Clinical implications

- Whether passive smoking is a cause of coronary heart disease is still controversial
- Previous studies have shown that coronary heart disease is associated with passive smoking at home but not with passive smoking at work
- This study shows that for women's passive smoking at work there were significant linear trends of increased risks of coronary heart disease with increasing exposures, even after adjustment for major risk factors and passive smoking from husband
- Urgent public health measures are needed to reduce smoking in China

exposed to tobacco smoke at home and at work. They are also unaware of the hazards of passive smoking and are in a disadvantaged position to protect themselves from environmental tobacco smoke. Urgent public health measures are required to reduce smoking in China so as not only to protect women from environmental tobacco smoke but also men from the hazards of active smoking.

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Passive exposure to tobacco smoke in children aged 5-7 years: individual, family, and community factors

Derek G Cook, Peter H Whincup, Martin J Jarvis, David P Strachan, Olia Papacosta, Andrew Bryant

Department of Public Health Sciences, St George's Hospital Medical School, London SW17 0RE
Derek G Cook, *senior lecturer in epidemiology*
David P Strachan, *senior lecturer in epidemiology*

Department of Public Health and Primary Care, Royal Free Hospital School of Medicine, London
Peter H Whincup, *senior lecturer in clinical epidemiology*
Olia Papacosta, *statistician*

Imperial Cancer Research Fund Health Behaviour Unit, Institute of Psychiatry, London
Martin J Jarvis, *assistant director*

National Poisons Unit, New Cross Hospital, London
Andrew Bryant, *scientific officer*

Correspondence to: Dr Cook.

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Abstract

***Objective*—To examine the importance of parental smoking on passive exposure to tobacco smoke in children and the social and geographical patterns of exposure.**

***Design*—Cross sectional study.**

***Setting*—Schools in 10 towns in England and Wales; five towns with high adult cardiovascular mortality and five with low rates.**

***Subjects*—4043 children aged 5-7 years of European origin.**

***Main outcome measures*—Salivary cotinine concentration and parents self reported smoking habits.**

***Results*—1061 (53·0%) children were exposed to cigarette smoke at home or by an outside carer. Geometric mean cotinine rose from 0·29 (95% confidence interval 0·28 to 0·31) ng/ml in children with no identified exposure to 4·05 (3·71 to 4·42) ng/ml in households where both parents smoked and 9·03 (6·73 to 12·10) ng/ml if both parents smoked more than 20 cigarettes a day. The effect of mothers' smoking was greater than that of fathers', especially at high levels of consumption. After adjustment for known exposures geometric mean cotinine concentrations rose from 0·52 ng/ml in social class I to 1·36 ng/ml in social class V ($P<0·0001$); and were doubled in high mortality towns compared with the low mortality towns ($P=0·002$). In children with no identified exposure similar trends by social class and town were observed and the cotinine concentrations correlated with the prevalence of parental smoking, both between towns ($r=0·69$, $P=0·02$) and between schools within towns ($r=0·50$, $P<0·001$).**

***Conclusions*—Mothers' smoking is more important that fathers' despite the lower levels of smoking by mothers. Children not exposed at home had low cotinine concentration, the level depending on the prevalence of smoking in the community.**

Introduction

In the past decade many studies have investigated the health effects of passive exposure to tobacco smoke in non-smoking children.[1] Most studies have relied on questionnaire measures of parental smoking as the indicator of exposure. Such measures ignore exposure by people from outside the household, the extent to which parents smoke in the presence of the child, and other modifying factors such as the ventilation of the room. These studies may therefore have underestimated the real effect of passive smoking in children. Biochemical measures allow recent exposure to be estimated directly.

Cotinine, a metabolite of nicotine, is the best biochemical marker for quantifying passive exposure to smoke.[2] It is specific to tobacco, has a half life of about 20 hours, and can be detected at low concentrations by gas-liquid chromatography.[3] Salivary concentrations are in approximate equilibrium with those in the blood[4] and provide a non-invasive way of measuring passive smoke exposure. We present an analysis of the relation between cotinine concentration and questionnaire data in children aged 5-7 from 10 towns in England and Wales. We examine the importance of parental exposure as opposed to other sources; exposure among children from non-smoking households; and the social and geographical

pattern of passive exposure to tobacco smoke in children.

Subjects and methods

The study was carried out in 10 towns in England and Wales—five with high adult cardiovascular mortality (Wigan, Burnley, Rochdale, Port Talbot, and Rhondda) and five with low mortality (Esher, Leatherhead, Chelmsford, Bath, and Tunbridge Wells). The selection of these towns has been described in detail.[5] Because of the strong geographical association between mortality from cardiovascular disease and that from respiratory disease, this resulted in five towns with high mortality from respiratory diseases and five with low mortality.[6] Within each town we selected a sample of 10 primary schools stratified by religious denomination and, in the case of county primary schools, by size and location. Any school unable to take part was replaced by the school that matched most closely in denomination, size, and location. Within each school two classes of children aged between 5 and 8 were randomly selected to provide a sample of 50-60 children who were invited to participate. The validation of the sampling method has been described.[7] We restricted analyses to children of European origin.

Between January and July 1990 a team of four trained nurses, working in pairs, visited each town. Towns in high and low mortality areas were examined alternately. The 10 schools in each town were visited over five days, each pair of nurses visiting one school for a whole day. Each nurse made about one quarter of all measurements in each town.

Subjects were asked to collect saliva in the mouth and to blow it through a plastic straw into a plastic container. Samples were frozen within eight hours of collection for assay by gas-liquid chromatography, which can detect cotinine concentrations as low as 0·1 ng/ml.[3]

A self-completion questionnaire was sent to the parents of all participants on the day of the examination. Social class was determined for both parents on the basis of present or most recent occupation, according to the Registrar General's classification. Analyses in this paper refer to the head of household (male in 93%) as defined by the Office of Population Censuses and Surveys.[8] Those few households that could not be assigned a social class were treated as a separate group; they were mostly single parent families in which the mother had never worked regularly. The parents were asked about their current smoking habits (number of cigarettes a day plus pipe and cigar smoking for fathers), whether anyone else in the household smoked cigarettes, and whether the child was looked after for two hours a week or more by anyone from outside the household who smoked cigarettes.

STATISTICAL METHODS

Cross tabulations and multiple regressions were done by the FREQ and GLM procedures in SAS.[9] The logarithm of the cotinine concentration was used in quantitative analyses of salivary cotinine. For the purpose of logarithmic transformation, undetectable concentations were coded as 0·05 ng/ml. Log cotinine concentrations were regressed on questionnaire measures of exposure to estimate the relative importance of the different sources. Adjusted geometric mean cotinine concentrations by social class were obtained by regressing log cotinine on social class (seven categories) as well as mothers' smoking habit (six categories: none, 1-5, 6-10, 11-15, 16-20, ≥21 cigarettes a day), fathers' smoking habit (eight categories: as for mother plus cigars, pipe), whether another person in the household smoked, and whether the child was looked after for more than two hours a week by someone from outside the household who smoked. In addition, we included a dummy variable taking the value 1 if both parents smoked to allow for the subadditive effect of mothers' and fathers' smoking habit on log cotinine. Adjusted geometric means for other factors were obtained in a similar way.

Results

Of 5213 children invited to participate, 4283 (82%) were screened. A total of 4043 were of European origin. Among these 4043 children, both salivary cotinine and complete questionnaire data were available for 2727 (67%), cotinine data only for 903 (22%); questionnaire data only for 320 (8%); and neither cotinine nor questionnaire data for 93 (2%). Social class was missing for six of the 2727 children with complete exposure data, leaving 2721 children for whom all information was available.

The distribution of salivary cotinine concentration was skewed (fig 1); 217 children (6·0%) had non-detectable concentrations, the median was 1·0 ng/ml, and 41 had values above 14·7 ng/ml. The value 14·7 ng/ml is a suggested cut off for active smoking in adults.[10] However, we believe that most of these values were due to passive exposure since only one of the 41 values was too high (56·3 ng/ml) to be consistent with passive exposure and the next highest was 34·1 ng/ml; most of these children were girls (27, 65%) and below the average age of study subjects (6·1 *v* 6·4 years), characteristics which are less likely to be associated with active smoking; and all but one of the 41 were exposed to tobacco smoke in the home.

Salivary cotinine concentration was strongly related to the number of smokers to whom the child was usually exposed, the geometric mean cotinine increasing from 0·29 ng/ml in children with no identified source of exposure to 1·36 ng/ml in children exposed to one source and 3·97 ng/ml in children exposed to two or more sources. Undetectable concentrations were rare among those exposed to one or more smokers (18/1465, 1·2%), while only one of the 1262 children not exposed to smokers had a value above 14·7 ng/ml and only seven had values above 5 ng/ml. Nevertheless, 1107 (88%) of children in the non-exposed group had detectable cotinine concentrations, presumably representing exposure from other sources. The

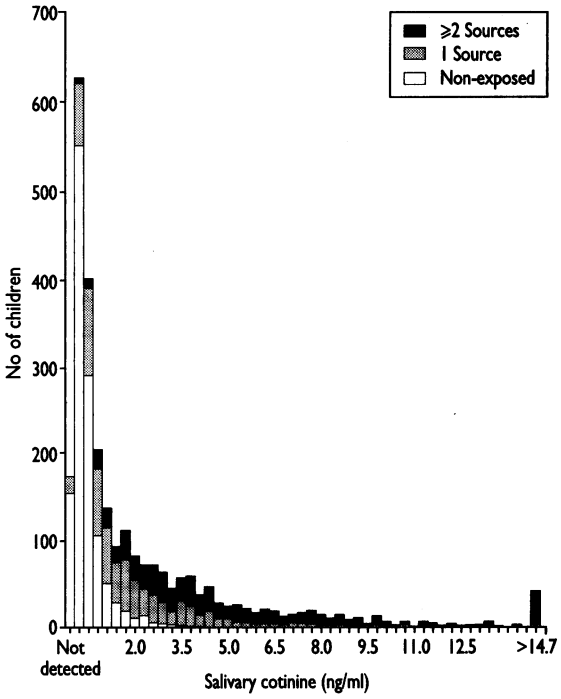

FIG 1—*Distribution of cotinine by number of sources of exposure*

903 children for whom questionnaire data were missing had a geometric mean cotinine concentration of 1·42 ng/ml, which is higher than the mean of 0·86 ng/ml for children with complete questionnaire data and similar to that of children exposed to one source.

SOURCES OF EXPOSURE

Fifty three per cent (1610/3040) of children were exposed to at least one smoker (table I). Parents were the commonest source of exposure; 10·8% (329/3040) of children had only a mother who smoked, 19·0% (576/3040) only a father, and 19·7% (598/3040) had both a mother and father who smoked. Another household member smoked in 3·2% (97/3040) of households; in 73% (71) of such households a parent also smoked. Only 8·7% (245/3040) of children were looked after by someone from outside the home who smoked, and 66% (162) of these had a smoking parent. Cotinine concentrations were available for 90% of children with complete questionnaire data, and this percentage did not vary with source of exposure. Further analyses are restricted to the 2721 children for whom complete data were available.

Geometric mean cotinine concentration varied greatly with source of exposure from 0·29 ng/ml in children with no identified source of exposure to 4·05 ng/ml when both parents smoked, a 13·7 fold increase (table I). In the 20 children in whom both parents smoked more than 20 cigarettes a day the geometric mean cotinine was 9·03 ng/ml (95% confidence interval 6·73 to 12·1). In addition to the effects of parental smoking, the presence of others in the household smoking multiplied the cotinine concentration by 1·93, and a carer from outside the household who smoked increased cotinine concentration by 1·62. Among children not exposed to any of these sources cotinine concentrations were unrelated to past smoking habit of the parents (geometric mean 0·29 ng/ml in children of never smokers, 0·28 ng/ml in children with one parent who used to smoke).

There were clear dose response relations with amount smoked by both parents (fig 2). Though mothers were less likely to smoke than fathers (see above), the effect on children's cotinine concentration when they did was greater. The difference between the effect of mothers' and fathers' smoking increased with the number of cigarettes smoked. The difference was small below 10 cigarettes but much greater at 20 or more cigarettes a day. Pipe and cigar smoking by the father was roughly equivalent to exposure to five cigarettes a day from one parent (fig 2).

CONTRIBUTION TO POPULATION BURDEN OF COTININE

A measure of the population burden of passive exposure to tobacco smoke is given by the sum of cotinine values over all children in this study. For a given group of children it is then possible to calculate the percentage of the total population burden found in that group by calculating the sum of their cotinine values and expressing it as a percentage of the total.

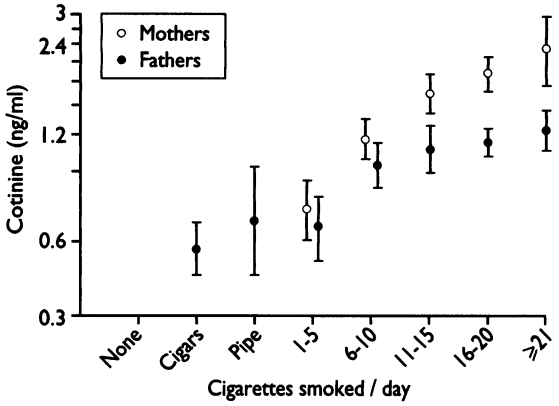

FIG 2—*Geometric mean cotinine concentration (±95% confidence interval) by parental smoking habits. The error bars represent 95% confidence intervals. Geometric means for each parent have been adjusted for effects of smoking level of the other parent and for presence of others in the household or an outside carer who smoked.*

The percentage population burden of cotinine for five mutually exclusive groups was as follows: both parents smoked (irrespective of whether another household member or outside carer smoked) 51%; only the mother smoked (irrespective of whether another household member or outside carer smoked) 19%; only the father smoked (irrespective of whether another household member or outside carer smoked) 17%; children exposed to another household member or a regular carer who smoked (but neither parent smoked) 3%; non-exposed children 11%. An indication of the potential effect of missing questionnaire data can be obtained by including the 903 children with cotinine values but no questionnaire data as though they were all exposed; the rate for the non-exposed children is then reduced to 7%.

SOCIO-DEMOGRAPHIC FACTORS

Table II presents the association between cotinine and sociodemographic factors. These confounding variables may be viewed either as markers of exposure or as modifiers of exposure. There were small but significant age and sex differences, with cotinine concentrations being higher in younger children and in girls. While the sex effect was present in non-exposed children the age effect was not. There were significant relations with day of the week on which screening occurred. After adjustment for differences in exposure, the highest cotinine concentrations were seen on Mondays.

Social class effects were strong. Overall, cotinine concentrations were eight to nine times greater in social classes IV and V compared with social class I. The differences were much reduced after adjustment for identified sources of exposure, but remained strongly significant. Perhaps most important, similar social class effects were seen in non-exposed children, with roughly a twofold difference in cotinine concentration between social class I and V. There were large differences between towns in cotinine concentrations,

TABLE I—*Sources of exposure and cotinine concentration of children aged 5-7*

Sources	No of children with questionnaire data (n=3040§)	No of children who also had cotinine measured (n=2721)	Geometric mean cotinine (ng/ml) (95% confidence interval)	Multiplicative effect compared with no exposure (95% confidence interval)
None	1430	1260	0·29 (0·28 to 0·31)	1
Mother only*	329	293	2·20 (1·95 to 2·48)	7·46 (6·54 to 8·52)
Father only*	576	521	1·17 (1·07 to 1·28)	3·98 (3·58 to 4·42)
Mother and father*	598	553	4·05 (3·71 to 4·42)	13·74 (12·40 to 15·23)
Other in household†	97	91	0·57 (0·46 to 0·71)	1·93 (1·55 to 2·40)
Outside carer‡	245	226	0·48 (0·41 to 0·55)	1·62 (1·40 to 1·87)

*Adjusted for the effect of others in the household and outside carer smoking.
†Adjusted for the effect of parental smoking and outside carer smoking.
‡Adjusted for the effect of parental smoking and others in household smoking.
§7 children with missing social class excluded.

TABLE II—*Relationship of cotinine to sociodemographic factors*

Variable	All children					Non-exposed children			
	No of children	Unadjusted geometric mean	Adjusted* geometric mean	*t* Test for trend	P value	No of children	Unadjusted geometric mean	*t* Test for trend	P value
Sex:				2·5	0·01			2·5	0·01
Boys	1377	0·79	0·82			650	0·27		
Girls	1344	0·94	0·91			610	0·31		
Age (years):				3·0	0·003			0·5	0·63
5-	241	1·03	1·00			105	0·31		
5·5-	562	0·90	0·92			273	0·27		
6·0-	594	0·88	0·83			267	0·31		
6·5-	677	0·80	0·84			327	0·27		
7·0-7·9	647	0·83	0·82			288	0·29		
Day of week:				7·0	0·0001			6·0	<0·0001
Monday	610	0·91	1·05			323	0·37		
Tuesday	551	1·15	0·99			222	0·35		
Wednesday	582	0·77	0·78			268	0·25		
Thursday	464	0·75	0·72			208	0·21		
Friday	514	0·77	0·77			239	0·25		
Social class:				11·5†	0·0001			8·4†	<0·0001
I	202	0·29	0·52			145	0·19		
II	790	0·45	0·66			499	0·24		
IIIN-M	262	0·82	0·91			120	0·30		
IIIM	991	1·29	1·01			368	0·35		
IV	317	1·51	1·10			94	0·42		
V	115	2·50	1·31			23	0·53		
Other	44	2·69	1·36			11	0·53		
Town:				$F_{9,2696}=22·6$‡	<0·0001			$F_{9,1250}=11·1$‡	<0·0001
Tunbridge Wells	344	0·54	0·57			161	0·19		
Bath	267	0·54	0·66			147	0·24		
Leatherhead	247	0·55	0·78			153	0·28		
Esher	250	0·59	0·72			135	0·21		
Chelmsford	333	0·69	0·78			175	0·25		
Burnley	276	1·16	0·93			103	0·39		
Port Talbot	260	1·36	1·22			109	0·44		
Rochdale	143	1·27	0·89			50	0·29		
Rhondda	258	1·45	1·18			98	0·48		
Wigan	343	1·46	1·25			131	0·34		

*Adjusted for mother's smoking habit, father's smoking habit, and smoking by another household member and a carer—see statistical methods.
†Other omitted from trend test.
‡*F* test for heterogeneity.

and these were only partly explained by adjusting for known sources of exposure (table II). In particular, cotinine concentrations in the high mortality towns in south Wales and northern England were roughly double those in the low mortality towns in southern England (P=0·002 for all children, based on comparing differences in cotinine between the two groups of towns to the variation between towns within groups). Similar effects were seen in non-exposed children (P=0·002). Adjustment of the town means for social class and other variables in table II, as well as for sources of exposure, had little effect on either the town means or the P values.

TOWN AND SCHOOL LEVEL ANALYSES IN NON-EXPOSED CHILDREN

If tobacco smoke is the only source of cotinine then measured concentrations among non-exposed children must be due either to visitors to the home or to exposure outside the home. Though we did not directly ask about these, we had indirect measures of exposure in the community at town and school level from the prevalence of smoking by parents in our samples. We therefore calculated the percentage of mothers who smoked for each town and for each school. At a town level, the correlation between this measure of community exposure and the geometric mean cotinine in non-exposed children was 0·69 (P=0·02).

School level analyses have much greater power than town level analyses because of the larger number of schools studied. To ensure that our between school analyses were independent of any between town differences, we calculated the cotinine concentrations in non-exposed children for each school as a percentage of the town mean and correlated this with the percentage of mothers who smoked for children at that school (fig 3). The correlation coefficient was 0·5 (P<0·001), providing clear evidence of the influence of community levels of smoking within towns. Other measures of community exposure incorporating fathers' smoking habits or number of cigarettes a day did not alter the conclusions.

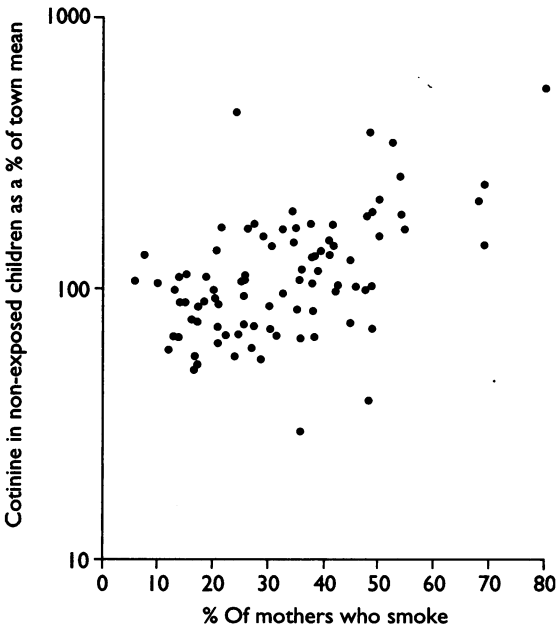

FIG 3—*Cotinine concentration in non-exposed children at each school (expressed as a percentage of the town mean) in relation to a percentage of all children whose mothers smoke in each school*

Discussion

Our data confirm that parental smoking is the most important source of passive exposure to smoke in young children and show a clear dose response with number of cigarettes smoked a day. While mothers were less likely to smoke than fathers, the effect on cotinine concentrations when they did so was greater, presumably because they spend more time with the children. The difference in effect was small at low levels of smoking, but pronounced at higher levels. One interpretation is that fathers who smoke heavily are less likely to do so in the presence of the child than mothers who smoke heavily. Overall, maternal smoking contributed more to the children's burden of cotinine than did paternal smoking. Other people

smoking in the household and being looked after by someone from outside the household who smoked also made small contributions to exposure. However, such sources of exposure were relatively uncommon and when present were less important than parental smoking.

In using cotinine as a measure of passive tobacco smoke exposure we have assumed that the effect on health is similar no matter what the source. Thus exposure to pipe and cigar smoke is equivalent on average to exposure to a parent smoking 1-5 cigarettes a day. The rarity of pipe and cigar smoking has meant that no study has been able to examine the health effects and exclusion of the children of pipe and cigar smokers does not alter our conclusions. A potentially more important issue is that nicotine is not entirely specific to tobacco. It is also found in small amounts in peppers, aubergines, and potato skins.[11] We have previously argued that these are unlikely to greatly influence cotinine concentration.[12] The uniformly low concentrations among our non-exposed children suggest that either the higher concentrations seen in the exposed children are due to smoking or the dietary factors are almost entirely confounded with smoke exposure in the home, which seems implausible. Even among our non-exposed children the concentrations of cotinine correlate with community smoking habits, which suggests that any other sources of cotinine make only a very small contribution.

Overall, the cotinine concentrations in our study were slightly higher than in a similar study of 7 year old children in Edinburgh.[12] In part this can be explained by the age of the cohort. Even within the narrow age range of this study, cotinine concentration fell with age, suggesting that as children become more independent of their parents, exposure declines. We found no evidence that differences in body size were related to cotinine concentration. In older children, active smoking and exposure by friends smoking become more important.[13] The marginally higher concentrations in girls than in boys were also observed in the Edinburgh study[12] and may be due to girls spending more time with their mothers. It seems unlikely that the effect is a metabolic one since among non-smoking adolescents boys have been found to have higher cotinine concentrations, in keeping with their higher reported exposure.[13] The higher concentrations seen on Mondays were also reported in the Edinburgh study[12] and presumably reflect the greater amount of time with parents at the weekend. Since cotinine has a half life of about 20 hours,[3] exposure during the weekend would significantly affect concentrations measured on a Monday.

SOCIAL CLASS AND GEOGRAPHICAL EFFECTS

The social class and geographical differences in cotinine concentrations emphasise the variation in passive exposure to tobacco smoke among children from different backgrounds. That these differences were much reduced, but not removed, by adjustment for known sources of exposure shows the value of measuring cotinine directly. The towns in northern England and in south Wales were selected because they had particularly high cardiovascular mortality in adults. They are therefore unlikely to be typical and are likely to have higher adult smoking rates than the average for their region. Equally, the towns in southern England are likely to have lower smoking rates than the average.

Though the identified sources of exposure were the most important determinants of variation in cotinine concentrations, other sources and modifying factors clearly existed. Eighty eight per cent of children not exposed at home and not looked after by a smoker had cotinine detected in their saliva. Cotinine concentration in non-exposed children was related to both social class and town of residence and was presumably attributable to sources we did not inquire about. This is supported by the finding that the cotinine concentrations in non-exposed children were directly related to the community level of smoking (fig 3). An alternative explanation for these findings might be that our questionnaire did not identify some parents who smoked. This could be due to misreporting or because some parents were occasional rather than regular smokers. In fact both explanations seem unlikely since the cotinine concentrations in non-exposed children were well below those expected from even a single source and were unrelated to past smoking habits of parents.

Public health implications

- Most studies of passive smoking in childhood have relied on questionnaire data and may have underestimated exposure
- In this study 53% of children were exposed to cigarette smoke in the home or by an outside carer
- Parental smoking was the most important determinant of exposure in children aged 5-7 years. Mothers' smoking was more important than fathers' because of greater responsibility for child care
- Children exposed to none of the above sources had evidence of low level exposure which correlated with the prevalence of smoking in the community where the child lived
- Passive smoking should be viewed as a community exposure rather than simply an aspect of family lifestyle

HEALTH EFFECTS

The importance of the levels of exposure reported depends on the established and potential health consequences. They also need to be placed in the context of the cotinine concentrations seen in active smokers. The average cotinine concentration in adult cigarette smokers is around 300 ng/ml.[14] The levels observed in this study in children exposed at home were about 100 times lower. Such concentrations have been associated with small decreases in lung function and increases in respiratory infections and glue ear.[15-17] In the longer term such concentrations may be associated with raised risk of lung cancer.[2] The average concentrations in non-exposed children were another order of magnitude lower still, at 0·29 ng/ml. Nevertheless, 7-11% of the population burden of cotinine was among these children because of the large number of children.

The public health importance of low level exposure depends on the relation of health outcomes to exposure. If health effects are linearly related to cotinine concentration, a low level of exposure (for example, 1 ng/ml) in 10 children is equivalent to one child being exposed at 10 ng/ml. Such an analysis may be misleading if health effects occur only above a certain cut off level of exposure. In studies reporting short term effects on respiratory symptoms and function and glue ear the effects have been related in a linear way to cotinine or to log cotinine,[15-17] implying that the gradient of the dose response relation is at least as great at lower doses as at higher exposure; no study has been large enough to directly assess the health effects of low level exposure. Though at an individual level the health risks seem likely to be negligible, effects may be seen at a community level since children not exposed at

home have roughly double the exposure in high smoking communities than in low smoking ones.

In conclusion, our data emphasise the value of measuring cotinine in the saliva as a simple non-invasive marker of passive exposure to tobacco smoke. Maternal smoking had a greater effect than paternal smoking on cotinine concentration despite its lower prevalence. However, 7-11% of the population burden of cotinine was in children not exposed to any of the sources we asked about. The correlation between cotinine concentrations in such children and the prevalence of smoking in the community suggests that passive smoking should be viewed as a community exposure rather than simply as an aspect of family lifestyle.

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Post-traumatic stress disorder in children after television programmes

D Simons, W R Silveira

Gulson Hospital, Coventry CV1 2HR
D Simons, *registrar in child and adolescent psychiatry*
W R Silveira, *consultant child psychiatrist*

Correspondence to: Dr Silveira.

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Post-traumatic stress disorder in children is now well documented,[1] although as recently as 1985 Garmezy and Rutter argued against the need for a diagnostic category, particularly as amnesia, psychic numbing, and intrusive flashbacks had not been reported in child survivors of disasters.[2]

On Hallowe'en (31 October) 1992 a programme with the title *Ghostwatch* was shown on television. Four months later two 10 year old boys were referred separately by their general practitioners to the child psychiatry unit at our hospital. Post-traumatic stress disorder was diagnosed, based on the criteria in the *International Classification of Diseases,* tenth revision (ICD-10) and *Diagnostic and Statistical Manual of Mental Disorders,* third edition, revised (DSM-III-R). We report here these two cases.

Case histories

CASE 1

This boy had been frightened by *Ghostwatch* and had refused to watch the ending. He subsequently expressed fear of ghosts, witches, and the dark, constantly talking about them and seeking reassurance. He suffered panic attacks, refused to go upstairs alone, and slept with the bedroom light on. He had nightmares and daytime flashbacks and banged his head to remove thoughts of ghosts. He became increasingly clingy and was reluctant to go to school or to allow his mother to go out without him.

His parents had separated when he was 4 years old, and he had a close relationship with his mother. His early development had been normal, although he was described as a sensitive child and a worrier. He was admitted to the hospital's child and family unit as his mother could no longer cope with his behaviour at home. A behavioural programme was established: he began to keep a diary of flashbacks; his mother was helped to regain control in their relationship; and both were discouraged from discussing ghosts and his fears. On discharge eight weeks later he was happier and confident and free of symptoms.

CASE 2

Immediately after watching *Ghostwatch* this child, also described as a worrier, had complained of being frightened. He had felt sick, and cried easily, and refused to go into his bedroom, complaining of someone watching him there. He was consequently allowd to sleep in his parents' room, where he talked excessively about his fears; his parents were drawn into a discussion and had to reassure him repeatedly throughout the night.

He was seen in the outpatient clinic. A behavioural programme was established, whereby he was encouraged to sleep in his own room with brief comforting when afraid and to ignore any discussion about the programme. After three appointments at weekly intervals, with telephone conversations in between, the situation had improved: he was sleeping in his own room and the whole family seemed more relaxed.

Comment

Post-traumatic stress disorder was diagnosed in our two patients on the basis of the criteria in the ICD-10 and DSM-III-R. The symptoms also corresponded to those described by Yule and Edwin[3]: sleep disturbances with problems settling down to sleep; waking at night; nightmares; fear of the dark and of sleeping alone; difficulties in concentrating; impaired memory; persistent intrusive thoughts and images of the traumatic event; raised levels of anxiety; panic attacks; separation anxiety and clinging behaviour; and depressed mood and irritability. The trauma in our two