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Trends in and predictors of second-hand smoke exposure indexed by cotinine in children in England from 1996 to 2006

Michelle Sims[1], Susannah Tomkins[2], Ken Judge[1], Gordon Taylor[1], Martin J. Jarvis[3] & Anna Gilmore[1]

School for Health, University of Bath, UK,[1] Department of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, UK[2] and Department of Epidemiology and Public Health, University College London, UK[3]

ABSTRACT

Aims To explore trends in and predictors of second-hand smoke (SHS) exposure in children. To identify whether inequalities in SHS exposure are changing over time. **Design** Repeated cross-sectional study with data from eight annual surveys conducted over an 11-year period from 1996 to 2006. **Setting** England. **Participants** Nationally representative samples of children aged 4–15 years living in private households. **Measurements** Saliva cotinine (4–15-year-olds), current smoking status (8–15-year-olds), smoking status of parents and carers, smoking in the home, socio-demographic variables. **Findings** The most important predictors of SHS exposure were modifiable factors—whether people smoke in the house on most days, whether the parents smoke and whether the children are looked after by carers who smoke. Children from more deprived households were more exposed and this remained the case even after parental smoking status has been controlled for. Exposure over time has fallen markedly among children (59% decline over 11 years in geometric mean cotinine), with the most marked decline observed in the period immediately preceding smoke-free legislation. Declines in exposure have generally been greater in children most exposed at the outset. For example, in children whose parents both smoke, median cotinine declined annually by 0.115 ng/ml compared with 0.019 ng/ml where neither parent smokes ($P < 0.05$). **Conclusions** In the 11 years leading up to smoke-free legislation in England, the overall level of SHS exposure in children as well as absolute inequalities in exposure have been declining. Further efforts to encourage parents and carers to quit and to avoid smoking in the home would benefit child health.

Keywords Children, cotinine, inequalities, passive smoking, second-hand smoke, socio-economic status.

Correspondence to: Michelle Sims, School for Health, University of Bath, Bath BA2 7AY, UK. E-mail: m.sims@bath.ac.uk
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INTRODUCTION

Children have little control over their home environment. If their parents, other family members or carers smoke, children may be exposed involuntarily to second-hand smoke (SHS) and may be unable to remove themselves from this exposure [1]. Parental smoking is a major determinant of children's SHS exposure, with maternal smoking shown to have a greater impact on exposure than paternal smoking [2,3]. The health impacts of SHS exposure are now well documented and in children include sudden infant death, pneumonia, bronchitis and other respiratory symptoms, middle ear disease and asthma [4,5]. Moreover, biological markers suggest that young children develop higher internal exposures than adults when exposed to the same external smoke concentrations, due probably to their higher relative ventilation rates [6].

In England, children's exposure to SHS has been declining over time [4]. There is some uncertainty as to whether exposure has declined significantly among children living with parents who smoke, and analyses to date have focused upon children aged 11–15 years [4]. To our knowledge no work has yet examined trends in exposure in younger children, the extent to which trends differ by socio-economic status or ethnicity and thus whether

inequalities in exposure are changing; nor have the factors influencing exposure or smoking in the home been explored in detail using quantitative data.

This study examines these issues and will inform current debates around how best to protect young people from SHS following the implementation in July 2007 of legislation mandating smoke-free public places in England [7]. By furthering understanding of the predictors of and trends in SHS exposure in children it will also inform comprehensive evaluations of the impacts of smoke-free legislation (SFL) on children's exposure.

METHODS

The Health Survey for England

Data were taken from the Health Survey for England (HSE), an annual, nationally representative survey of individuals living in private households [8]. We took all available data on 4–15-year-old children sampled in the core surveys from 1996 to 2006 inclusive (excluding 1999, 2000 and 2004, when representative cotinine samples were not available). The included data were linked to selected data from their parental figures (defined as biological, adopted, step- or foster-parents) living in the same households.

Children who were active smokers (defined as those with a salivary cotinine over 12 ng/ml [9] or who self-report smoking at least one cigarette a week) were excluded from the analyses of SHS exposure. For parents, smokers were defined as those answering 'Yes' to the question 'Do you smoke cigarettes at all nowadays?'. Measures of exposure to SHS were based on responses to two questions: whether someone smokes inside the home on most days (available for all children) and whether children were looked after for more than 2 hours a week by someone who smokes while looking after them (asked for those aged 2–12 years only and labelled 'carer smoking'). Appendix S1 in Supplementary material gives further details of the HSE.

Statistical analysis

Linear regression analysis was performed to explore the predictors of SHS exposure as measured by salivary cotinine. The cotinine data were skewed strongly positively due to the large number of low and 0-values. To reduce the skewness and satisfy assumptions for linear regression, the natural log of cotinine was obtained by first reassigning a nominal value of 0.05, half the minimum detectable limit of 0.1 ng/ml [10], to raw values of 0.

Predictors of SHS exposure

Univariate regression. Univariate regression analysis was used to evaluate the relationships between predictors of interest and log cotinine levels. Predictor variables included age and gender of child, markers of household socio-economic status and measures of probable exposure. Markers of socio-economic status included social class and employment of the head of the household, education (defined as the highest educational qualification of either parent), degree of crowding (defined as the number of people per bedroom) and ethnicity of child. Social class of the head of household was measured using the British Registrar General's classification, which groups occupations into five categories: I (professional), II (managerial and technical), III (skilled non-manual and manual), IV (semi-skilled manual) and V (unskilled manual). Measures of probable exposure included parental smoking, whether someone smokes inside on most days and, for those aged 4–12 years, 'carer smoking'. In addition, a variable for year was included to investigate changes in cotinine levels over time. Year and age were included as continuous linear variables, as a linear trend was observed between these and the mean of the outcome variable, log cotinine. All other variables were included as binary or categorical variables.

We repeated the univariate regression analysis, adjusting for year and age (given the impacts of both on SHS exposure shown in the univariate analysis). We assessed the strength of each univariate predictor by computing the proportion of the total variation in the children's cotinine concentrations explained by each univariate model (R^2). For the age/year-adjusted model, part correlations were computed by first computing the proportion of variance explained in a model with age and year as explanatory variables, and then determining the additional variance (part R^2) that could be explained by adding the variable of interest to the model.

Multivariate regression. We then performed a multivariate regression analysis adjusting for year, age, gender, the various socio-economic markers and the effects of parent and carer smoking patterns to identify the most important predictors of SHS exposure. A backward selection procedure was used to select a subset of significant ($P < 0.05$) variables associated with mean log cotinine, adjusting simultaneously for other variables in the model.

As carer smoking was asked only in those aged up to 12 years, we performed the multivariate analysis twice, first on all children and then on children aged 4–12 years, with carer smoking included only in the latter. For both multivariate models we calculated the percentage variation in log cotinine explained by the model as a whole (R^2). All analyses were performed using R software version 2.8.0.

Changes over time. To assess trends in the degree of absolute inequalities in SHS exposure over time, a median

regression [11] (which allows analysis of positively skewed data without requiring a transformation) on salivary cotinine levels was performed. Age/year-adjusted univariate models were fitted, as described above, with an interaction term included between the determinant and year (e.g. to explore whether trends varied by social class, the interaction term year × social class was fitted).

Comparison of households that do and do not allow smoking

Finally, households that do and do not allow smoking inside on most days were compared using χ^2 tests.

RESULTS

Sample sizes and validity of smoking status

Of the 19 784 children aged 4–15 years interviewed across the eight surveys, 13 875 (70.1%) had a valid cotinine sample, although this proportion declined over time from 83.5% in 1996 to 58.3% in 2006 (Table 1). The samples of children interviewed and those with valid cotinine were similar in terms of gender, parental smoking status, social class and whether someone smokes inside their home on most days. There was, however, a slight under-representation of younger children in the group with valid cotinine samples in recent years (data not shown).

Using the cut-off point of 12 ng/ml, 5.1% (711/13 875) of children aged 4–15 years were defined as active smokers. In the subset of children aged 8–15 years, among whom both self-reported and cotinine-validated smoking data were available, 2.6% (239/9289) self-report as weekly smokers, compared with 6.5% (603/9289) identified as active smokers using cotinine (Table 1), suggesting that relying on self-report alone will underestimate youth smoking.

Predictors of second-hand smoke exposure

The univariate (unadjusted) analysis in 4–15-year-olds (Table 2) shows an important association between year and SHS exposure: the linear trend over time was significant, with a decline of 6.3% (1 minus the exponential of –0.065) [95% confidence interval (CI) 5.6, 7.0] in geometric mean cotinine per year. There was also an association with age, geometric mean cotinine declining by 3.2% (95% CI 2.5, 4.0) with each 1-year increase in age. Observed geometric mean salivary cotinine (the exponential value of the mean of the log-transformed values) varies among subgroups of children defined by their socio-economic position, parental and carer smoking status (Table 2). Social class, employment and parental education were all important predictors of SHS exposure, with the highest geometric mean cotinine observed in

Table 1 Sample sizes and smoking status by year and age group.

	Children aged 4–15 years					*Children aged 8–15 years*				
		Those with valid cotinine sample					*Those with valid cotinine sample and self-reported smoking status*[a]			
				Smokers based on cotinine >12 ng/ml				*Smokers based on self-report*	*Smokers based on cotinine >12 ng/ml*	
Year	*Total no. in core HSE sample*	*No.*	*% of total*	*No.*	*% of valid sample*	*Total no. in core HSE*	*No.*	*% of valid sample*	*No.*	*% of valid sample*
1996	3 303	2 757	83.5	145	5.3	2 100	1798	48	2.7	120	6.7
1997	1 830	1 509	82.5	87	5.8	1 183	983	29	3.0	79	7.3
1998	3 170	2 393	75.5	131	5.5	2 041	1524	40	2.6	109	7.2
2001	3 027	1 990	65.7	86	4.3	2 041	1343	32	2.4	74	5.5
2002	1 479	1 030	69.6	47	4.6	1 026	729	19	2.6	42	5.8
2003	2 856	1 827	64.0	107	5.9	1 935	1277	38	3.0	95	7.4
2005	2 409	790	56.1	29	3.7	957	530	12	2.3	24	4.5
2006	2 710	1 579	58.3	79	5.0	1 841	1105	21	1.9	67	6.1
Total	19 784	13 875	70.1	711	5.1	13 124	9289	239	2.6	603	6.5

[a]Self-reporting smoking recorded only in those aged 8+.

Table 2 Univariate and age/year-adjusted regression analysis of predictors of second-hand smoke (SHS) exposure (log cotinine) in non-smoking children aged 4–15 years, 1996–2006 inclusive.

Predictor		*Sample size*	*Observed geometric mean*	*Regression coefficients (unadjusted)*				*Regression coefficients (adjusted for year and age[a])*			
				Estimate[b]	*95% CI*		*$R^2 \times 100$*	*Estimate[b]*	*95% CI*		*part $R^2 \times 100$*
Year		12 743	–	–0.065	–0.073	–0.058	2.3	–0.063	–0.071	–0.056	
Age		12 743	–	–0.033	–0.041	–0.025	0.5	–0.028	–0.036	–0.020	
Gender	Male[c]	6 367	0.459	–	–	–	<0.01	–	–	–	<0.01
	Female	6 376	0.468	0.019	–0.033	0.071		0.029	–0.022	0.081	
Social class[d]	I and II[c] (professional, managerial and technical)	4 953	0.281	–	–	–	8.6	–	–	–	8.3
	III (skilled non-manual and manual)	4 914	0.546	0.662	0.606	0.719		0.638	0.582	0.693	
	IV and V (semi-skilled and unskilled manual)	2 396	0.877	1.137	1.067	1.206		1.119	1.050	1.187	
Employment status[d]	Employed[c]	10 166	0.386	–	–	–	6.3	–	–	–	5.8
	Unemployed	788	1.037	0.988	0.884	1.093		0.914	0.810	1.018	
	Other (inc. looking after home)	1 602	0.982	0.934	0.857	1.010		0.914	0.839	0.990	
Education status of parents	Higher education qualification[c]	5 032	0.284	–	–	–	9.0	–	–	–	8.5
	School level (or other) qualifications[e]	6 164	0.560	0.679	0.626	0.732		0.665	0.613	0.717	
	No qualification	1 503	1.093	1.348	1.266	1.430		1.308	1.227	1.390	
Crowding (people per bedroom)	<1[c]	1 101	0.323	–	–	–	1.3	–	–	–	1.0
	1–1.5	8 905	0.446	0.323	0.230	0.410		0.277	0.185	0.369	
	>1.5	2 737	0.609	0.634	0.530	0.737		0.555	0.452	0.658	
Ethnicity[f]	White[c]	11 345	0.477	–	–	–	0.5	–	–	–	0.7
	Black or Asian	990	0.318	–0.405	–0.502	–0.309		–0.406	–0.501	-0.310	
Parental smoking status	Neither parent smokes[c]	7 904	0.241	–	–	–	34.7	–	–	–	34.0
	Father only smokes	1 239	0.709	1.080	1.008	1.152		1.053	0.982	1.124	
	Mother only smokes	2 259	1.546	1.859	1.802	1.915		1.851	1.796	1.907	
	Both parents smoke	1 223	2.141	2.185	2.112	2.257		2.151	2.079	2.222	
Someone smokes most days inside the home?	No[c]	8 645	0.245	–	–	–	38.6	–	–	–	37.3
	Yes	4 096	1.780	1.984	1.940	2.027		1.959	1.915	2.002	
Carer smoking (>2 hours per week)[g]	No[c]	6 921	0.317	–	–	–	23.9	–	–	–	23.7
	Yes	2 366	1.708	1.684	1.623	1.745		1.684	1.624	1.745	

[a]Age is adjusted for year only, year is adjusted for age only. [b]The exponential of the regression coefficients are reported in main text. Exponentiating a regression coefficient estimate associated with a continuous predictor produces a multiplier that describes the ratio of the geometric mean cotinine for each unit increase in the predictor variable. For categorical predictors, it describes a multiplicative change compared with the baseline category. [c]Baseline category. [d]Of head of household. [e]Also includes qualifications obtained outside United Kingdom, Nursery Nurse Examination Board, Clerical and Commerical qualifications. [f]Those classed as other ethnicities were excluded from the analysis due to their very small numbers. [g]Only asked in those aged 4–12 years. CI: confidence interval.

children living in social classes IV and V households (3.1 times that of social classes I and II), unemployed households (2.7 times that of employed) and whose parents had no qualifications (3.8 times that of those with a higher education qualification). Geometric mean cotinine was 1.89 times higher in children living in more crowded households (>1.5 people per bedroom) compared with the least crowded households (<0.5 people per bedroom). White children had geometric mean cotinine concentrations that were 1.5 times higher than black or Asian children.

Compared with these socio-demographic variables, parental and carer smoking had a larger impact on children's cotinine levels. Children whose parents both smoke had the highest levels of exposure, followed by children whose mother smokes (8.9 and 6.4 times the geometric mean cotinine, respectively, of children for whom neither parent smokes), paternal smoking having less impact (2.9 times greater than children for whom neither parent smokes). Geometric mean cotinine concentrations were 5.4 times higher in children whose carers smoke compared with non-smoking carers. Children from households in which someone smokes inside on most days had a geometric mean cotinine concentration that was 7.3 times that of children from smoke-free households. There was no significant difference between genders. All predictor variables identified as significant in the univariate analysis remained so, albeit somewhat attenuated, having adjusted for the effects of year and age (Table 2).

In the final multivariate models similar variables remained significant, although their impacts were attenuated substantially once other variables in the model were controlled for (Table 3). Overcrowding became non-significant, probably as a result of colinearity with other variables in the model. In 4–15-year-olds the most important markers remained parental smoking status, whether someone smokes inside the home, parental education, ethnicity, social class and employment of head of household. The model for 4–12-year-olds (Table 3) was very similar, although carer smoking and gender were also significant, with geometric mean cotinine 7% higher in females than males.

The percentage of variation ($R^2 \times 100$) explained by each unadjusted univariate model indicated, in line with the regression coefficients, that households in which someone smokes inside and parental and carer smoking status were strong predictors of SHS exposure, explaining 38.6%, 34.7% and 23.9% of the variation in log cotinine concentrations, respectively. These remained the strongest predictors after adjusting for year and age (part R^2: 37.3%, 34%, 23.7%). The percentage of the overall variation in the log cotinine explained by the multiple regression model was reasonably high (48.8% and 45.5% for the 4–12-year-old and 4–15-year-old age groups, respectively).

Trends over time and changing inequalities in exposure

As indicated above, geometric mean cotinine declined significantly over time. This is illustrated further in Fig. 1, which displays an overall decline in observed geometric mean cotinine of 59% from 0.59 ng/ml in 1996 to 0.24 ng/ml in 2006. The greatest change, in both absolute and relative terms, occurred between 2005 and 2006 (37% decline) and, in the absence of data from 2004, from 2003 to 2005 (20% decline).

The decline over time tended to be greatest in children who were most exposed, indicating that absolute inequalities in SHS exposure have fallen (Figs 1 and 2). This was confirmed in our median regression analysis (Table 4). For example, median cotinine levels declined by 0.06 ng/ml per year in children living in social classes IV and V households and 0.039 ng/ml per year in children in social class III households, both significantly greater annual declines compared with the 0.023 ng/ml in social classes I and II households ($P < 0.05$). Children whose parents had no qualifications, the most exposed group in 1996, experienced a 0.071 ng/ml annual decline in median cotinine compared with a 0.024 ng/ml annual decline in those whose parents had a higher education qualification ($P < 0.05$), although the decline in those with school-level qualifications did not differ from that in the most educated group. Median cotinine levels also declined significantly faster in white compared with black and Asian children.

Importantly, not only do we show that exposure fell in children of smokers but that median cotinine levels declined significantly more in the most exposed groups, children whose parents both smoke or whose mother smokes (annual falls of 0.115 ng/ml and 0.065 ng/ml, respectively), compared with children with non-smoking parents (0.019 ng/ml). This provides further evidence that absolute differences in cotinine levels have narrowed over this 11-year period (Tables 4 and Fig. 2). Children whose father only smokes also experienced a significant decline, but this was no greater than that seen in children of non-smoking parents (Table 4). Similarly, between 1996 and 2006, the median cotinine level in children from households that allow smoking declined by 0.042 ng/ml per year, significantly faster than in households that do not allow smoking (0.018 ng/ml).

There was, however, no evidence of a difference in trends over time in children from households with an unemployed compared with employed head of household or between children whose carers smoked compared with those who did not.

Table 3 Multivariate regression analysis of predictors of second-hand smoke (SHS) exposure (log cotinine) in non-smoking children, 1996–2006 inclusive.

		Regression coefficients			*Regression coefficients*		
		4–15-year-olds[a]			*4–12-year-olds*[b]		
Predictor		*Estimate*[c]	*95% CI*		*Estimate*[c]	*95% CI*	
Year		−0.042	−0.048	−0.037	−0.044	−0.051	−0.037
Age		−0.025	−0.031	−0.018	−0.043	−0.052	−0.033
Gender	Male[d]	Not significant; dropped from model			–	–	–
	Female				0.068	0.022	0.113
Social class[e]	I and II (professional, managerial and technical)[d]	–	–	–	–	–	–
	III (skilled non-manual and manual)	0.133	0.084	0.181	0.113	0.057	0.170
	IV and V (semi-skilled and unskilled manual)	0.253	0.189	0.316	0.222	0.150	0.295
Employment status[e]	Employed[d]	–	–	–	–	–	–
	Unemployed	0.268	0.180	0.356	0.228	0.128	0.328
	Other (inc. looking after home)	0.343	0.273	0.412	0.311	0.233	0.388
Education status of parents	Higher education qualification[d]	–	–	–	–	–	–
	School level (or other) qualifications[f]	0.208	0.161	0.255	0.205	0.151	0.260
	No qualification	0.397	0.319	0.475	0.382	0.291	0.473
Crowding (people per bedroom)	<1[d]	Not significant; dropped from model			Not significant; dropped from model		
	1–1.5						
	>1.5						
Ethnicity[g]	White[d]	–	–	–	–	–	–
	Black or Asian	−0.183	−0.260	−0.105	−0.097	−0.187	−0.008
Parental smoking status	Neither parent smokes[d]	–	–	–	–	–	–
	Father only smokes	0.311	0.230	0.392	0.309	0.217	0.402
	Mother only smokes	0.748	0.671	0.826	0.703	0.615	0.791
	Both parents smoke	1.082	0.992	1.172	1.029	0.926	1.132
Someone smokes most days inside the home?	No[d]	–	–	–	–	–	–
	Yes	1.127	1.056	1.199	0.941	0.856	1.026
Carer smoking (>2 hours per week)[h]	No[d]				–	–	–
	Yes				0.512	0.447	0.578

[a]$n = 11\,645$. Includes all variables listed other than those indicated as dropped from the model and carer smoking. [b]$n = 8448$. Includes all variables listed other than those indicated as dropped from the model. [c]The exponential of the regression coefficients are reported in main text. Exponentiating a regression coefficient estimate associated with a continuous predictor produces a multiplier that describes the ratio of the geometric mean cotinine for each unit increase in the predictor. For categorical predictors, it describes a multiplicative change compared with the baseline category. [d]Baseline category. [e]Of head of household. [f]Also includes qualifications obtained outside United Kingdom, Nursery Nurse Examination Board, Clerical and Commercial qualifications. [g]Those classed as other ethnicities were excluded from the analysis due to their very small numbers. [h]Only asked in those aged 4 to 12 and therefore not included in 4–15-year-olds' analysis. CI: confidence interval.

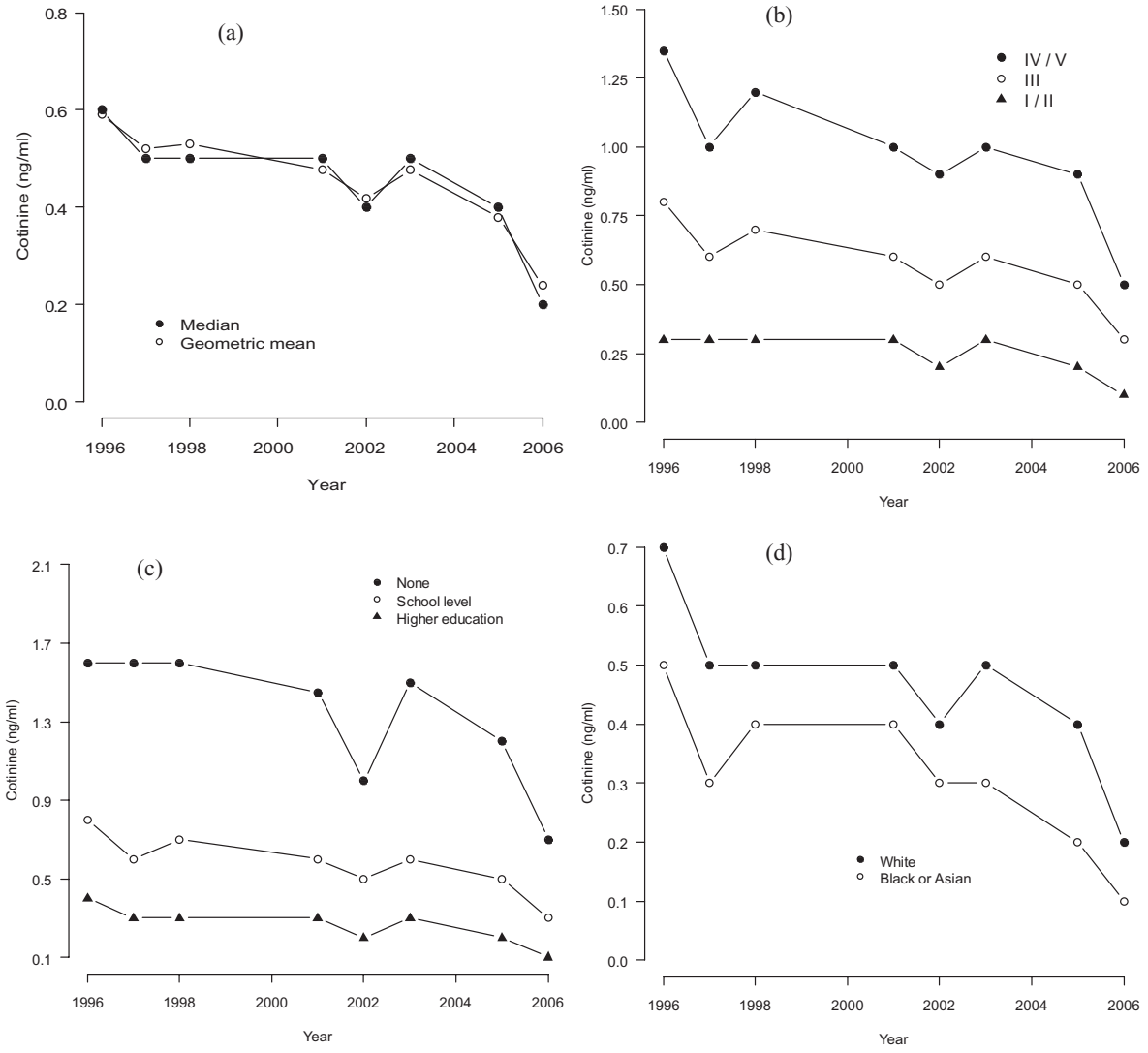

Figure 1 Median cotinine levels (ng/ml) over time for non-smoking children aged 4–15 years (a) overall and by (b) social class of head of household, (c) highest educational qualification of parents and (d) ethnicity. Geometric mean cotinine levels (ng/ml) also displayed in (a) for comparison

Comparison of households that do and do not allow smoking

The proportion of households in which someone smokes inside on most days varies widely with parental smoking status and socio-economic position (Appendix S2). In only 3.9% of households where neither parent smokes does someone smoke inside on most days. This compares to 88.3% of households where both parents smoke, 82.1% where the mother only smokes and 64.7% where the father only smokes. Households in which someone smokes inside most days are more likely to have a head of household who is not currently employed or of lower social class, have parents with a lower level of education and children of white ethnicity; χ^2 tests confirm that these results were statistically significant for all comparisons.

DISCUSSION

To our knowledge, this is the most comprehensive study conducted to date of the predictors of and trends in exposure to SHS among children in England. It covers a wider age range and a longer time-period than previous work and provides a more comprehensive analysis of the factors influencing exposure. It is also, to our knowledge, the largest data set on cotinine available internationally.

Nevertheless, a number of issues need to be considered. First, the proportion of respondents with valid cotinine samples reduced over time with a reduction in the proportion of younger children in later years. However, given that the profile of respondents did not change significantly in relation to other parameters under consideration (social class, ethnicity, etc.) and all the regression models, except the unadjusted univariate models,

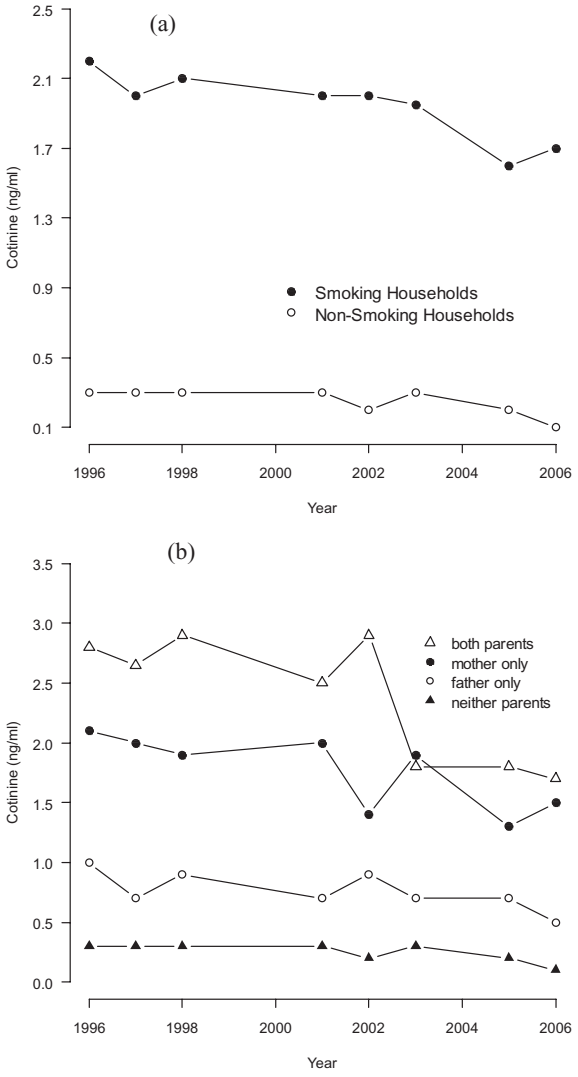

Figure 2 Median cotinine levels (ng/ml) over time in children aged 4–15 years by (a) household smoking status and (b) parental smoking status

adjusted for age, we believe that the observed decline in cotinine is unlikely to be an artefact of changes in the sampling. A further potential source of bias is misclassification of smoking status in children. However, we took a conservative approach by using both salivary cotinine and self-report to define smoking and thus believe that most smokers will be identified correctly. Indeed, the data in 8–15-year-olds shows that relying on self-report alone will underestimate youth smoking and supports our decision to also use cotinine.

The study identifies the major predictors of SHS exposure. Our multivariable model explains almost 50% of the variation in childrens' cotinine levels. We show that the most important predictors are modifiable factors—whether people smoke in the house, whether the parents smoke and whether the children are looked after by carers who smoke. While the identification of the importance of parental smoking is not new, our analysis shows that the role of carers must not be overlooked. The proportion of children looked after by a carer who smoked varied with parental smoking status, increasing from 9% in children of non-smoking parents to 59% in children whose parents both smoke. Beyond this, more deprived children tend to be more exposed, while black and Asian children are less exposed than white children.

This last finding contrasts with evidence from the United States that black children have higher cotinine levels than white children when exposed to the same amount of smoke [12], due probably to differences in cotinine metabolism [13]. However, the majority of our ethnic minority sample (71%) comprised Asian children, and we were unable to identify any study on cotinine metabolism in Asian populations. In addition to this, our findings are probably explained by the black and Asian children in our sample being more likely to live in a smoke-free home (Appendix S2), to have non-smoking parents, and when their parents smoke they had lower levels of cigarette consumption than their white counterparts (data not shown). These findings are also consistent with recent qualitative work in London showing that white smokers were more likely to allow smoking in their home than Turkish or Somali smokers [14].

The fact that markers of deprivation remain important, even once parental smoking status has been controlled for, suggests that they may influence children's exposure independenly of parental or carers' smoking status and whether smoking is allowed in the home. This may be because community exposure is also greater in these groups, an issue we are unable to explore further given the absence of markers of community exposure in the survey.

Marked declines in SHS exposure were observed with the largest decline seen between 2005 and 2006, the period immediately preceding the implementation of SFL. This rapid recent decline may be due partially to the growing number of public venues becoming smoke-free in advance of 1 July 2007. However, our findings, that smoking in the home, parental and carer smoking are important predictors of children's exposure and that exposure fell more steeply in children whose parents smoke and those living in households allowing smoking, suggest that wider positive behavioural change was prompted by public debates and information campaigns on SHS. This is supported by evidence that, in England, the proportion of children living with non-smoking parents and in smoke-free homes has increased over time, changes which have inevitably played a part in reducing children's exposure to SHS [15].

The declines in SHS exposure over time are consistent with previous analyses of HSE data [4], but we show for

Table 4 Median regression analysis of trends in second-hand smoke (SHS) exposure (cotinine) in non-smoking children aged 4–15 years, from 1996 to 2006.

Predictor		*Annual change in median cotinine*[a]
Social class	I and II[b] (professional, managerial and technical)	−0.023*
	III (skilled non-manual and manual)	−0.039*
	IV and V (semi-skilled and unskilled manual)	−0.060*
Employment status	Employed[b]	−0.028*
	Unemployed	−0.006
	Other (inc. looking after home)	−0.069*
Education status of parents	Higher education qualification[b]	−0.024*
	School level (or other) qualifications	−0.032
	No qualification	−0.071*
Ethnicity	White[b]	−0.039*
	Black or Asian	−0.028*
Parental smoking status	Neither parent smokes[b]	−0.019*
	Father only smokes	−0.029
	Mother only smokes	−0.065*
	Both parents smoke	−0.115*
Someone smokes most days inside the home?	No[b]	−0.018*
	Yes	−0.042*
Carer smoking (>2 hours per week)	No[b]	−0.027*
	Yes	−0.052

An asterisk (*) beside a baseline category indicates a significant difference ($P < 0.05$) from zero, while in other categories it indicates a significant difference ($P < 0.05$) from their respective baseline categories. [a]The annual change in median cotinine for a baseline category was obtained from the regression coefficient for year in the model. The annual changes for all other categories were derived from the sum of the regression coefficients for year and the interaction between year and the category. [b]Baseline category.

the first time that absolute declines have been greatest in the most exposed children and that exposure has fallen significantly in children of smokers. Both the graphical representation and median regression analysis show clearly that absolute inequalities in exposure have declined. A number of issues are nevertheless worth noting. First, there were two main groups where exposure did not decline fastest in the most exposed—among children whose parents were unemployed and whose carers smoked. Secondly, despite these declines, we show that exposure remains greatest in children living in households where the head of the household has no qualification, is unemployed or in social classes IV or V, and that these households are also less likely to have smoking restrictions in place, as other research has indicated [16]. Thirdly, there are various ways of examining inequalities and, had we instead examined relative inequalities (the ratio between the most and least deprived groups), we may have seen slightly different results. For example, a brief exploration of relative changes in inequalities, by fitting an interaction model to log cotinine values, indicated no significant difference in the percentage decline per year in geometric mean cotinine among children grouped according to socioeconomic status (−6.1%, −6.0% and −5.3% for social classes I/II, III and IV/V, respectively) or by parental smoking status (−5.7%, −4.5%, −4.3% and −6.3% for neither parent smokes, father only smokes, mother only smokes and both parents smoke, respectively). In contrast, percentage declines were highest in the least exposed when children were grouped by whether their carer smokes (−6.7% when carer does not smoke compared with −3.8% when carer smokes) or someone smokes inside their home on most days (−4.8% when no one smokes in the home compared with −2.3% when someone smokes).

Although it is difficult to quantify the exact health benefits of these declines in exposure, recent evidence suggests that even very low exposures to SHS in children can lead to developmental impacts and endothelial damage [17,18]. This implies that even those children whose SHS exposure levels are now low remain at risk from the health impacts of exposure, while substantial numbers of children still remain highly exposed.

Implications for policy and practice

The importance of carer and parental smoking and household exposure indicates that reducing exposure in the home is key to reducing children's morbidity from SHS exposure. A 2002 comparison of the United Kingdom with the United States, Canada and Australia showed that

UK smokers were the least likely to live in smoke-free homes. If these disparities remain, it suggests significant scope for improvement in the United Kingdom [19].

Strict no-smoking policies in the home have been associated with significantly lower levels of exposure in children [20,21], while less restrictive measures, such as opening windows or limiting smoking to a single room, have little impact [1]. Strict policies may also have other benefits, including encouraging cessation in household members and discouraging uptake among adolescents [22,23]. However, evidence on the effectiveness of interventions that reduce children's exposure is limited [24,25]. A recent Cochrane review identified intensive counselling interventions targeted to smoking parents as the only intervention effective in reducing exposure and changing parental smoking location [24]. However, the studies failed to address carer smoking, and none of the studies that found evidence of an impact were from the United Kingdom [24]. Although smoke-free homes initiatives are being developed in England, they have been evaluated inadequately to date [26].

Qualitative work provides insights into the barriers to and reasons for restricting smoking in the home, particularly among disadvantaged parents [27–29]. Although smokers have some knowledge of the risks of SHS [28] and health concerns, notably around not exposing children or grandchildren, play a role in restricting smoking, one study shows that mothers construct alternative explanations for their children's ill health and few continue their efforts to protect babies from SHS into infancy [28]. An Australian study suggested that a desire to smoke in comfort, the difficulty of asking family and friends who may be helping out with the children not to smoke, the difficulty of supervising children and the expense of quit products were all important barriers, while a lack of outdoor space may be less of an issue [27].

Implementing legal restrictions on home smoking is controversial and opposed strongly by key population groups [1]. However, several jurisdictions have introduced successfully bans on smoking in cars carrying children [30]. Similar restrictions may be feasible elsewhere and might help to reinforce health promotion messages around SHS [7].

Our findings also shed light on some of the complexities of evaluating the impacts of SFL. They suggest that if evaluations do not allow for long-term declines in SHS exposure, e.g. by including geographical controls or adjusting for the decline by modelling the trend appropriately in a regression model, they may *overestimate* the effectiveness of legislation; but they also suggest that controlling for these declines in the period immediately before implementation may *underestimate* the impacts of legislation, as some of this decline probably reflects true impacts of the legislation mediated both through increased public debate around (and thus understanding of) the health impacts of SHS and through public places becoming smoke-free in advance of legislation. Those evaluating the impacts of SFL need to take these issues into account.

SUMMARY

This study highlights that the most important predictors of SHS exposure in children (parental and carer smoking and allowing smoking in the home) are factors that are amenable to change. Although this paper documents marked declines in exposure over time, international comparisons suggest that further change is possible. Reducing parental and carer smoking rates is likely to be the most effective means of reducing children's exposure and must be the ultimate aim. However, even with effective tobacco control policies [31] and cessation services in place, quit rates remain low [32], making it necessary in the interim to encourage other behavioural changes, such as opting to smoke outdoors. Unfortunately, however, evidence of the effectiveness of interventions to reduce children's exposure is limited and even less is known about carer smoking. More research is therefore needed to explore what works to change parental and carer smoking habits, particularly among children most at risk of exposure or of negative health outcomes through exposure (e.g. asthmatic children).

Declarations of interest

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References

1. Ashley M. J., Ferrence R. Reducing children's exposure to environmental tobacco smoke in homes: issues and strategies. *Tob Control* 1998; **7**: 61–5.
2. Royal College of Physicians (RCP). *Going Smoke-Free: The Medical Case for Clean Air in the Home, at Work and in Public Places*. London: RCP; 2005.
3. Jarvis M. J., Russell M. A., Feyerabend C., Eiser J. R., Morgan M., Gammage P. *et al.* Passive exposure to tobacco

smoke: saliva cotinine concentrations in a representative population sample of non-smoking school children. *BMJ* 1985; **291**: 927–9.
4. Royal College of Physicians Tobacco Advisory Group. *Going Smoke-Free: The Medical Case for Clean Air in the Home, at Work and in Public Places*. London: Royal College of Physicians of London; 2005.
5. California Environmental Protection Agency. *Proposed Identification of Environmental Tobacco Smoke As a Toxic Air Contaminant. Part B: Health Effects*. As aproved by the Scientific Review Panel on 24 June 2005. Sacramento: California Environmental Protection Agency; 2005.
6. Willers S., Skarping G., Dalene M., Skerfving S. Urinary cotinine in children and adults during and after semiexperimental exposure to environmental tobacco smoke. *Arch Environ Health* 1995; **50**: 130–8.
7. Department of Health. *Consultation on the Future of Tobacco Control*. London: Department of Health; 2008.
8. Health Survey for England Homepage. Available at. http://www.dh.gov.uk/en/Publicationsandstatistics/PublishedSurvey/HealthSurveyForEngland/index.htm (last accessed 4 April 2009).
9. Jarvis M. J., Fidler J., Mindell J., Feyerabend C., West R. J. Assessing smoking status in children, adolescents and adults: cotinine cut-points revisited. *Addiction* 2008; **103**: 1553–61.
10. Feyerabend C., Russell M. A. A rapid gas–liquid chromatographic method for the determination of cotinine and nicotine in biological fluids. *J Pharm Pharmacol* 1990; **42**: 450–2.
11. Koenker R., Hallock K. F. Quantile regression. *J Econ Perspect* 2001; **15**: 143–56.
12. Wilson S. E., Kahn R. S., Khoury J., Lanphear B. P. The role of air nicotine in explaining racial differences in cotinine among tobacco-exposed children. *Chest* 2007; **131**: 856–62.
13. Benowitz N. L., Perez-Stable E. J., Fong I., Modin G., Herrera B., Jacob P. III. Ethnic differences in N-glucuronidation of nicotine and cotinine. *J Pharmacol Exp Ther* 1999; **291**: 1196–203.
14. Lock K., Adams E., Pilkington P., Duckett K., Thoms M., Gilmore A. *et al*. Evaluation of the impact of smokefree legislation in different ethnic groups in Islington. A study for Islington Primary Care Trust. London: London School of Hygiene and Tropical Medicine; 2008.
15. Jarvis M. J., Mindell J., Gilmore A., Feyerabend C., West R. Smoke-free homes in England: prevalence, trends and validation by cotinine in children. *Tob Control* 2009; **18**: 491–5.
16. Phillips R., Amos A., Ritchie D., Cunningham-Burley S., Martin C. Smoking in the home after the smoke-free legislation in Scotland: qualitative study. *BMJ* 2007; **335**: 553.
17. Yolton K., Dietrich K., Auinger P., Lanphear B. P., Hornung R. Exposure to environmental tobacco smoke and cognitive abilities among U.S. children and adolescents. *Environ Health Perspect* 2005; **113**: 98–103.
18. Kallio K., Jokinen E., Raitakari O. T., Hamalainen M., Siltala M., Volanen I. *et al.* Tobacco smoke exposure is associated with attenuated endothelial function in 11-year-old healthy children. *Circulation* 2007; **115**: 3205–12.
19. Borland R., Yong H. H., Cummings K. M., Hyland A., Anderson S., Fong G. T. Determinants and consequences of smoke-free homes: findings from the International Tobacco Control (ITC) Four Country Survey. *Tob Control* 2006; **15**: iii42–50.
20. Wakefield M., Banham D., Martin J., Ruffin R., McCaul K., Badcock N. Restrictions on smoking at home and urinary cotinine levels among children with asthma. *Am J Prev Med* 2000; **19**: 188–92.
21. Johansson A. K., Hermansson G., Ludvigsson J. How should parents protect the children from environmental tobacco smoke in the home? *Pediatrics* 2004; **113**: 291–5.
22. Wakefield M. A., Chaloupka F. J., Kaufman N. J., Orleans C. T., Barker D. C., Ruel E. E. Effect of restrictions on smoking at home, at school, and in public places on teenage smoking: cross sectional study. *BMJ* 2000; **321**: 333–7.
23. Messer K., Mills A. L., White M. M., Pierce J. P. The effect of smoke-free homes on smoking behavior in the U.S. *Am J Prev Med* 2008; **35**: 210–6.
24. Priest N., Roseby R., Waters E., Polnay A., Campbell R., Webster P. *et al.* Family and carer smoking control programmes for reducing children's exposure to environmental tobacco smoke. *Cochrane Database of Systematic Reviews 2008*, Issue 4. Art. No.: CD001746. DOI: 10.1002/14651858. CD001746.
25. Hovell M. F., Zakarian J. M., Wahlgren D. R., Matt G. E. Reducing children's exposure to environmental tobacco smoke: the empirical evidence and directions for future research. *Tob Control* 2000; **9**: ii40–7.
26. Al-alawy K., Wellington N., Dalton E., Fendall L., MacDonald T., Williamson M. Smoke-free homes: outcomes of a local service development. *Commun Pract* 2008; **81**: 30–3.
27. Hill L., Farquharson K., Borland R. Blowing smoke: strategies smokers use to protect non-smokers from environmental tobacco smoke in the home. *Health Promot J Aust* 2003; **14**: 196–201.
28. Robinson J., Kirkcaldy A. J. 'You think that I'm smoking and they're not': why mothers still smoke in the home. *Soc Sci Med* 2007; **65**: 641–52.
29. Robinson J., Kirkcaldy A. J. Disadvantaged mothers, young children and smoking in the home: mothers' use of space within their homes. *Health Place* 2007; **13**: 894–903.
30. Rees V. W., Connolly G. N. Measuring air quality to protect children from secondhand smoke in cars. *Am J Prev Med* 2006; **31**: 363–8.
31. Joossens L., Raw M. Progress in tobacco control in 30 countries, 2005–2007. European Conference on Tobacco or Health (Berne); 2007.
32. Royal College of Physicians. *Harm Reduction in Nicotine Addiction: Helping People Who Can't Quit*. London: RCP; 2007, p. 218–31.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1: The Health Survey for England

Appendix S2: Comparison of households containing non-smoking children aged between 4 and 15 years that do and do not allow smoking inside (based on whether someone smokes inside the home on most days) by socio-economic indicators

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