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# **Household smoking behaviours and exposure to Environmental Tobacco Smoke amongst infants: are current strategies effectively protecting our young?**

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## **Abstract**

**Objective:** To determine: the prevalence of infant exposure to environmental tobacco smoke (ETS) amongst infants attending Child Health Clinics in regional NSW; the association between such exposure and household smoking behaviours; and the factors associated with smoking restrictions in households with infants.

**Methods:** Parents completed a computer based questionnaire and infant urine samples were collected. Information was obtained regarding the smoking behaviours of household members and samples were analysed for cotinine.

**Results:** Twenty seven percent of infants had detectable levels of cotinine. Infant ETS exposure was significantly associated with smoking status of household members, absence of complete smoking bans in smoking households and having more than one smoker in the home. Smoking households were significantly less likely to have a complete smoking ban in place.

**Conclusions:** This study suggests that a significant proportion of the population group most vulnerable to ETS were exposed.

**Implications:** Future efforts to reduce children's exposure to ETS need to target cessation by smoking parents, and smoking bans in households of infants where parents are smokers if desired reductions in childhood ETS related illness are to be realised.

## **Introduction**

Childhood exposure to ETS is associated with an increased risk of lower respiratory illness, asthma, otitis media and Sudden Infant Death Syndrome.<sup>1</sup> Infants are particularly vulnerable because of their small body size, higher ventilation rates and underdeveloped immune and pulmonary systems.<sup>1</sup>

Increased public awareness of the harmful effects of ETS exposure has prompted the introduction of initiatives such as smoke free workplaces. However, such policies offer little protection in environments such as the home, the most common location of ETS exposure for children.<sup>1</sup> Whilst community surveys suggest that an increasing awareness of the dangers of ETS exposure for children has led to increased protective behaviours by parents, there is evidence to suggest that many children continue to be exposed.<sup>2-4</sup>

Few studies have investigated the prevalence of ETS exposure amongst infants. Those that do exist primarily rely on parent self report rather than biochemical measures such as cotinine, the preferred indicator of ETS exposure.<sup>4</sup> The only known Australian research using biochemical measures found detectable levels of cotinine in the urine of as many as 40% of a non representative sample of infants.<sup>2</sup>

The most common source of infant ETS exposure is smoking by parents and other household members.<sup>2,5</sup> In Australia, up to 34% of infants aged less than 12 months live with smokers. In addition to parental smoking, the smoking behaviours most highly correlated with biochemically measured ETS exposure are the number of cigarettes smoked per day by parents, the number of household smokers, and the presence or absence of household smoking restrictions.<sup>5-11</sup> Given such associations the two key protective behaviours most likely to reduce ETS exposure are for parents to either quit smoking or introduce complete household smoking restrictions.<sup>1</sup> Although various studies have reported an increased

prevalence of such restrictions in households with children<sup>1</sup>, no research has described the factors associated with their implementation.

Given the limited data available, this study sought to determine the:

- prevalence of infant exposure to ETS amongst a sample of infants attending Child Health Clinics in regional NSW.
- association between infant ETS exposure and household smoking behaviours.
- factors associated with the implementation of household smoking restrictions.

## **Method**

### ***Sample***

One third of all 39 Child Health Clinics located within the Hunter Region of NSW were randomly selected. Such clinics are attended by up to 92% of Australian infants and provide information and support for parents of children aged 0-4 years.<sup>12</sup> Infants were eligible to participate if they were 12 months of age or younger and if their parents had sufficient English to enable them to complete the study procedures.

The sample size was estimated using the normal approximation to the binomial and assumed a worst case scenario estimate of prevalence of exposure of 50%.<sup>13</sup> A final sample size of 385 consenting infant/parent pairs was required to estimate the prevalence of ETS exposure within +/- 5% accuracy. The sample size selected from each of the 13 clinics was proportional to their average monthly throughput and ranged from 2 infants to 81 infants per clinic.

### ***Procedures***

Infant/parent pairs attending each clinic were approached for consent to complete a computer based questionnaire and to allow an infant urine sample to be obtained. Parents were informed that the sample was to be analysed for exposure to environmental health

risks. Cigarette smoke was not specifically mentioned. Urine samples were obtained by placing, a cotton wool pad in the nappy of each infant.<sup>5</sup> Samples were frozen and transported to an accredited laboratory for cotinine analysis. Study approval was obtained from the relevant research ethics committees.

## **Measures**

### ***Exposure to ETS***

Urine samples were analysed for the presence of cotinine using capillary gas chromatography.<sup>14</sup> The lower detection limit for cotinine with this method was 6nmol/L. Creatinine levels were also measured to correct for the dilution of the urine,<sup>15</sup> and a cotinine/creatinine ratio (CCR) determined. As in previous studies, infants were classified as exposed if they had measurable amounts of cotinine in their urine.<sup>2;16</sup> Single cotinine measures have been shown to be an accurate reflection of an infant's recent exposure to ETS.<sup>5;17</sup>

In order to allow for the possible effects of breastfeeding on cotinine levels,<sup>18</sup> the breastfeeding status of the infant was identified.

### ***Household smoking behaviours***

#### ***Smoking status of household members***

Information was collected regarding the smoking status of each household member aged 16 years and older (never smoker, ex-smoker, occasional smoker, regular smoker).

#### ***Cigarettes smoked each day***

Information was sought on the number of cigarettes smoked daily by mothers who smoked (10 or less, 11-20, 21-30, 31 or more).

### *Household smoking restrictions*

Parents were asked which of the following options best described smoking in their home: “there is no smoking inside at all”, “smoking is limited to part of the house where the baby/children rarely go”, “smoking does not occur in the baby’s children’s bedrooms but occurs elsewhere in the house”, “smoking is allowed in any room”. Parents were also asked if there were any exceptions to this situation.<sup>10</sup>

### ***Infant and parent characteristics***

Information was collected regarding the age and gender of the infant and of the accompanying parent as were details of parental educational attainment (Yr 10 or less, Yr 11 & 12, trade certificate, tertiary qualifications) and marital status (single, married/de facto, separated/divorced, widowed).

## **Analysis**

### ***Prevalence of exposure to ETS***

The number of infants with detectable levels of cotinine is reported distinguishing the sub-sample of infants who are breast fed by smoking mothers. Intra-cluster correlation coefficients were calculated for all outcome measures to investigate the potential correlation due to sampling from within clinics.

### ***Association between household smoking behaviours and exposure to ETS***

The relationships between household smoking behaviours and ETS exposure as defined by the presence of urinary cotinine (yes/no) was investigated using Pearson Chi square analyses. *t*-tests were used to examine the relationships between household smoking behaviours and mean CCR.

The skewed distribution of CCR necessitated a transformation using the  $\log_{10}$  function to improve the approximation to the normal distribution. To determine where significant associations existed simple linear regression was performed with each behavioural variable and the transformed CCR. The coefficient of determination ( $R^2$ ) describing the proportion of the variability in the exposure variable explained by the association with the behavioural variable, is reported along with geometric means and their 95% CI.

Attempts were made to develop multivariate models, however collinearity between variables relating to the identity and number of smokers in the house as well as the dominance of mother's smoking status, prevented the formulation of any informative models beyond the univariate analyses.

### ***Factors associated with implementation of household smoking restrictions***

Associations between household smoking ban, mother's smoking, number of cigarettes smoked per day by mother, mother's marital status, mother's education, father's smoking, other smokers in the home and number of household smokers were assessed using Pearson Chi square analyses.

The data was analysed using SAS V8.2.<sup>19</sup>

## **Results**

### ***Sample***

701 infant/parent pairs attended the clinics during the study period. Of these 280 (40%) were ineligible, 274 being older than 12 months and 6 with parents who could not complete the questionnaire because of language difficulties.

Of the 421 eligible infant/parent pairs, 404 (96%) consented to participate. When compared to the characteristics of attendees at all 39 Child Health Clinics in the Hunter Region the



characteristics of the study sample were similar. With reference to babies born in NSW, the sample had more parents born in Australia (92% vs 73%) and more smokers (18.8% vs 17.1%).<sup>20</sup>

Urine samples were collected from 100% of participating infants. 9.6% of infants were being breastfed by smoking mothers.

### ***Prevalence of exposure to ETS***

One hundred and nine (27%) [95% CI 23-31] infants had detectable levels of cotinine. After removal of the 39 breastfed infants of smoking mothers, 76 (21%) [95% CI 17-25] infants had detectable levels of cotinine. The median concentration of cotinine for the entire sample was 36nmol/L with a range of 6nmol/L to 1069nmol/L. When breast feeding smokers were removed the median was 19.5 nmol/L and values ranged from 6nmol/L to 536nmol/L.

Intra class correlations for the exposure outcomes were non-significant: cotinine 0.016 (CI - 0.008-0.09), CCR -0.007 (CI -0.02-0.036), log<sub>10</sub>ccr 0.005 (CI -0.01-0.07).

### ***Association between household smoking behaviours and exposure to ETS***

Forty one percent of infants lived in a home with at least one smoker, 19.3% lived with a mother who smoked, 29.8% lived with a father who smoked, and 6.9% lived with someone else in the household who smoked. In households where the mother was a smoker, 62% of fathers were also smokers.

Ninety one percent of infants lived in a home with a total smoking ban. Given this, the variable was collapsed into a dichotomous variable, "Complete ban" - no smoking inside at all with no exceptions and "Partial ban"- smoking allowed inside or where exceptions to the ban we accepted.

The presence of urinary cotinine and higher CCRs were significantly associated with mother's and father's smoking status, other smokers in the home, absence of complete smoking bans in smoking households and having more than one smoker in the household (Table 1).

In the simple linear regression analyses, number of household smokers accounted for the greatest amount of variation in urine CCR ( $R^2$  0.170,  $P < .001$ ), followed by household smoking restrictions ( $R^2$  0.127,  $P < .0001$ ), maternal smoking ( $R^2$  0.122,  $p < .0001$ ) and paternal smoking ( $R^2$  0.101,  $P < .0001$ ).

[Insert Table 1 about here]

### ***Factors associated with implementation of household smoking restrictions***

Homes where the mother or father were smokers were less likely to have complete bans in place than those where the parents were non smokers (Table 2). Mothers who smoked greater than 10 cigarettes per day were less likely than lighter smokers to have a complete smoking ban and the greater the number of smokers in the home the less likely a complete ban was in place.

[Insert Table 2 about here]

## **Discussion**

This study is the first to examine the prevalence of bio-chemically measured ETS exposure amongst a representative sample of infants residing in NSW. The finding that 27% of infants were exposed to ETS is consistent with estimates of the number of Australian infants living in households with smoking parents.<sup>10</sup>

A significantly lower prevalence, and a significantly lower level of infant ETS exposure were evident in homes where complete smoking bans existed. Previous studies in specific

populations groups such as asthmatic or hospitalised children have indicated similar associations.<sup>6;7;10;11</sup> The presence of urinary cotinine and mean CCR were significantly associated with mother's and father's smoking status. Whilst a relationship between maternal smoking child ETS exposure has been demonstrated previously,<sup>10</sup> paternal smoking has not always demonstrated a similar relationship.<sup>10;11</sup> Whilst father's smoking was found to be highly significant, mean CCR for infants with smoking mothers was twice that for infants with smoking fathers. In addition, maternal smoking accounted for a greater proportion of the variance in infant urine CCR. However, given that for 62% of infants both the mother and the father smoked, it is possible that the stronger effect found for mothers in this case may be a result of an association between maternal and paternal smoking.

In this sample of infants the household smoking behaviour that accounted for the greatest variance in CCR was the number of smokers in the home. These findings indicate that if more than one smoker lives in a home, then the effectiveness of household smoking bans may be negated. Winkelstein et al,<sup>21</sup> reported similar findings where smoking outside the home offered no protection when more than one smoker lived in the home.

Whilst the evidence suggests that the implementation of smoking bans can reduce ETS exposure amongst infants, it appears that households where these bans are most needed are less likely to implement them. Homes where the mother and/or father were smokers were significantly less likely to have complete bans (81% and 83%) compared to homes where parents were non-smokers (93% and 94%). In addition, mothers who were heavier smokers were less likely to have a complete smoking ban in place (46% vs 88%). This is consistent with findings of previous studies in the US.<sup>9;22</sup> The proportion of homes with smoking bans significantly decreased with increasing number of smokers living in the household. It appears then that whilst there is report of increasing rates of smoke free households in the community generally,<sup>23</sup> those homes where infants are at the greatest risk, that is where there are

smoking parents and multiple smokers in the home, are less likely to have smoking bans in place.

The findings of this study suggest that future campaigns and interventions aimed at reducing children's exposure to ETS will need to incorporate far more targeted and tailored strategies for smokers including strategies encompassing the importance of quitting smoking in reducing child ETS exposure<sup>24</sup> and the importance of strict household smoking bans. The effectiveness of such campaigns should be rigorously evaluated, not in terms of their capacity to increase the prevalence of bans across the community, but specifically in terms of increasing bans amongst smoking parents.

The findings should be considered in light of potential study limitations. Whilst it is suggested the majority of Australian infants attend Child Health Clinics,<sup>20</sup> it is possible that infants who do not attend such clinics are at disproportionately greater risk of ETS exposure. Further, the research was conducted within a single health region where the number of Australian born women was much higher than for the rest of the state. Given this, the current sample may not be representative of all infants. Lastly, the lowest detectable limit of cotinine was used to define ETS exposure. This was deemed appropriate given that there is currently no evidence regarding what might constitute a safe level of infant ETS exposure.

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Table 1: Association of smoking behaviours with infant urinary cotinine and cotinine/creatinine ratio (CCR)\*

	N	Cotinine %	<i>p</i>	Geometric mean CCR (nmol/L)	95% CI		<i>p</i>	R <sup>2</sup>	SE
					Lower	Upper			
<b>Mother smoker</b>									
Yes	39	64	<.0001	7.38	4.18	13.02	<.0001	0.122	0.093
No	326	16		1.60	1.41	1.83			
<b>No. cigarettes smoked per day by mother</b>									
≤10	15	60	0.851	7.96	2.70	23.41	0.7677	0.004	0.338
11+	11	64		10.04	2.67	37.83			
<b>Father smoker</b>									
Yes	88	39	<.0001	3.62	2.49	5.27	<.0001	0.101	0.067
No	250	13		1.41	1.25	1.60			
<b>Other smokers in household (not parent)</b>									
Yes	27	41	0.007	3.71	1.89	7.30	0.0381	0.020	0.116
No	338	19		1.79	1.55	2.06			
<b>Number of smokers in household</b>									
More than one smoker	35	71	<.0001	6.02	3.35	10.84	<.0001	0.170	0.107
One Smoker	90	42		3.37	2.32	4.89			
No smokers	238	10		1.29	1.15	1.43			
<b>Smoking ban in home – non-smoking household</b>									
Complete ban	227	9	0.175	1.28	1.14	1.43	0.8622	0.0001	0.124
Partial ban	9	22		1.24	0.89	1.74			
<b>Smoking ban in home – smoking household</b>									
Complete ban	106	34	<.0001	3.01	2.17	4.18	<.0001	0.127	0.176
Partial ban	20	85		16.73	8.32	33.64			

\*Breastfed infants of smoking mothers removed from this analysis.

**Table 2: Factors associated with the implementation of smoking restrictions in homes of infants\***

	<b>N</b>	<b>Complete Ban</b>		<b>p</b>
		<b>n</b>	<b>%</b>	
<b>Mother smoker</b>				
Yes	78	63	81	0.0007
No	323	301	93	
<b>No. cigarettes smoked per day by mother</b>				
≤10	32	28	88	0.007
11+	13	6	46	
<b>Mother's marital status</b>				
Single	29	26	90	0.729
Married/defacto	352	322	92	
<b>Mother's educational status</b>				
Year 10 or less	130	117	90	0.52
> Year 10	249	229	92	
<b>Father smoker</b>				
Yes	111	92	83	0.0005
No	260	245	94	
<b>Number of smokers in household</b>				
More than one smoker	59	45	76	<.0001
One Smoker	106	92	87	
No smokers	236	227	96	

\*Breast fed infants of smoking mothers included in this analysis.

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