1.07 to 2.57). The estimate of the risks of passive smoking using the two different reference groups (as shown in tables 3 and 4) are also shown in the figure.



A comparison of the association (odds ratio) between active smokers, ex-smokers, and passive smokers (PS) and the risk of stroke using two different references groups (group 1 = non-smokers exposed and not exposed to ETS; group 2 = non-smokers not exposed to ETS).

Discussion

This study contributes to the published work on the relation between smoking and stroke in three important ways. Firstly, it confirms a substantially increased risk of stroke in men and women who are active cigarette smokers.[2 4 5 16 17] Secondly, this study demonstrates that the estimated risk of stroke associated with active smoking is even higher when the non-smoking reference group excludes people exposed to ETS. These results suggest that published reports of the association of smoking with stroke using either non-smokers or never-smokers as the reference group without differentiating between those exposed and those not exposed to ETS, are likely to have underestimated the adverse effects of smoking. Thirdly, of particular significance, our results also suggest that passive smoking, known to be associated with an increased risk of heart disease, is also associated with subsequent stroke in men and women.

This is one of the few population-based case-control studies of the association of stroke with smoking status in which the controls are derived from the same population as the patients and with sufficient power to examine associations with precision. Studies based on hospital admissions cannot be regarded as optimum for the investigation of passive smoking and the risk of stroke. In most countries patients with stroke are often not admitted to hospital, either because they die before admission or because they are treated at home. Further, analyses based on deaths from stroke are not sufficient as passive smoking may be associated with either relatively mild or relatively severe strokes. The strength of this study is that all strokes, whether fatal or not, within the Auckland population have been identified. Patients were defined under strict criteria and there is good reason to believe that all patients were correctly identified through the multiple case finding procedures. Controls obtained from a large random sample of the same population were interviewed by the same interviewers using the same questions.

Despite the efforts to make the two study groups as comparable as possible, there may have been bias in obtaining information about smoking habits; it is possible that individuals with stroke report smoking consumption differently from healthy individuals, and vice versa. The low proportion of smokers in the community controls reflects the low rates of smoking in adults (but not adolescents and children) in New Zealand. The two nurses who identified the patients with stroke also interviewed the community controls using identical questions asked in the same standard manner. The questions were embedded in a large number of other questions thus reducing the potential for information bias.

No attempt was made in the analyses to differentiate between exposure at home, at work, or elsewhere. Exposure at work has been found to confer a greater risk for coronary heart disease than exposure at home.[18 19] Studies that report exposure only at home or at work could lead to an underestimation of the effect because of the different employment patterns of men and women. The lower association between passive smoking and the risk of stroke in women could be due to gender related differences in exposure—for example, at home and at work—or may be due to chance. Among those people exposed to ETS, more men (21.2%) stated that their exposure was "moderate" to "heavy" compared with women (14.0 %) ($p = 0.016$).

The difficulty in ascertaining exposure to ETS may also explain the lack of precision of these estimates.[20] The effect of this form of bias is to make the distinction between those exposed and those not exposed to ETS less clear; this bias would have the effect of reducing the strength of the associations demonstrated.

In this study we have not estimated the reliability of the classification of the subjects who reported themselves respectively exposed and not exposed to ETS. Other studies have found that the reliability of self-classification of exposure to ETS is moderate to very good.[21 22] Uncertainties about the true classification of the subjects will decrease the precision of the data and reduce the estimated association provided the misclassification is non-differential.[23] We have no evidence that people with stroke would over-report their exposure to ETS, and publications suggest that, at the population level, the knowledge as to what causes stroke is sparse. Furthermore, few studies have addressed the issue of the association between passive smoking and stroke, so this is not generally known as a risk factor for stroke. In a study of the association between passive smoking and lung cancer, an association which is likely to be known to the general population, no differences between patients and controls were found with regard to questions on the perceived harmfulness of ETS.[21]

Another limitation is the inability to differentiate between subtypes of stroke. Among the patients, 9.2% (n = 48) had a sub-

arachnoid haemorrhage and 7.3% (n = 38) had a known intracerebral haemorrhage. These numbers were too small to undertake meaningful separate analyses. However, there is no reason to believe that the inclusion of all acute strokes would have biased the results as it has been previously reported that smoking is associated with all stroke subtypes.[24] Information on education and socioeconomic status could not be accurately obtained in the Auckland stroke study because most participants were elderly; almost 60% of patients with acute stroke in this study were 65–74 years of age, well past the age of retirement. The exclusion of Maori and Pacific people, who have both lower socioeconomic status, lower mean age, higher smoking rates, and a higher risk of stroke than Europeans,[25] would have reduced any potential confounding by socioeconomic status.

The association of active smoking with stroke has now been demonstrated in many studies of different designs and is likely to be causal. The high odds ratios found in this study are in accord with a number of recent well-designed studies.[25 26] The only study to examine the association of ETS and the risk of stroke is a case-control study that was restricted to hospital patients.[6]

The major finding of an independent increased risk of stroke associated with exposure to environmental tobacco smoke provides support for current efforts to reduce the prevalence of passive smoking and strengthens public health arguments against smoking. This information is also important for the development of comprehensive policies for the control of the health damaging effects of the tobacco industry. Strong public health action is overdue.[27]

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