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Passive exposure to tobacco smoke: saliva cotinine concentrations in a representative population sample of non-smoking schoolchildren

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Abstract

Saliva cotinine concentrations in 569 non-smoking schoolchildren were strongly related to the smoking habits of their parents. When neither parent smoked the mean concentration was 0.44 ng/ml, rising to 3.38 ng/ml when both parents were cigarette smokers. Mothers' smoking had a stronger influence than did fathers' ($p < 0.01$). In addition, there was a small independent effect of number of siblings who smoked ($p < 0.01$). The dose of nicotine received from fathers' smoking was estimated as equivalent to the active smoking of 30 cigarettes a year, that from mothers' smoking as equivalent to smoking 50 cigarettes a year, and that from both parents smoking as equivalent to smoking 80 cigarettes a year. This unsolicited burden may be prolonged throughout childhood and poses a definite risk to health.

Introduction

An increasing number of studies have implicated passive smoking as a definite threat to non-smokers' health.¹ Reported effects have ranged from an increase in respiratory illness in

infancy and childhood^{2,3} to impaired lung function⁴ and lung cancer⁵ in adults. These epidemiological results are now being complemented and made more plausible by findings that concentrations of biochemical markers of smoke absorption in non-smokers are raised in proportion to their reported exposure to other people's smoke.⁶⁻⁹ Nevertheless, most studies of the dose received from passive exposure to tobacco smoke have been conducted in somewhat atypical groups—for example, hospital outpatients⁶ or men attending for health screening examinations.⁷ There remains a need to quantify the dose from passive exposure in more representative samples of the population in order to inform estimates of the burden that passive smoking may impose on the whole community.

There is now agreement that cotinine is the marker of choice for quantifying passive exposure to smoke. As a major metabolite of nicotine it shares with its parent compound the advantage of being specific to tobacco smoke, but its longer half life (about 24 hours) means that it reflects exposure over the past few days rather than hours. We have examined the relation of saliva cotinine concentrations in a representative sample of non-smoking secondary school children to their exposure to tobacco smoke within the family.

Subjects and methods

The data were gathered as part of a survey of smoking attitudes and behaviour carried out in the spring of 1983 among schoolchildren aged 11-16. Preliminary data relating to the prevalence of smoking have been published elsewhere.¹⁰ Questionnaires were completed by 10 579 children attending 10 mixed secondary comprehensive schools selected at random from the Bristol area. The schools represented a range of inner urban and suburban environments. The sample comprised all those in the first five years who were present on the days of the survey. The questionnaires were administered by 26 trained research assistants, who followed a carefully standardised procedure. Before completing the questionnaire the children were told that some of them would be selected at random and asked to provide a quantity of saliva, which would be analysed to see if they had been smoking recently. This procedure has been shown to increase the truthfulness of self reports of smoking.¹¹ Saliva samples were gathered from 2594 pupils. Table I shows that the social class

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distribution of the study sample closely matched national figures. The prevalence of reported parental cigarette smoking was also similar to that found nationally. Thirty nine per cent of fathers were reported to smoke cigarettes compared with 40% among men aged 30-49 nationally, while the figure of 37% of mothers was the same as the 1982 General Household Survey figure for women aged 30-49.¹²

Non-smoking children were defined as those who ticked one of the following three items in the questionnaire—namely, "I have never smoked a cigarette, not even a puff"; "I have only ever tried smoking once or twice"; "I used to smoke sometimes, but I don't now"—and who in addition said that they had not smoked any cigarettes in the past week. Passive exposure to smoke was indexed by responses to the questionnaire items "Does your father smoke cigarettes?" and "Does your mother smoke cigarettes?" In addition, children were asked how many of their siblings smoked. No quantitative information was available about cigarette consumption.

Analyses for saliva cotinine by gas chromatography¹³ were performed on a random subsample of 1018. Analysis was blind to questionnaire results. A total of 150 specimens had to be discarded owing to insufficient volume (n=110) or contamination (n=40), leaving 868 subjects for whom measured cotinine concentrations were available. Of these, 233 reported smoking within the past week or had missing data on smoking state. A further 60 non-smoking children were excluded either because one parent was absent from home or because of missing data on parental smoking. Finally, six subjects were excluded from the sample of non-smokers because their concentrations of saliva cotinine were above 11 ng/ml (an arbitrary but conservative cut off value) and hence were regarded as not consistent with non-smoking (saliva cotinine values of 212.2, 81.6, 47.2, 39.3, 14.0, and 11.1 respectively). This left 569 non-smoking children who had measured cotinine concentrations and complete questionnaire data relating to passive exposure to tobacco smoke within the family.

Results

Tables II and III show the mean concentrations and percentiles of the distributions for saliva cotinine according to parental smoking state. Where neither parent smoked concentrations were low, the mean value being 0.44 ng/ml and the median 0.20 ng/ml. Over 30% of children from non-smoking homes had undetectable concentrations, and the highest value encountered in this group was 4.8 ng/ml. Concentrations were increased when only father was a cigarette smoker (mean 1.31 ng/ml, median 1.00 ng/ml), and were still higher when only mother smoked (mean 1.95 ng/ml, median 1.35 ng/ml). When both parents were cigarette smokers the average saliva cotinine concentration reached 3.38 ng/ml and the median was 2.70 ng/ml. The effects of parental smoking were additive, so that the mean for father only exposure added to the mother only mean closely approximated the mean when both parents smoked.

TABLE I—Socioeconomic distribution of study children and comparison with national data.* Values are percentages

	Whole sample (n = 10 579)	Passive smoking subsample (n = 569)	General Household Survey, 1982
Professional	5	6	5
Employees and managers	17	17	18
Intermediate and junior non-manual	16	14	16
Skilled manual and own account non-professional	45	47	40
Semiskilled manual and personal service	13	13	16
Unskilled manual	4	3	5

*Socioeconomic group determined by father's occupation (Office of Population Censuses and Surveys classification, 1980). General Household Survey data are for male heads of household.

TABLE II—Saliva cotinine concentrations by parental cigarette smoking

	No	Saliva cotinine (ng/ml)		
		Mean (SD)	Median	Interquartile range
Neither parent smokes	269	0.44 (0.68)	0.20	0.00*-0.50
Only father smokes	96	1.31 (1.21)	1.00	0.40-1.80
Only mother smokes	76	1.95 (1.71)	1.35	0.73-2.68
Both parents smoke	128	3.38 (2.45)	2.70	1.53-4.40
Total	569	1.45 (1.88)	0.70	0.15-1.90

*0.00 indicates undetectable concentration of cotinine.

TABLE III—Percentiles of distributions of saliva cotinine concentrations by parental cigarette smoking

Percentiles	Neither parent smokes (n = 269)	Only father smokes (n = 96)	Only mother smokes (n = 76)	Both parents smoke (n = 128)
10	0.00*	0.10	0.24	1.00
20	0.00	0.30	0.60	1.28
30	0.00	0.50	0.90	1.80
40	0.10	0.88	1.10	2.20
50	0.20	1.00	1.35	2.70
60	0.30	1.32	1.72	3.30
70	0.50	1.60	2.47	3.93
80	0.60	1.90	3.12	5.04
90	1.30	2.88	4.79	7.72

*0.00 indicates undetectable concentration of cotinine.

Log saliva cotinine concentrations in 569 non-smoking schoolchildren by parental cigarette smoking (means $\pm$ 95% confidence limits). Transformation used was $\log(x+0.1)$, 0.1 ng/ml being smallest concentration of cotinine detectable.

Because distributions were skewed the data were subjected to logarithmic transformation before statistical comparisons were made. Analysis of variance showed that the effects of parental smoking state on cotinine concentrations were highly significant ($F=154.4$, $df=3$, 565 ; $p<0.001$). Comparisons among groups by Duncan's multiple range test showed that each of the four passive exposure categories (neither parent smokes, father only, mother only, both smoke) was significantly different from every other at the 0.01 level, with the exception of the father only *v* mother only comparison, where the significance level was 0.05. The figure shows log mean saliva cotinine concentrations $\pm$ 95% confidence limits as used in the statistical analysis.

To investigate whether other measured variables besides parental smoking influenced cotinine concentrations in these non-smoking children a multiple regression analysis was performed. Table IV gives the results. When all variables were entered into the equation there was a small independent effect of smoking by siblings ($p<0.01$) in addition to the major effects of mothers' and fathers' smoking. None of the other variables entered had any significant independent influence. In particular, the age, sex, and socioeconomic background of the child and the school attended had no bearing on cotinine concentrations. Nor was there any difference according to whether the child had never smoked, had tried smoking once or twice, or used to smoke but had given up.

Discussion

Our results show that schoolchildren with parents who smoke receive a significant dose of nicotine through passive exposure. The average concentration of cotinine in saliva rose in a dose related fashion from 0.44 ng/ml in children with non-smoking parents to 3.38 ng/ml when both parents smoked. These findings

TABLE IV—Multiple regression predicting saliva cotinine concentration*

	Simple correlation r	Stepwise analysis		Final model	
		Multiple r	r ²	Beta coefficient	Significance (p)
Mother's smoking state	0.56	0.56	0.31	0.42	<0.0001
Father's smoking state	0.51	0.65	0.43	0.34	<0.0001
Number of siblings who smoke	0.16	0.66	0.44	0.11	<0.01
Age	-0.04	0.66	0.44	-0.04	0.30
Sex	-0.07	0.66	0.44	-0.01	0.75
Child's academic achievement	-0.13	0.66	0.44	-0.04	0.25
School attended	-0.15	0.66	0.44	-0.03	0.45
Mother's socioeconomic group	0.12	0.66	0.44	0.01	0.89
Child's smoking history	-0.04	0.66	0.44	0.01	0.71
Father's socioeconomic group	0.23	0.66	0.44	0.05	0.21

*Concentrations subjected to logarithmic transformation. Significance levels are those when all variables were in equation.

relate to children from a range of living environments and whose socioeconomic backgrounds were representative of Britain as a whole. They are therefore likely to provide an accurate guide to the picture throughout the United Kingdom.

One concern with studies of the effects of passive smoking is that there are potentially important sources of bias. In particular any inclusion of active smokers among the passive smoking sample would give rise to spurious results. This is especially problematic since those who conceal their smoking are likely to claim to be heavily exposed to other people's smoke.⁶ We are confident that our data do not suffer seriously from problems of deception. Firstly, the self reported prevalence of smoking was similar to other survey data,¹⁴ and only six children who claimed to be non-smokers had cotinine concentrations which exceeded our conservative cut off value (11 ng/ml). In two of these the concentrations were close to the cut off value, so that we may inadvertently have excluded true non-smokers with exceptionally heavy passive exposure. Another consideration is that if deception rates were a problem we might expect to have seen differences in the results according to whether the children claimed to be never smokers, triers, or ex-smokers, since current smokers might be more likely to claim to be ex-smokers or triers than never smokers. The results, however, were similar for children in each of these non-smoker categories. Finally, that the distributions for the passive smoking categories were shifted throughout their range rather than differing because of a few outliers strongly suggests that our findings reflect true passive smoking effects.

Although we sought no information about parental cigarette consumption, it is reasonable to suppose that this would reflect national averages, particularly since the prevalence of cigarette smoking among parents closely matched national rates. The General Household Survey for 1982 found that male cigarette smokers averaged 20 cigarettes a day and women smokers 15 a day.¹² Despite this higher consumption in men we found that mothers' smoking had a stronger influence on children's exposure than did fathers'. This is presumably because children spend comparatively more of their time with their mothers or because proportionally more of the mothers' smoking takes place in the home. Our findings on dose parallel several reports that maternal smoking is a stronger risk factor than paternal smoking for respiratory disease among non-smoking children.^{3 15 16}

It is interesting to quantify the dose of nicotine received by our sample of schoolchildren by comparison with adult cigarette smokers. Few studies have measured saliva cotinine concentrations in smokers, but the concentration is in approximate equilibrium with that in blood,^{6 17 18} so that blood cotinine concentrations may be used for comparison. Studies have shown that the concentration of cotinine in serum and plasma averages about 300 ng/ml in smokers consuming 20-30 cigarettes a day.¹⁹⁻²² Thus the dose of nicotine in our sample of schoolchildren expressed as a percentage of the amount in heavy cigarette

smokers was 0.1 in children neither of whose parents smoked, 0.4 when only father smoked, 0.7 when only mother smoked, and 1.1 when both parents smoked. If we assume that an average smoker of 20 cigarettes a day derives a concentration of 300 ng/ml in blood and saliva, then the nicotine intake of these children was equivalent to the active smoking of roughly 11, 30, 50, and 80 cigarettes a year respectively in the four passive smoking categories. Since the concentration of some smoke components in sidestream relative to mainstream smoke is greater than in the case of nicotine, these percentages may be conservative in providing an estimate of children's passive exposure to cigarette smoke.²³

Most studies of the health effects of passive smoking have been on infants or young children or on adults. One recent study, however, has examined effects among schoolchildren of the same ages as those studied here.²⁴ Prevalence of coughs was found to be related to parental smoking, with mothers' smoking relatively more important than fathers' and the highest rates of reported coughs in children both of whose parents smoked. Those results mirror the findings on dose in our study and confirm that passive smoking in the range that we have documented may have deleterious effects on health.

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