Infant environmental tobacco smoke exposure following a smoking in pregnancy intervention programme

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ABSTRACT

Objective

To measure whether the cognitive, environmental and behavioural changes to household smoking reported in the *SmokeChange* programme reduced the amount of environmental tobacco smoke (ETS) exposure to infants aged six-months compared to controls.

Methods

A matched controlled trial as a subset of a clustered randomised controlled trial within Christchurch, known as the *SmokeChange* trial, was used. ETS exposure was assessed using a structured questionnaire, infant hair nicotine and infant urine cotinine creatinine ratio (CCR) measurements.

Results

Eighty-five infants were enrolled in the study; 36 (42%) *SmokeChange* and 49 (58%) controls. Median levels of hair nicotine were 3.6ng/mg (Q_1=1.8ng/mg, Q_3=10.6ng/mg) for *SmokeChange* infants and 3.3ng/mg (Q_1=1.7ng/mg, Q_3=5.4ng/mg) for controls (P=0.48). Median CCR levels were 93.5ng/mg (Q_1=18.7ng/mg, Q_3=416.0ng/mg) for *SmokeChange* infants and 119.0ng/mg (Q_1=13.9ng/mg, Q_3=311.7ng/mg) for controls (P=0.67). Compared to control households, fewer *SmokeChange* households reported usually making efforts to protect their infants from ETS exposure over the last week (98% vs 83%, P=0.04) or since infants' birth (100% vs 92%, P=0.07). After adjusting for confounders, no significant difference was seen between treatment groups for either biochemical measure.

Conclusions

ETS protection strategies adopted by the intervention group did not reduce infants' biochemically measured ETS exposure compared to control infants.

Key words

Environmental tobacco smoke (ETS) exposure, infant, intervention

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Introduction

Environmental tobacco smoke (ETS) exposure is unacceptably high,[1] unquestionably deleterious to infant health,[2,3] and increases health services use and cost.[4] Despite this, there is a dearth of studies in the literature targeting ETS reduction among children.[1]

Commencing in October 1995, a programme called *SmokeChange* was trialled within Christchurch.[5] The programme was designed to provide personalised counselling to smoking pregnant women, matched to individual readiness for change. The emphasis of this programme was to encourage sustainable reductions in smoke consumption, with an ultimate aim of cessation, and to motivate cognitive, environmental and behavioural changes to smoking that would protect the infant.[5] The majority of intervention mothers reported substantial reductions in their cigarette

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consumption, and positive changes in the smoking habits and smoking environments of their families.[5] With the on-going education provided by the *SmokeChange* programme to mothers of infants aged up to six-months, it was opined that these infants would have a reduced exposure to ETS when compared to infants of mothers with similar smoking levels outside the programme.

To accurately ascertain ETS exposure, objective biochemical measurements such as hair nicotine[6,7] and urinary cotinine[8] measurements have been preferred. The therapeutic benefits of counselling mothers on their children's exposure to ETS has been previously demonstrated in randomised controlled trials using such biochemical measurements.[9,10] However, these results appear largely due to significant decreases in smoke consumption of members within the household for those in the intervention group compared to their control group counterparts. This raises the important question, for a given level of smoking within a household, how well can behaviour modifications and strategies reduce the amount of ETS to children (e.g. opening windows, smoking in separate rooms or outdoors, using air purifiers)?[1] Currently, this question remains unanswered in the literature.

In this study we report the level of biochemically validated passive smoking exposure in six-month-old infants of smoking mothers given regular *SmokeChange* advice during pregnancy and postnatally compared to a control group of mothers, matched for smoking levels at the infant's birth, receiving standard antenatal and postnatal services.

Methods

Study design

The full *SmokeChange* study has been described in detail elsewhere.[5] Briefly, general practices in Christchurch were randomly assigned to the *SmokeChange* intervention (n=30) and the control arm (n=30) of the trial. Between October 1995 and September 1997, general practitioners (GPs) were to register all smoking pregnant women that they consulted, seek patient consent to participate in the study and issue a short questionnaire containing basic socio-demographic and smoking details. Those in the intervention arm were invited to participate in *SmokeChange* programme if they responded with 'yes' or 'unsure' to any of five questions for assistance with smoking cessation, namely: *'Would you like to learn how to (1) have a smokefree home? (2) smoke less? (3) stop smoking completely? (4) continue to stay smokefree? and (5) the* SmokeChange *programme can help with all of these. Would you like to find out more?'* The intervention, delivered by the *SmokeChange* Educators, was provided cost free and undertaken within the home using a four to seven visit protocol. Women were enrolled early in pregnancy (median gestation 10 weeks: Q_1=7 weeks, Q_3=15 weeks) and participated in the programme at their discretion until their baby was six months of age. Women in the control arm of the trial received standard antenatal and postnatal care.

The majority of intervention mothers reported substantial reductions in their cigarette consumption, and positive changes in the smoking habits and smoking environments of their families

The *SmokeChange* Educators undertook an intensive two-week training course to develop a working knowledge of the Stages of Change model,[11] motivational interviewing,[12] educational and motivational strategies for influencing change and community orientation. Training continued with weekly sessions throughout the study period.

Participants

For this study, we investigated a subset of participants enrolled in full *SmokeChange* trial.

Power calculations: A two group χ^2 test with an α=0.05 two-sided significance level has 75% power to detect the difference between the estimated proportion of control infants exposed to ETS of 0.45[13] and the estimated proportion of intervention infants exposed to ETS of 0.2 for a sample size of 48 in each group. To allow for a small drop-out rate, the sample size was rounded up to 50.

Intervention group: A consecutive sample of all women enrolled within the *SmokeChange* intervention group giving birth to a live infant after 1 June 1997 was invited to participate in this secondary ETS assessment study when their infants were six months old until consent was obtained from 50 mothers.

Control group: Comprised of infants drawn from the control arm of the *SmokeChange* trial who had a birth between 1 June 1997 and 31 December 1997. Controls infants were matched to intervention infants for maternal smoking at the end of pregnancy, the number of household smokers and mother's ethnicity. Mothers of matched control infants were approached to participate in this study until 50 consents were received.

Main outcome measures

ETS questionnaire: A brief assessment questionnaire on the extent of smoking in the household and environmental tobacco exposure to the infant was undertaken when the baby was six months old, administered by the research nurse. Questions included: cigarette consumption (categories: 0, 1–5, 6–10, 11–15, 16–20, 20+) when first pregnant, at the end of pregnancy and over the last week; the number of usual residents that smoked over the last week; whether the house and car was smokefree since birth and over the last week;

and whether efforts were usually made to protect infants from ETS when smoking inside (doors closed, window open, smoke in different room) since birth and over the last week. Infant feeding details were collected by asking how the mother was feeding her baby over the last week (categories: breast only, mainly breast, mainly bottle, bottle only).

Hair nicotine concentration: Hair segment analysis has shown that environmental nicotine can be incorporated into hair through the hair bulb via inhalation and by possible adsorption to the outside of hair. Also, maternal nicotine is transferred to the foetus through the placenta and retained in foetal hair. Once incorporated into hair, nicotine is permanently fixed thus providing a permanent record of long-term smoke exposure. Hair has a reasonably uniform growth rate of approximately one centimetre per month, and so provides a historical long-term record of smoke exposure. Hair samples were harvested from behind the ear by the research nurse and stored in a paper envelope at room temperature. Nicotine is stable under these conditions for many years. Nicotine assay was performed by High Performance Liquid Chromatography (HPLC) at Wellington Hospital and was calculated in standard units (ng/mg).

Cotinine creatinine ratio (CCR): Cotinine is a metabolic breakdown product of nicotine with a half-life of approximately one day, although this half-life is longer in non-smokers such as infants. Cotinine is concentrated in the urine by the kidney and so becomes a sensitive indicator of ETS exposure over the last few days. Urine creatinine measurements are then used to adjust for urine concentration. The urinary cotinine creatinine ratio (CCR) measurement has become a common method for measuring the levels of short-term ETS exposure. Urine samples were collected in a plastic specimen bag and transferred to a sterile pottle and stored at -20°C until analysis. Urinary cotinine assay was performed using the SCT Diagnostics Cotinine ELISA technique (SolarCare Technologies Corporation, 1745 Eaton Avenue, Bethlehem, PA 18018-1799, USA) at Medlab South Laboratories. Urine creatinine was also measured at this laboratory, using a BM 747 analyser. The cotinine creatinine ratio (CCR) was calculated in standard units (ng/mg).

Analysis: Due to the skewed nature of the continuous variables, medians and quartiles (Q_1, Q_3) were reported, and the Kolmogorov-Smirnov (KS) non-parametric test was used to compare groups. Categorical variables were compared using Fisher's exact test. Generalised Linear Models (GLM) were employed on logarithmic transformed hair nicotine and CCR values in multivariable comparisons. An α-level of 5% was considered statistically significant for all comparisons.

Ethical approval was obtained from the Southern Regional Health Authority Ethics Committee. Written consent was obtained from all mothers who participated in the trial.

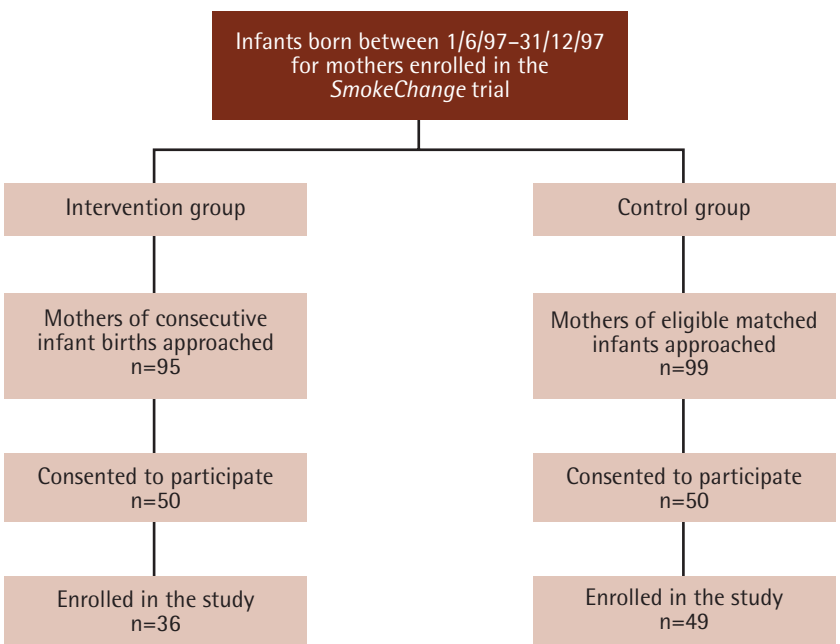
Results

Some 95 *SmokeChange* mothers were approached before consent was received from 50 (53%). Of the control infants born within the study period, mothers of 99 matched infants were approached before consent was received by 50 (51%) women. The process of recruitment is presented in Figure 1.

Overall, 85 infants were enrolled into the study; 36 (42%) *SmokeChange* and 49 (58%) controls. Table 1 includes a breakdown of the socio-demographic and baseline smoking characteristics of the groups.

Due to the matching, there was no difference between maternal smoking at the end of pregnancy, the number of household smokers and ethnicity between groups. No difference emerged between the two groups with respect to the rate of breastfeeding (P=0.48), a recognised confounder of CCR, or the proportion holding a Community Services card (P=1.00), a measure of socioeconomic status. However, the maternal age was significantly different between groups (P=0.03), with controls tending to be older.

Figure 1. Process of recruitment of infants into the matched controlled trial substudy of the clustered randomised controlled SmokeChange *trial.*



Questionnaire elicited smoking variables

A comparison of the recent smoking consumption of mothers and usual household residents, and two summary measures of ETS prevention made over the last week and since birth between groups is presented in Table 2.

No difference emerged between groups for maternal cigarette consumption, the number of other usual household residents who smoked and whether the home and car was smokefree over the last week and since the baby's birth. However, *SmokeChange* households were reported as making significantly less effort to protect their infants from ETS over the last week (P=0.04).

Hair nicotine

Hair nicotine samples were available from 63 (74%) infants. Figure 2 depicts a box-plot of the infant hair nicotine (ng/mg) results partitioned by the two groups.

Measurements were available from 26 (72%) *SmokeChange* infants, with median 3.6ng/mg (Q_1=1.8ng/mg, Q_3=10.6ng/mg), and 37 (76%) control infants, with median 3.3ng/mg (Q_1=1.7ng/mg, Q_3=5.4ng/mg), a difference in hair nicotine that was not significant between groups (KS=0.22, P=0.48).

GLM analyses were conducted to investigate whether adjusted infant hair nicotine levels were different between groups and to determine whether the elicited parental ETS prevention strategies since birth affected infants' measured hair nicotine levels, after controlling for mothers' smoking consumption, mothers' age and the number of usual resident smokers. As most, 33 (92%), *SmokeChange* parents and all, 49 (100%), control parents indicated that efforts were usually made to protect baby from ETS since birth (see Table 2), we only investigate the variable corresponding to whether the house and car had been smokefree since infant's birth. Complete data was available for 63 (74%) infants. Infants' logarithmic transformed hair nicotine values were significantly related to their mothers' level of smoking (P=0.02 for 1–10 cigs/day; P=0.005 for 11–20 cigs/day; P=0.003 for >20 cigs/day compared to non-smoking mothers) and two or more other usual resident smokers (P<0.001 compared to no other usual resident smokers), but there was no difference between groups (P=0.39) or smokefree houses and cars (P=0.93). This GLM model explained 39% of the variability in the data.

Table 1. Socio-demographics and matched smoking related characteristics of the SmokeChange *(n=36) and Control (n=49) groups.*

	SmokeChange		Control		
	n	(%)	n	(%)	P-value†
Maternal age (years)*					
<20	2	(6)	2	(4)	0.03
20–24	13	(36)	8	(17)	
25–29	13	(36)	12	(26)	
30–34	4	(11)	19	(40)	
≥35	4	(11)	6	(13)	
Maternal ethnicity					
Maori	4	(11)	8	(16)	0.55
Non-Maori	32	(89)	41	(84)	
Community Services Card					
Yes	22	(61)	29	(59)	1.00
No	14	(39)	20	(41)	
Method of infant feeding					
Exclusively or mainly breast	9	(25)	16	(33)	0.48
Exclusively or mainly formula	27	(75)	33	(67)	
Maternal smoking at the end of pregnancy (cigs/day)					
0	4	(11)	6	(12)	1.00
1–10	21	(58)	27	(55)	
11–20	9	(25)	13	(27)	
>20	2	(6)	3	(6)	
Number of household smokers (excluding mother) at the end of pregnancy					
0	17	(47)	22	(45)	0.94
1	14	(39)	21	(43)	
2	4	(11)	4	(8)	
≥3	1	(3)	2	(4)	

* Maternal age was not provided by two control mothers.
† P-values calculated using Fisher's exact test.

Cotinine creatinine ratio (CCR)

Urine samples were available for analysis from 81 (95%) infants. A box-plot of infants CCR (ng/mg) results separated by the two groups appears in Figure 3.

Measurements were available from 34 (94%) *SmokeChange* infants, with median 93.5ng/mg (Q_1=18.7ng/

mg, Q_3=416.0ng/mg), and 47 (96%) control infants, with median 119.0ng/mg (Q_1=13.9ng/mg, Q_3=311.7ng/mg). No significant difference emerged between the distributions of infant CCR values between groups (KS=0.1, P=0.67). However, CCR is confounded by infants' feeding status, with breastfeeding infants having CCR values, median 508.7ng/mg (Q_1=87.4ng/mg, Q_3=1371.4ng/mg), significantly greater than their formula fed counterparts, median 79.3ng/mg (Q_1=13.9ng/mg, Q_3=147.6ng/mg), (KS=0.51, P<0.001). Examining infants' logarithmic transformed CCR values, after adjusting for infants' feeding status, failed to show any difference between treatment groups (P=0.94).

GLM analyses were conducted to investigate whether adjusted infant CCR levels were different between groups and to determine whether the elicited parental ETS prevention strategies over the last week affected infants' measured urine CCR levels, after controlling for mothers' smoking consumption, mothers' age, the number of usual resident smokers and infants' feeding status. Again, due to the preponderance of parents declaring that efforts were usually made to protect baby from ETS over the last week (see Table 2), we only investigate the variable corresponding to whether the house and car had been smokefree over the last week. Complete data was available for 79 (93%) infants. Infant's logarithmic transformed CCR values were significantly related to their mother's level of smoking (P<0.001 for 1–10 cigs/day; P<0.001 for 11–20 cigs/day; P<0.001 for >20 cigs/day compared to non-smoking mothers), two or more other usual resident smokers (P=0.05 compared to no other usual resident smokers) and infant feeding status (P<0.001), but there was no difference between groups (P=0.45) or smokefree houses and cars (P=0.22). This second GLM model explained 53% of the variability in the data.

Table 2. A comparison of questionnaire elicited short-term and long-term maternal and household smoking environment of six-month infants between SmokeChange *and control groups.*

	SmokeChange		Control		
	n	(%)	n	(%)	P-value†
Maternal smoking over the last week (cigs/day)					
0	2	(6)	8	(16)	0.26
1–10	14	(39)	18	(37)	
11–20	13	(36)	19	(39)	
≥20	7	(19)	4	(8)	
Number of other usual household residents who smoked over the last week					
0	17	(47)	22	(45)	0.95
1	14	(39)	21	(43)	
≥2	5	(14)	6	(12)	
Efforts usually made to protect baby from ETS over the last week					
Yes	30	(83)	48	(98)	0.04
No	6	(17)	1	(2)	
Home and car smokefree over the last week					
Yes	20	(56)	22	(45)	0.38
No	16	(44)	27	(55)	
Efforts usually made to protect baby from ETS since birth					
Yes	33	(92)	49	(100)	0.07
No	3	(8)	0	(0)	
Home and car smokefree since baby's birth					
Yes	14	(39)	19	(39)	1.00
No	22	(61)	30	(61)	

† P-values calculated using Fisher's exact test.

Discussion

Using two validated biochemical indicators of ETS exposure, hair nicotine,[6,7] as an indication of long-term exposure (the last few months), and urinary CCR,[8] as an indication of short-term ETS (the last few days), we found no evidence that women participating on the *SmokeChange* programme were any better than the control group, matched for maternal smoking at the end of pregnancy, the number of household smokers and ethnicity, in protecting their infant from ETS.

The overwhelming majority of *SmokeChange* and control parents reported that they usually made efforts to protect their infant from ETS since birth and over the last week. However, the majority of infants investigated were exposed to ETS, some to apparently very high doses. This suggests that adopted ETS avoidance techniques may be inadequate or the smoke exposure was under-reported.[14] One unexpected finding was that significantly fewer *SmokeChange* households, 30 (83%), usually made efforts to protect their infants from ETS over the last week than controls, 48 (98%), (P=0.04). Although not statistically significant, fewer *SmokeChange* households, 33 (92%), also usually made efforts to protect their infants from ETS since infants' births

compared to controls, 49 (100%), (P=0.07). This trend has not been observed in other intervention studies[1,9,13] and may be a chance finding. It might also be opined that the observed difference is a result of reporting bias due to *SmokeChange* mothers' differing level of honesty and knowledge in their reporting.

When considering the specific strategy of smoking outside the home and car, thereby keeping the house and car smokefree, no significant reduction in hair nicotine or CCR levels was observed. While perhaps surprising, this result is consistent with that reported in two previous studies.[15,16]

There are various weaknesses associated with the study that limits its findings. The generalisability of the study population is restricted by the 52% of maternal smokers in pregnancy who declined to participate in the *SmokeChange* trial. It is likely that those women who chose to enrol were more sensitised to the deleterious effects of tobacco smoke and, therefore, more motivated to instigate behaviours or practices that would reduce ETS exposure to infants. The weakness of self-reported questionnaires include recall bias, the reluctance of parents to admit to smoking,[17] and the influence on indoor smoke distribution on factors such as distance from the ETS source, time of exposure, room size, ventilation and air circulation. Smoking by multiple individuals, other than the parents, significantly contributes to the ETS exposure of infants.[18] Moreover, smoking in confined non-domicile places, such as a baby-sitters' or friends' houses, may have significant ETS exposure contributions. We also assumed that smoking levels, determined at the infants' birth, remained similar between the invention and control groups. However, if the invention group increased their smoking levels after the birth (as this group had decreased more during pregnancy), then any apparent decrease in measured ETS exposure might be masked by the differential level of smoke consumption.

Figure 2. Box-plot of measured infant hair nicotine (ng/mg) by SmokeChange *(n=26) and control (n=37) groups.*

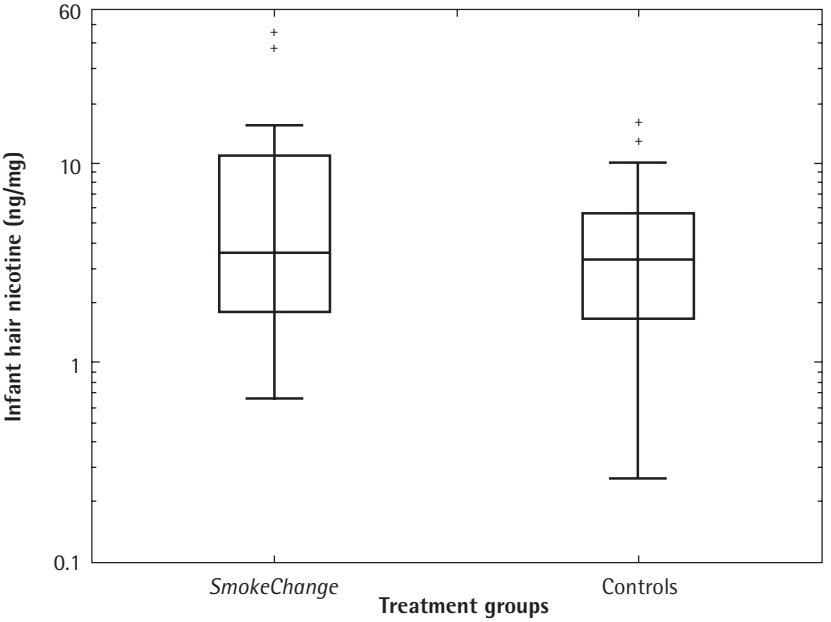
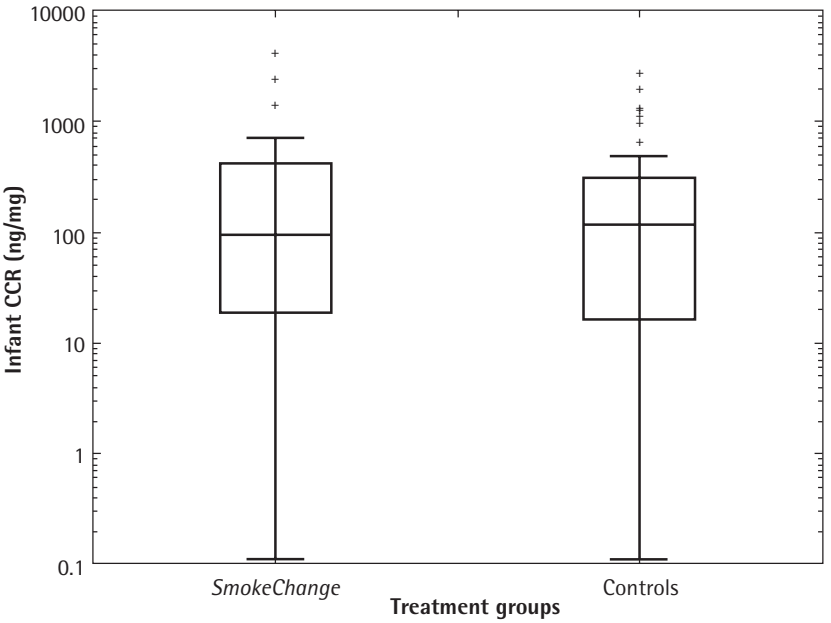


Figure 3. Box-plot of measured infant cotinine creatinine ratio (ng/mg) by SmokeChange *(n=34) and control (n=47) groups.*



While biochemical measures appear superior to questionnaire elicitations, they are not without problems.[19] Some parents declined infant hair harvesting, due to cultural beliefs or the scant availability of hair on some infants. There are other sources of nicotine exposure that might confound the biomarker measures of cotinine and nicotine. It has been reported that nicotine remains on hard surfaces and clothing for many hours, if not weeks.[6] Thus, in a home with smokers, it is quite likely

that the furniture, carpeting, crib, diapers and the fingers of the mother may be contaminated with nicotine. Physiological exposure can take place by hand to mouth practices common in infants and by contact and transdermal absorption or inhalation of contaminated dust.

Maternal smoking affects urine cotinine levels directly though breastmilk. Breastfed infants have tenfold higher concentrations than bottle-fed infants and potentially confound reported results.[20] Other problems with cotinine include its relatively short half-life of approximately one day and the considerable individual variations inherent within urine cotinine measurement. The process of measuring ETS is complex and fraught with difficulties; however, single hair nicotine levels appear reliable for epidemiological studies.[6,7]

It was anticipated that after intensive *SmokeChange* counselling that parents participating in the programme would be acutely cognisant of the effects of both active and passive smoking. While effective in reducing smoking in pregnancy, the *SmokeChange* intervention programme did not reduce ETS exposure to six-month aged infants.[5] Results from this and other similar studies demonstrate that protection from ETS is difficult.[12,16] For successful ETS protection post-partum, specific targeted approaches and proper evaluation must be developed. Only once a clear understanding of successful yet feasible strategies for ETS protection have been developed can these targeted approaches receive large-scale promotion. Because smoking individuals (other than the parents) significantly contribute to the ETS exposure in infants, efforts to reduce the adverse health effects of this exposure should extend beyond parental smoking.[18]

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