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# Background Paper

## **Children's exposure to passive smoking: survey methodology and monitoring trends**

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## **Introduction**

Identification of the main sources of exposure and quantification of the dose of smoke inhaled are fundamental to the study of passive smoking effects in children. Two main measurement approaches have been adopted in surveys: questionnaire items inquiring about smoking by parents and others in the child's environment; and biochemical markers of inhaled smoke, particularly saliva and urinary cotinine concentrations. Neither questionnaires nor intake markers provide a complete solution. Questionnaires have been widely used in epidemiological studies of ETS effects, and have yielded much useful data. Simple indicators such as reported parental smoking status have proved highly predictive of adverse health outcomes. But the limitations of question-based measures of ETS exposure are widely acknowledged. Commentators have noted that they have generally not been standardised, may have limited validity and reliability, and cannot comprehensively describe total ETS exposure, exposure to individual ETS components, nor doses of biologically relevant agents at target sites (1). Cotinine levels, by contrast, give an integrated measure of the total amount of nicotine absorbed from all sources of exposure over the previous two or three days, and can be regarded as providing a 'gold standard' for assessing questionnaire validity (2). While strictly a measure of one particular smoke constituent, cotinine is valuable as a surrogate marker for the multiplicity of other smoke components, some of which may be of more concern for adverse health effects than nicotine itself. Cotinine may not give a precise quantitative indication of dose of other smoke components, but in between-individual comparisons it will identify those who are relatively more or relatively less exposed to all smoke constituents.

Like questionnaire-based methods, cotinine has obvious limitations. It is expensive and requires sophisticated laboratory assay facilities found only in a few centres around the world, ruling it out for routine application in many countries. It can only index current exposure and will therefore need supplementing in order to address lifetime issues such as mother's active and passive smoking in pregnancy and the child's cumulative exposure over previous months and years. It is also blind to the sources of current exposure, and cannot distinguish the relative contributions to measured concentrations of smoking by different individuals in different settings. From a public health perspective of planning interventions to reduce children's burden of exposure to ETS, as well as knowing the absolute levels of exposure it is vital to identify the main sources so that appropriate reduction strategies can be devised and tested.

From this, it is evident that questionnaires and intake markers are complementary rather than alternative approaches. Each can help to address some of the limitations of the other. Cotinine measures can point to which of the many potential questionnaire indicators of current exposure contribute most useful information, and which can be safely discarded. This is essential for development of a set of items that is sufficiently brief for widespread application in general population surveys but still informative. Question-based approaches add information about the past as well as the present, permit the assessment of potential confounders, and aid in the interpretation of observed differences in cotinine levels. In an ideal world, all studies of ETS effects on children would include both questionnaire and intake measures. This brief review aims to provide a perspective on survey approaches to ETS exposure measurement and will draw on intake data, where available, to validate and inform questionnaire-based methods.

### **Potential questionnaire indicators of the main household determinants of children's exposure to ETS**

Different studies have employed a wide variety of questionnaire items to index the main determinants of children's exposure to ETS. Some of these are listed in Table 1. The most commonly used are mother's and father's cigarette smoking status, supplemented in many cases by their reported cigarette consumption as a potential dose-response indicator. Other studies (e.g. in China, where few women smoke) have used total daily cigarette consumption

by family members (3) (4); or the total number of cigarettes per day smoked in the house (5). Household smoking has been measured as a yes/no dichotomy (6) (7) or as numbers of smokers (8). Although family and household structures vary in different cultures and parts of the world, it is probably not unreasonable to suggest that these various approaches, all of which have shown significant relationships to outcome variables, largely serve to index parental smoking. Whether direct questions about mother's and father's smoking have been asked, or more general questions about the household may have been related to a desire to cover all smoking, rather than simply parental, occurring in the home, or in some instances to perceived issues of sensitivity/confidentiality and a desire to achieve truthful reporting. Numerous studies have examined the relationship of parental and household smoking to cotinine concentrations in children. A consistent finding has been that both mother's and father's smoking status predict the child's cotinine, but with smoking by mothers having a greater impact than that by fathers (9-11). This parallels findings from epidemiological studies where disease endpoints have likewise been more strongly associated with mothers' than with fathers' smoking (12). In view of the consistency of these observations, it would seem desirable wherever possible to ascertain mothers' and fathers' smoking separately, rather than rely simply on a global measure of household smoking.

### **Ascertaining parental cigarette consumption**

Is there much to be gained by asking about parental cigarette consumption in addition to their smoking status? The answer to this question may not be wholly straightforward. Epidemiological studies have often found dose-response relationships between disease endpoints and cigarette consumption, and cotinine concentrations in children increase in a linear fashion with increased parental cigarette consumption (see, for example, Fig 1, which uses data from the Health Survey for England, a representative survey of the population living in private households (13)). However, in the Health Survey data, mothers' and fathers' smoking status together accounted for 33 per cent of the variance in children's cotinine and adding in their reported cigarette consumption only increased the percentage of variance explained to 36 per cent. Thus although parental cigarette consumption added predictive power, the simpler yes/no smoking dichotomy was not markedly worse as a predictor. Whether or not items on parental cigarette consumption are included in surveys may depend mainly on practical considerations. Where the parents themselves are responding, as in household-based surveys, there would seem to be no good reason not to ask about their consumption. But the situation in school-based surveys, where children rather than parents are the respondents, is different. Children are able to report accurately whether or not their parents smoke, but they may have little idea of the level of their cigarette consumption. In this context there is probably little to be gained by asking children about parental consumption.

### **Parental smoking or smoking specifically in the child's presence?**

Is it worthwhile to ask about smoking specifically in the house, or in the presence of the child in order to refine the exposure indicators? It is evident a priori that children are going to be most affected by any cigarettes smoked in their presence, and that a considerable proportion of their cigarettes will be smoked by parents outside the home. Some studies have reported that cotinine concentrations are affected by the frequency with which parents smoke in the same room as the child (14), and that where parents reported that they never smoked in the presence of their children, or that they smoked only in restricted home areas and regularly opened windows to improve ventilation, cotinine concentrations in the child were substantially reduced (15). In the USA, the percentage of households with an adult smoker and children and in which smoking was allowed in some or all areas of the home ranged from 70% to 96% across different states (16). In a Norwegian study, 47 per cent of smoking families reported that they did not smoke indoors (17). Similarly, only 25 per cent of a sample of 1003 Finish children with smoking parents were reported to have been regularly exposed to smoke at home in the previous year (18). Intervention trials in which smoking parents of asthmatic children received behavioural counselling to reduce their exposure have

been reported to have successful outcomes (19, 20) and further trials of this kind are under way.

If these findings are replicated and a convincing body of evidence emerges that parents can smoke without exposing their children to smoke within the home, it may be necessary to rethink the inclusion in questionnaire-based surveys of items exploring smoking specifically in the child's presence. However, the most convincing evidence for such 'harm reduction' approaches will always come from studies which incorporate quantitative measures of smoke intake. Reliance on parental self-reports of strategies to limit their children's exposure invites socially desirable responses and risks serious bias in findings (21-23).

### **Smoking by household members other than parents, by visitors to the house and by child care providers**

A number of studies have included items asking about smoking by household members other than parents, with responses usually being scored as a yes/no dichotomy. Consistent evidence has emerged that the presence of such smokers increases children's cotinine concentrations, although the magnitude of this effect is small compared with that of parental smoking (9, 10, 24). Similarly, where children spend considerable amounts of time with carers who smoke, their cotinine concentrations are substantially raised (10, 24);. Peterson et al looked at a variety of potential sources of exposure: visitors to the home; baby-sitters; people outside the home; as well as mothers, fathers and other household residents (24). Only smoking by child carers outside the house added significantly to the effects of parental and other household residents' smoking in predicting cotinine concentrations. There would seem to be a firm basis for including in survey instruments items asking about smoking in the household by others than parents, and, for pre-schoolers, smoking by carers.

### **Other determinants of exposure**

Table 2 lists variables which have consistently been found to be associated with children's exposure to ETS as indicated by cotinine concentrations. Some of these observations are not particularly surprising and probably do not have significant implications for survey monitoring. Higher concentrations in winter than in summer (25, 26) no doubt reflect lower rates of room ventilation in colder months and more time spent inside, similarly higher concentrations in children on Mondays than other days of the week (7, 15) show the importance of increased time spent with parents at the weekend. Regional differences in exposure may be due to variation both in climate (6) and in overall population smoking prevalence (10).

Of special interest is the reliable observation that cotinine concentrations are higher in children from more disadvantaged backgrounds. This has emerged consistently in several studies, using a variety of indicators of socio-economic position. Cotinine concentrations have been reported to be related to size of dwelling (15, 27), crowding in the home (7, 14, 28), mothers' and fathers' educational level (18, 27, 28), occupational class (7, 10)), and to composite indices of disadvantage incorporating information on occupation, employment status, housing tenure, and access to a car. Figure 2, which uses data from the 1996 Health Survey for England (8) illustrates how the measured concentration of cotinine in children at each level of parental cigarette consumption increases in a graded fashion with increasing social disadvantage. This is an effect of considerable magnitude, which has important implications for studies of children's exposure to ETS.

Epidemiological studies of health effects of passive smoking relying on questionnaire measures of exposure have usually treated socio-economic status as a potential confounder and have controlled for it in examining the association between exposure and disease outcomes. While SES may indeed serve as a confounder in certain respects, it is also apparent that it serves as an indicator of real differences in exposure. Quantitative data on uptake of smoke components show that socio-economic position is an important determinant

not only of whether a child is exposed to other people's tobacco smoke, but also of the extent of that exposure.

Studies monitoring children's exposure to passive smoking certainly need to incorporate good questionnaire measures of the family's socio-economic position, but fully adequate assessment of the role of socio-economic factors in determining exposure will require inclusion of objective markers of smoke intake.

### **Suggested outline requirements for questionnaires to monitor children's exposure to passive smoking**

Monitoring the extent of children's exposure to passive smoking in order to inform estimates of the public health impact requires large surveys that are representative of the general population. Household-based surveys, in which an attempt is made to interview all the adults living in the household, provide a suitable means of reaching dependent children aged 0-15 years, since the overwhelming majority of children will be resident in private households. Examples of such surveys are the National Health Interview Survey (NHIS) (29) and the National Health and Nutrition Examination Survey (NHANES) (6) in the USA, the National Health Survey in Australia (30), and the General Household Survey (31) and the Health Survey for England (8, 13) in the UK. Some of these surveys already incorporate measures of cotinine (NHANES III; Health Survey for England; Scottish Health Survey), which greatly enhance the value of the questionnaire measures. Modules in these surveys cover areas such as: adults' smoking habits and cigarette consumption; household composition and socio-economic status; and number and age of dependent children. By linking parental responses to their children's records in such surveys it is possible to estimate the prevalence of children's exposure to adult smoking and its variation with socio-economic status, and by combining data across years to monitor changes over time as national smoking habits change. An example of how trends in children's exposure to passive smoking to ETS can be estimated is given below in the section on monitoring.

General household-based surveys are not designed to examine specific health impacts of passive smoking. Studies aimed at examining health outcomes require designs addressing concerns appropriate to the particular issue under study and careful elucidation of the range of potential confounders. The strength of population surveys is that they enable broad trends in exposure to be monitored. Assessment of the resulting public health impact will be informed by the more detailed studies of exposure-outcome associations.

The required items to assess adults' smoking habits are fairly brief. The precise wording (and with it the definition of what constitutes a current cigarette smoker) varies somewhat from country to country. The questions used to monitor current and ex-cigarette smoking status in the UK General Household Survey and Health Survey for England are shown in Table 3 as one example.

A second method of estimating children's exposure to ETS, at least in those who are school-aged, is through school-based surveys. As well as asking about the child's own smoking, these can inquire about smoking by mothers and fathers and other household residents. Table 4 gives the questions used in the UK's Office for National Statistics surveys of smoking in secondary schoolchildren (32). The child is asked only if parents smoke, not how many. These surveys have included measures of saliva cotinine since 1988, and the questionnaire responses have proved highly predictive of cotinine concentrations. Table 5 shows geometric mean saliva cotinine concentrations in children by their parents' smoking as reported by the children across successive surveys since 1988.

### **Monitoring trends in children's exposure to ETS**

The need for reliable estimates of the proportion of children at different ages exposed to ETS, and of the extent of their exposure, is readily evident. Such data are required in order to

assess the public health burden of disease caused by passive smoking in children and the resulting medical expenditures and economic costs (33, 34). Progress in reducing children's exposure to ETS is a significant marker of more general progress in reducing the prevalence and health impact of tobacco smoking (35). In some instances, specific targets for reducing children's exposure to ETS have been adopted as part of national health planning objectives. For example, in the USA one of the national health objectives for the year 2000 is to reduce to 20 percent or less the number of children aged under 7 exposed to passive smoking in the home (36).

In the light of the discussion earlier in this paper, the ideal method of monitoring children's exposure to ETS would be by a combination of questionnaire based surveys with quantitative biomarkers of actual smoke intake. Application of biomarkers would permit a judgement about whether self-reported parental rules about allowing smoking in the home and their use of 'harm reduction' strategies more generally is effective in reducing children's exposure. Indeed, simply from the perspective of estimating overall population exposure to ETS, without partitioning it into its various sources, cotinine concentrations by themselves in large and representative population samples, without any corresponding questionnaire measures, if repeated across the years would provide a valid means of tracking exposure. However, the reality of the current situation is that not only are such cotinine-based population surveys very much a rarity and an exception even in those countries where smoking control and surveillance is most developed, but even questionnaire-based tracking is patchy at best, and there are few if any published data on trends across years.

The proportion of children exposed to ETS has been estimated in a number of particular population groups and also in large surveys of the general population in some countries. Examples of the former are the studies by Greenberg (37) and by Chilmonczyk (25). Surveying a representative sample of neonates and their mothers in North Carolina, Greenberg found that 55% of infants lived in households where at least one smoker was present. Chilmonczyk and co-workers recruited their subjects through mother/infant pairs attending a well-child clinic in Maine and found that 41% of infants lived in a smoking household. Both of these studies were cross-sectional, and provide no evidence on how infants' exposure may be evolving over time. A further limitation to these studies is that they illustrate the situation in particular places, and may not be representative of the country more widely.

General population surveys address the latter of these problems, but again available data are cross-sectional only. In Australia, the National Health Survey, a household based survey of 54,000 individuals provided estimates for 1989-1990 of the proportions of children at different ages with smoking parents (30). A total of 56.6% of children aged under 15 lived in homes where neither parent smoked or in non-smoking one-parent homes. 27.1% had a smoking mother, 29.4% a smoking father, and in 13.2% of households both parents smoked. Thus a total of 43% lived in homes where at least one parent smoked. This figure is similar to recent estimates for Canada, where it has been reported that 47% of children are exposed to ETS in the home, whether by parents, visitors or baby-sitters (33).

In the USA, Pirkle et al (6), using data from NHANES III covering the period 1988 to 1991 estimated that 43 percent of US children aged 2 months to 11 years lived in a home with at least one smoker. More recently, the US Centers for Disease Control have published state-specific estimates of the proportion of children exposed to ETS in the home (16). This study estimated that approximately 21.9% of children and adolescents aged under 18 were exposed to ETS in homes in the USA in 1996. This figure is markedly lower than the estimate from NHANES and very different also from the Australian and Canadian figures.

The CDC estimates were based on a complex methodology, which involved combining information from two different surveys, the Behavioral Risk Factor Surveillance System (BRFSS) and the Current Population Survey (CPS). The BRFSS data were used to derive

estimates of adult smoking prevalence and the proportion of households with an adult smoker and any children. These figures were then combined with data from CPS on the percentage of households in which smoking was allowed in the home (defined as “smoking is allowed in some places or at some times” and “smoking is permitted anywhere”). An obvious reason for the CDC’s lower estimates of the proportion of children exposed to ETS is that they take parents’ self-reported rules about permitting smoking in the home at face value. In this study the percentage of households with an adult smoker and children and in which smoking was allowed in some or all areas of the home ranged from 70.6% (Washington) to 95.6% (District of Columbia). It is precisely to check on the appropriateness of making allowance for such household rules that biomarker-based surveys would have most value.

The United Kingdom has a strong tradition in monitoring smoking habits among both adults and children. The General Household Survey has employed essentially the same methodology to measure smoking habits since 1972, and school-based surveys have been conducted regularly since 1982. Neither of these sources has been exploited specifically to monitor trends in children’s exposure to ETS in published reports (31, 32), but both have the potential to be utilised for this purpose.

Table 6 shows estimates of children’s exposure in the home across selected years since 1973 using data from the GHS. The table gives (top) the proportion of children in different age groups with smoking mothers and fathers. The lower part of the table looks at these data in another way to estimate the percentage of children from homes where one, both or neither parent smoked. The estimates were derived by linking parents’ responses about their smoking habits (ascertained by the questions given in Table 3) with their children’s records in the same household. It can be seen that the percentage of children aged 0-15 with a smoking father has declined steeply, from 56.9% in 1973 to 32.1% in 1994. There has been a parallel decline in the percentage of children with a smoking mother, from 48.2% in 1973 to 30.3% in 1994. The table also explores alternative ways of estimating the percentage of children with smoking mothers and fathers. If, on average, adults with and without dependent children have similar smoking habits, then the percentage of smokers among all adults in the age range of those with dependent children (taken here to be 20-49) should provide a good approximation of the percentage of children with smoking parents. The figures in italics give cigarette smoking prevalence in all men and women aged 20-49 in the GHS. For women, the agreement with mothers’ smoking is particularly good, with an average difference of less than 1 percent. There is slightly less agreement for men, with a mean difference of just under 2 percent. The important implication of this correspondance between more and less direct methods of estimating the percentage of exposed children is that national surveys of smoking prevalence in adults can serve as proxies for smoking in households with children and can be used to get reasonably informative estimates of the percentage of children at risk. This is likely to be particularly helpful in those countries that have less developed survey approaches.

The figures for household smoking shown in the lower part of the table show a steep increase since 1973 in the percentage of children living in homes where neither parent smokes (or only one, non-smoking, parent is present), from 31.3% in 1973 to 58.6% in 1994. The figures for 1990 tally well with the 1989-90 figures from Australia (52.7% vs 56.6 from non-smoking homes; 33.0 vs. 30.2% with one smoking parent; and 14.2% vs 13.2% with both parents smokers).

Surveys of smoking among schoolchildren conducted in schools can serve as an alternate means of estimating parental smoking, and hence children’s exposure to ETS. Table 7 shows estimates for the percentage of children aged 11-15 in England with smoking fathers and mothers across the years 1988 to 1996. The data were gathered using the questions in Table 4, and the corresponding saliva cotinines are shown in Table 5. The reliable increases in measured cotinine concentrations across exposure categories confirm the value of the questionnaire data.

As with the General Household Survey, the schools' survey permits estimates of the percentage of children living in non-smoking homes, and the percentage with smoking mothers and fathers. Comparison with the percentage of adult smokers in the age range 25-49 from the GHS indicates a reasonably good degree of correspondence, with an average difference of about 2 percentage points for fathers' and 1 for mothers' smoking.

The percentage of children living in non-smoking homes as estimated from the schools' survey gives a slightly different picture to the GHS (shown in Table 6). In 1988 the schools' survey yielded a figure of 48.1% compared with 50.8% from the GHS; and in other years the figures were: 49.4% vs 52.7% in 1990, 51.7% vs 57.9% in 1992; and 56.3% vs 58.6% in 1994. Differences in the estimates from the two sources may be attributable to a variety of factors, including differences in the sampling frame (schools vs households) and differences in the children's age range. Nevertheless, the degree of agreement suggests that school-based surveys, like household surveys, can be used to derive reliable estimates of the percentage of children at risk from ETS exposure.

**Table 1**

**Potential questionnaire indicators of children's exposure to ETS**

1. Mother's and father's cigarette smoking status
2. Mother's and father's cigarette consumption
3. Household indicators:
  - Daily cigarette consumption by family members
  - Cigarettes per day smoked in the house
  - Household smoking (yes/no or number of smokers)
  - "Does anyone smoke cigarettes, cigars or pipes anywhere inside this house?"
  - "Have you or anyone else smoked regularly indoors during the past 12 months?"
  - "On the average, about how many days per week is there smoking anywhere inside this house?"
4. Smoking by child carer
5. Smoking by other household residents
6. Smoking by baby-sitters
7. Smoking by home visitors
8. Smoking by people the child visited away from home for more than 2 hours

**Table 2**

**Variables reported to bear an independent predictive relationship to cotinine concentrations in children**

Mothers' smoking and consumption  
Fathers' smoking and consumption  
Other smokers in the household  
Number of children in household  
Day of the week  
Season of the year  
Region  
Socio-economic factors

- Deprivation score
- Crowding
- Parental education
- Size of dwelling

**Table 3**

**Questions used in the General Household Survey to ascertain current and ex-cigarette smoking status**

Do you smoke cigarettes at all nowadays?

About how many cigarettes a day do you usually smoke at weekends?

About how many cigarettes a day do you usually smoke on weekdays?

Have you ever smoked cigarettes regularly?



**Table 4**

Does either your mother or father smoke? Please just tick the third box if they don't live at home with you.

	Smokes	Does not smoke	Not living at home with me
Your mother?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Your father?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Do any of your brothers and sisters who are living at home smoke?  
If you have no brothers or sisters just tick box four.

	Smokes	Does not smoke	Not living at home with me	No brother/sister
Your brother(s)?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Your sister(s)?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Is there anyone else living at home with you who smokes?

Yes	<input type="checkbox"/>
No	<input type="checkbox"/>

**Table 5**

**Geometric mean saliva cotinine concentrations (ng/ml) by parental smoking:  
ONS schools surveys 1988-1996, England**

	Neither parent smokes	<i>Base</i>	Father only smokes	<i>Base</i>	Mother only smokes	<i>Base</i>	Both parents smoke	<i>Base</i>
<b>1988</b>	0.43	504	1.20	234	1.94	161	3.29	212
<b>1990</b>	0.55	545	1.40	221	2.28	167	3.06	220
<b>1992</b>	0.33	989	1.02	336	2.61	290	3.69	305
<b>1994</b>	0.25	549	0.86	191	1.94	142	2.96	142
<b>1996</b>	0.33	469	1.00	165	1.78	183	2.84	149

**Table 6****Percentage of children aged 15 and under whose mothers and fathers smoke**

Child's age	Fathers' smoking				men	Mothers' smoking				women
	0-5	6-10	11-15	0-15		0-5	6-10	11-15	0-15	
1973	56.9	55.8	58.1	56.9		47.3	47.7	49.8	48.2	
<i>1973 age 20-49 prevalence</i>					55.2					48.7
1976	49.5	51.6	50.5	50.6		41.9	45.0	45.0	44.0	
<i>1976 age 20-49 prevalence</i>					49.0					44.3
1980	47.5	49.7	47.9	48.4		40.8	45.1	44.5	43.5	
<i>1980 age 20-49 prevalence</i>					45.8					42.7
1988	36.2	38.5	39.2	37.9		36.5	35.5	36.4	36.2	
<i>1988 age 20-49 prevalence</i>					37.0					35.1
1990	33.6	38.0	32.4	34.6		36.2	37.2	33.3	35.7	
<i>1990 age 20-49 prevalence</i>					35.5					34.2
1992	33.2	30.5	29.7	31.3		34.4	30.3	28.7	31.5	
<i>1992 age 20-49 prevalence</i>					33.9					32.1
1994	32.9	31.1	32.2	32.1		31.6	30.5	28.2	30.3	
<i>1994 age 20-49 prevalence</i>					33.3					30.1

	Parent(s) nonsmokers	One parent smokes	Both parents smoke
<b>1973</b>	31.3	38.0	30.6
<b>1976</b>	35.9	37.3	26.8
<b>1980</b>	38.7	34.5	26.8
<b>1988</b>	50.8	33.3	15.8
<b>1990</b>	52.7	33.0	14.2
<b>1992</b>	57.9	30.4	11.7
<b>1994</b>	58.6	30.6	10.6

% were derived by linking mothers' and fathers' self-reported smoking with the child's data in the same household.

The alternative simpler estimate of cigarette smoking prevalence in those aged 20-49 was made without taking into account whether or not respondents had children. Nevertheless, this is clearly a reasonable estimate of the % children exposed.

Source: General Household Survey

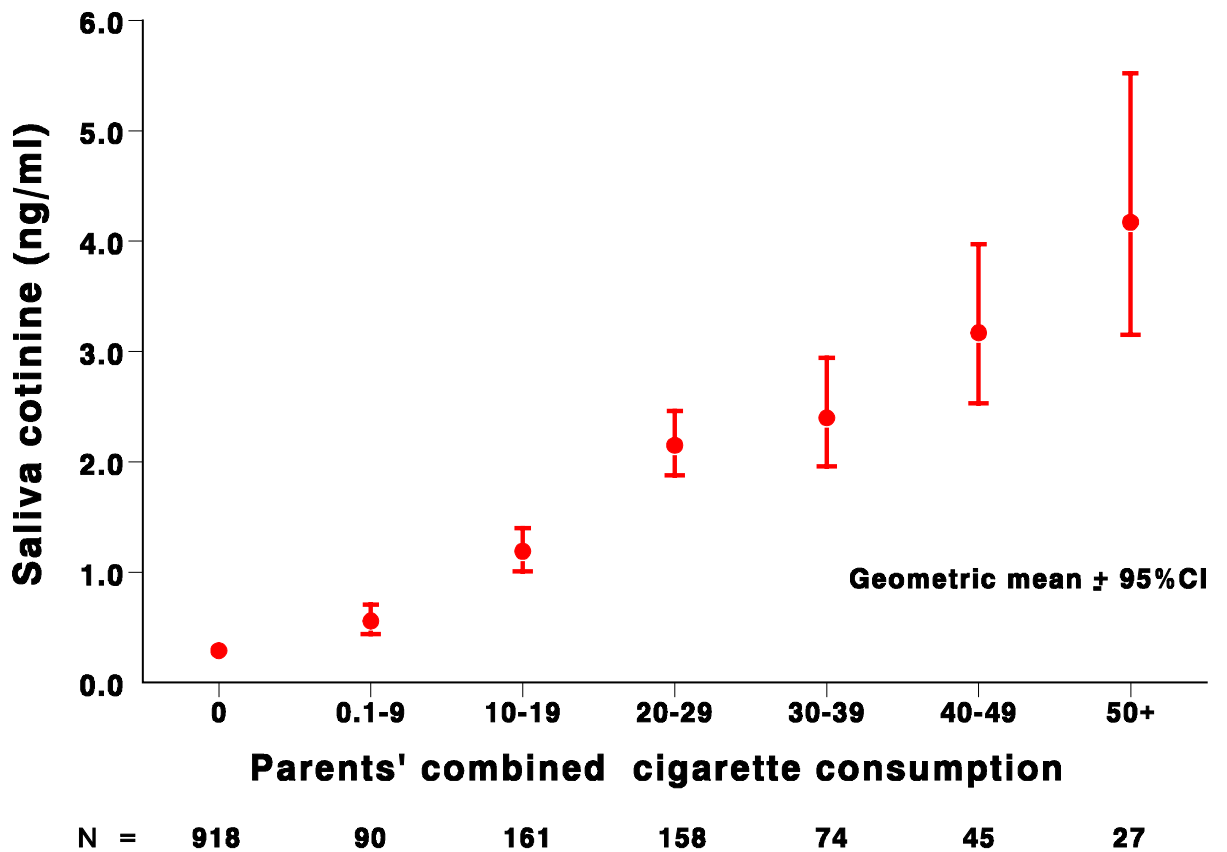
**Table 7**

**Smoking in parents of children aged 11-15: ONS surveys of secondary schoolchildren in England, Wales & Scotland**

	<b>1988</b>	<b>1990</b>	<b>1992</b>	<b>1993</b>	<b>1994</b>	<b>1996</b>
<b>Father smokes</b>	37.9	36.7	32.7	27.8	30.0	28.9
<i>% men 25-49 smoke *</i>	36.8	34.3	31.8		31.8	31.7
<b>Mother smokes</b>	32.9	32.9	32.2	26.0	27.5	30.9
<i>% women 25-49 smoke *</i>	34.7	32.6	30.1		28.5	30.4
<b>Both parents smoke</b>	18.9	18.9	16.7	12.6	13.8	13.6
<b>Non-smoking household</b>	48.1	49.4	51.7	58.8	56.3	53.9

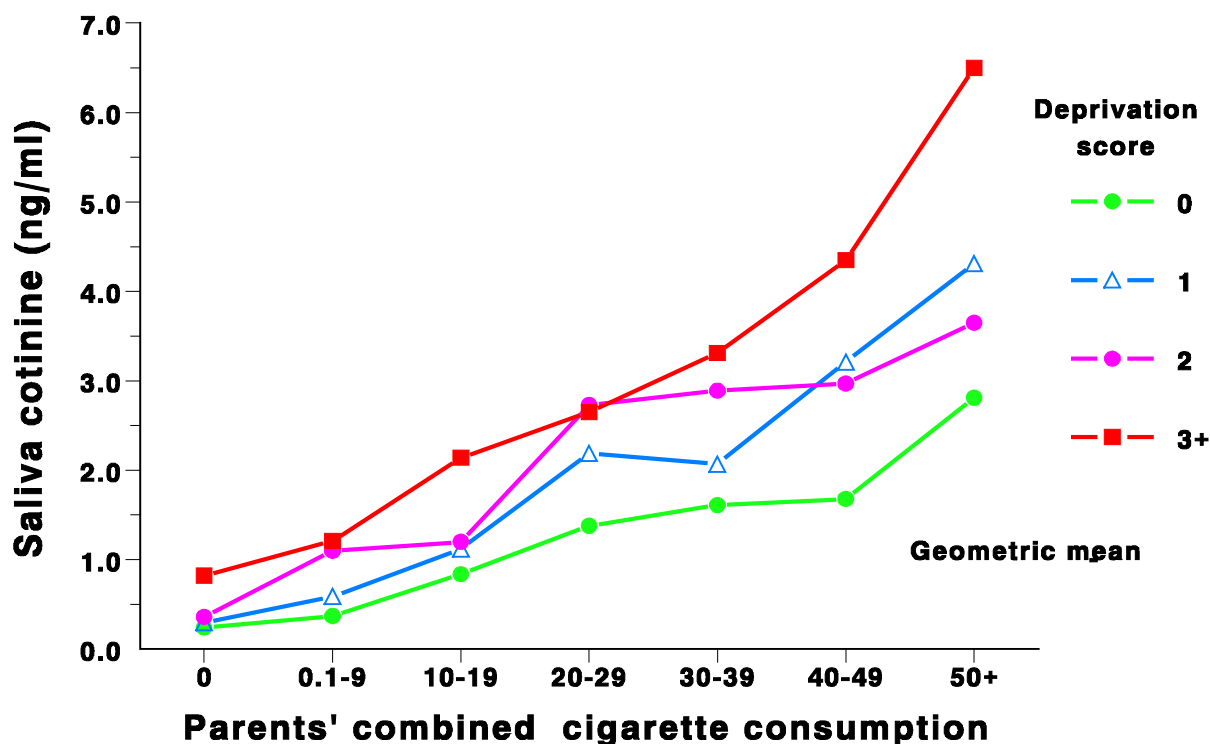
\* Data for cigarette smoking prevalence in adults aged 30-49 are taken from the General Household Survey

## CHILDREN'S COTININE BY PARENTS' CIGARETTE CONSUMPTION: HSE 1996



**Figure 1:** Geometric mean ( $\pm$  95% confidence intervals) saliva cotinine concentrations in children age 0-15 by their parents' combined cigarette consumption. Data from Health Survey for England, 1996 (8)

## CHILDREN'S COTININE BY PARENTS' SMOKING AND DEPRIVATION: HSE 1996



**Figure 2:** Geometric mean saliva cotinine concentrations by parental smoking and level of household disadvantage, Health Survey for England 1996 (8). At each level of parental smoking, children from more disadvantaged backgrounds had higher cotinine levels, indicating greater exposure to ETS.

Disadvantage was measured by an index on which children scored 1 for each of the following: manual occupation for head of household; rented housing tenure; no access

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