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Effect of a family-centered, secondhand smoke intervention to reduce respiratory illness in indigenous infants in Australia and New Zealand: a randomized controlled trial

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Abstract

Introduction: Secondhand smoke (SHS) is a significant cause of acute respiratory illness (ARI) and 5 times more common in indigenous children. A single-blind randomized trial was undertaken to determine the efficacy of a family centered SHS intervention to reduce ARI in indigenous infants in Australia and New Zealand. Methods: Indigenous mothers/infants from homes with ≥1 smoker were randomized to a SHS intervention involving 3 home visits in the first 3 months of the infants' lives (plus usual care) or usual care. The primary outcome was number of ARI-related visits to a health provider in the first year of life. Secondary outcomes, assessed at 4 and 12 months of age, included ARI hospitalization rates and mothers' report of infants' SHS exposure (validated by urinary cotinine/creatinine ratios [CCRs]), smoking restrictions, and smoking cessation. Results: Two hundred and ninety-three mother/infant dyads were randomized and followed up. Three quarters of mothers smoked during pregnancy and two thirds were smoking at baseline (as were their partners), with no change for more than 12 months. Reported infant exposure to SHS was low (≥95% had smoke-free homes/cars). Infant CCRs were higher if one or both parents were smokers and if mothers breast fed their infants. There was no effect of the intervention on ARI events [471 intervention vs. 438 usual care (reference); incidence rate ratio = 1.10, 95% confidence intervals (CI) = 0.88-1.37, p = .40]. Conclusions: Despite reporting smoke-free homes/cars, mothers and their partners continue to smoke in the first year of infants' lives, exposing them to SHS. Emphasis needs to be placed on supporting parents to stop smoking preconception, during pregnancy, and postnatal.

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Effect of a Family-Centered, Secondhand Smoke Intervention to Reduce Respiratory Illness in Indigenous Infants in Australia and New Zealand: A Randomized Controlled Trial

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Abstract

Introduction: Secondhand smoke (SHS) is a significant cause of acute respiratory illness (ARI) and 5 times more common in indigenous children. A single-blind randomized trial was undertaken to determine the efficacy of a family centered SHS intervention to reduce ARI in indigenous infants in Australia and New Zealand.

Methods: Indigenous mothers/infants from homes with ≥1 smoker were randomized to a SHS intervention involving 3 home visits in the first 3 months of the infants' lives (plus usual care) or usual care. The primary outcome was number of ARI-related visits to a health provider in the first year of life. Secondary outcomes, assessed at 4 and 12 months of age, included ARI hospitalization rates and mothers' report of infants' SHS exposure (validated by urinary cotinine/creatinine ratios [CCRs]), smoking restrictions, and smoking cessation.

Results: Two hundred and ninety-three mother/infant dyads were randomized and followed up. Three quarters of mothers smoked during pregnancy and two thirds were smoking at baseline (as were their partners), with no change for more than 12 months. Reported infant exposure to SHS was low (≥95% had smoke-free homes/cars). Infant CCRs were higher if one or both parents were smokers and if mothers breast fed their infants. There was no effect of the intervention on ARI events [471 intervention vs. 438 usual care (reference); incidence rate ratio = 1.10, 95% confidence intervals (CI) = 0.88–1.37, $p = .40$].
Conclusions: Despite reporting smoke-free homes/cars, mothers and their partners continue to smoke in the first year of infants' lives, exposing them to SHS. Emphasis needs to be placed on supporting parents to stop smoking preconception, during pregnancy, and postnatal.

Introduction

In Australia and New Zealand (NZ), deaths and hospitalization among indigenous children aged 0–4 years, due to acute respiratory illness (ARI), is higher compared with nonindigenous children ([1–3]). Secondhand smoke (SHS) exposure is the most modifiable risk factor for ARI among these populations. The effect of parental smoking on the frequency and severity of respiratory illness, asthma, otitis media, and chronic middle ear effusion is strongest in younger children ([4–11]). The World Health Organization has prioritized the need to educate parents about the impact of SHS on children's health (12), especially among populations with high smoking rates. Indigenous Australians and NZ Māori are twice as likely to smoke as their nonindigenous counterparts ([13,14]), and thus indigenous children in these countries have greater SHS exposure ([3,15,16]).

A systematic review of 36 randomized trials that investigated family/carer tobacco control programs for reducing children's exposure to SHS found insufficient evidence to support any one intervention ([17]). However, a modest effect was found for intensive parental counseling that focused on changing attitudes and behaviors, premised on behavior change theory ([18]), as opposed to changing knowledge alone. More recent trials support this finding ([19,20]). Qualitative research on smoking in remote Northern Territory Aboriginal communities has found that indigenous parents/carers are concerned about the health effects of SHS ([21,22]). In addition to positive role modeling, the health of their children is their primary motivation to quit ([23]). NZ Māori similarly stop smoking for the benefit of their children ([24,25]). These data suggest that a family-based SHS intervention that focuses more on the welfare of children as opposed to adults stopping smoking may be particularly salient among these two indigenous populations. Thus, we undertook a randomized trial with blinded outcome assessment to determine the impact of a culturally appropriate, family-centered SHS program (where we involved as many extended family members as possible and focused on strategies for reducing SHS exposure in children, which included positive role modeling, within family support and smoking cessation support for all) on the respiratory health of indigenous infants in Australia and NZ. We hypothesized that such a program would reduce the number of ARI-related visits to a health provider in the first year of infants' lives.

Methods

Participants

The rationale and methodology for this trial has been reported previously ([26]) and is summarized below. Between December 2009 and January 2012, Community Workers (CWs) in Darwin, Australia, and Auckland, NZ, approached potentially eligible mothers through antenatal clinics and hospital birth records. Mother/infant dyads were eligible if (a) the infant was aged between 0–5 weeks; (b) the mother self-identified as Māori or Australian Aboriginal/Torres Strait Islander; (c) the mother was aged ≥16 years; (d) the mother was a current smoker or the infant lived in a household where there was at least one other smoker; (e) the mother resided permanently with the infant in Darwin/Greater Darwin area of Australia or within the Counties Manukau District Health Board region, NZ; (f) the infant was a singleton or the first born in a multiple pregnancy delivery; and (g) the mother spoke English and/or Māori. Ethics approval was obtained from the Menzies Human Research Ethics Committee (Australia) and the Northern Region Human Ethics Committee (NZ).

Randomization and Blinding

Eligible and interested mothers were consented and mother/infant dyads were randomized in a 1:1 ratio to one of two arms by central computer, using blocked randomization stratified by country (Australia, NZ). Participants were not blinded to treatment allocation, but research staff assessing the primary outcome were blinded.

Intervention

The control group received "usual care" comprising standard management by hospital and primary care providers, which ranged from brief quit advice to the provision of cessation treatment. In the intervention group, all mothers (and family members that were present) who smoked received usual care plus behavioral "coaching" about the dangers of SHS exposure to children, commitment to smoking restrictions in the home/car, positive role modeling, and strategies for overcoming obstacles to making smoke-free changes. Those who smoked were also given either brief advice or more intensive counseling to quit (depending on how receptive they were) and offered free nicotine replacement therapy (NRT; 21 mg patches and/or 4 mg gum) and/or a Quitline referral, unless it became clear as part of the conversation with them that they were not interested in such options. The program was founded on Māori and Aboriginal holistic models of health ([24,27,28]) and was delivered by CWs (who were mainly indigenous and received identical training in motivational interviewing and program delivery) through three face-to-face home visits conducted over the first 3 months of the infants' lives. Both groups received brief health promotion messages (focused on immunization, infant nutrition/breast feeding, and safe sleeping for baby) from the CWs at baseline and when the infants were 4 and 12 months of age.

Outcomes

Baseline data were collected through a face-to-face visit at the mothers' homes, as close as possible to 6 week after the infants' birth. Baseline measures are described in detail in the published protocol ([26]). In the intervention group, at baseline, 2 months, and 3 months, a mix of

quantitative and qualitative process evaluation indicators was collected related to the program delivery, including the amount of the program that was delivered, commitment to smoke-free changes, and parent satisfaction. No information was collected on usual care practices.

Outcome data were collected at 4 and 12 months of age via a face-to-face visit at mothers' homes. The primary outcome was rate of health provider presentations for new primary episodes of ARI in the first year of life, obtained from the mothers and confirmed by two study clinicians in each country (blinded to treatment allocation), who reviewed the infants' health provider and hospital clinic records. In Australia, 20 participants had their primary outcome data reviewed by clinicians in NZ, in order to assess intercountry reliability. Secondary outcomes included rate of hospitalizations for ARI, mothers' breast feeding status ([29]) and self-report of smoking restrictions in the home ("Is smoking ever allowed inside the house?"—reported as Yes/No, but multiple options were allowed, e.g., Yes—but restricted to certain rooms only, Yes—in any room but only when the infant is not there) and car ("Is smoking allowed inside any car when your infant is in it?"—Yes/No option). Data were also collected on the mothers' report of their infants' exposure to SHS in the last 7 days, specifically whether the infant had been near (within arm's length) people smoking cannabis or an open fire used for cooking/heating or a camp fire, whether they had been around tobacco smoke (e.g., in the same room in a house as someone that was smoking, in a car with someone that was smoking, or sitting outside within arm's length of someone who was smoking), and whether they had been cared for in other houses or childcare where people smoked. Mothers' self-report of a quit attempt was also asked, defined as not smoking a cigarette for ≥24 hr, as was the presence of day- and nighttime coughing by the infant over the last 2 days (using verbal category descriptive scores from 0–5, as per Chang *et al.* ([30]).

To confirm SHS exposure in the last 2–3 days, a single urine sample was collected from each infant ([31]) at baseline, 4 months, and 12 months of age ([26]). Samples were tested for urinary cotinine and creatinine using gas chromatography/mass spectrometry. Results are expressed as the cotinine/creatinine ratio (CCR, ng/mg), with values of ≥30 ng/mg indicating that the infant was exposed to SHS (sensitivity 80%, specificity 100%) ([32]).

Sample Size/Analyses

A study of disease burden and clinic attendances for young indigenous children in two remote Northern Territory communities found the median number of presentations for upper respiratory illness in the first year of life was 7.5 (interquartile range 4–11) and for lower respiratory illness, 2.5 (interquartile range 1–5) ([33]). Our clinical experience with infants and limited data from children suggested that there would be much fewer episodes of ARI in urban compared with remote settings ([34]). Thus, it was estimated that an average of three visits per year would occur in the control group. Each population had a sample size estimate of 210 mother/infants dyads, which provided 90% power ($p = .05$) to detect a 25% reduction in new episodes of ARI in the intervention group compared with the usual care group (based on three health provider visits per year in the usual care group and 2.25 visits in the intervention group), assuming a Poisson distribution and a 10% loss to follow-up. Combining data from the two countries ($n = 420$) provided 90% power ($p = .05$) to detect an 18% reduction in the primary outcome.

All analyses were undertaken using SAS Version 9.3. Complete case analysis was undertaken, and sensitivity analyses using a modified intention-to-treat (ITT) approach (excluding those randomized but who did not enter the protocol) with a multiple imputation method (using 50 imputations) applied to the missing data ([35]) and analyses adjusted for potential confounding factors. The incidence rate for ARI between the two groups was analyzed using negative binomial regression as there was evidence of overdispersion. The incidence rate ratio (IRR) and 95% confidence intervals (95% CI) were reported (with usual care as the reference). Dichotomous outcomes were compared using chi-square tests and continuous outcomes were compared using *T*-tests or Mann–Whitney tests. Due to the skewed nature of the CCR data, they were log transformed and presented as geometric means. The difference between groups in log-transformed CCR are presented as a ratio of geometric means (95% CI) and adjusted (using linear regression) for infants' birth weight and baseline measures of CCR, mothers' age, education, smoking status, breast feeding status, and crowding index. For log-transformed values that were not significantly different, the 95% CI of the ratio included 1. Intra-rater and intercountry agreement for grading of the primary outcome assessment were assessed using the Kappa statistic (unweighted).

Results

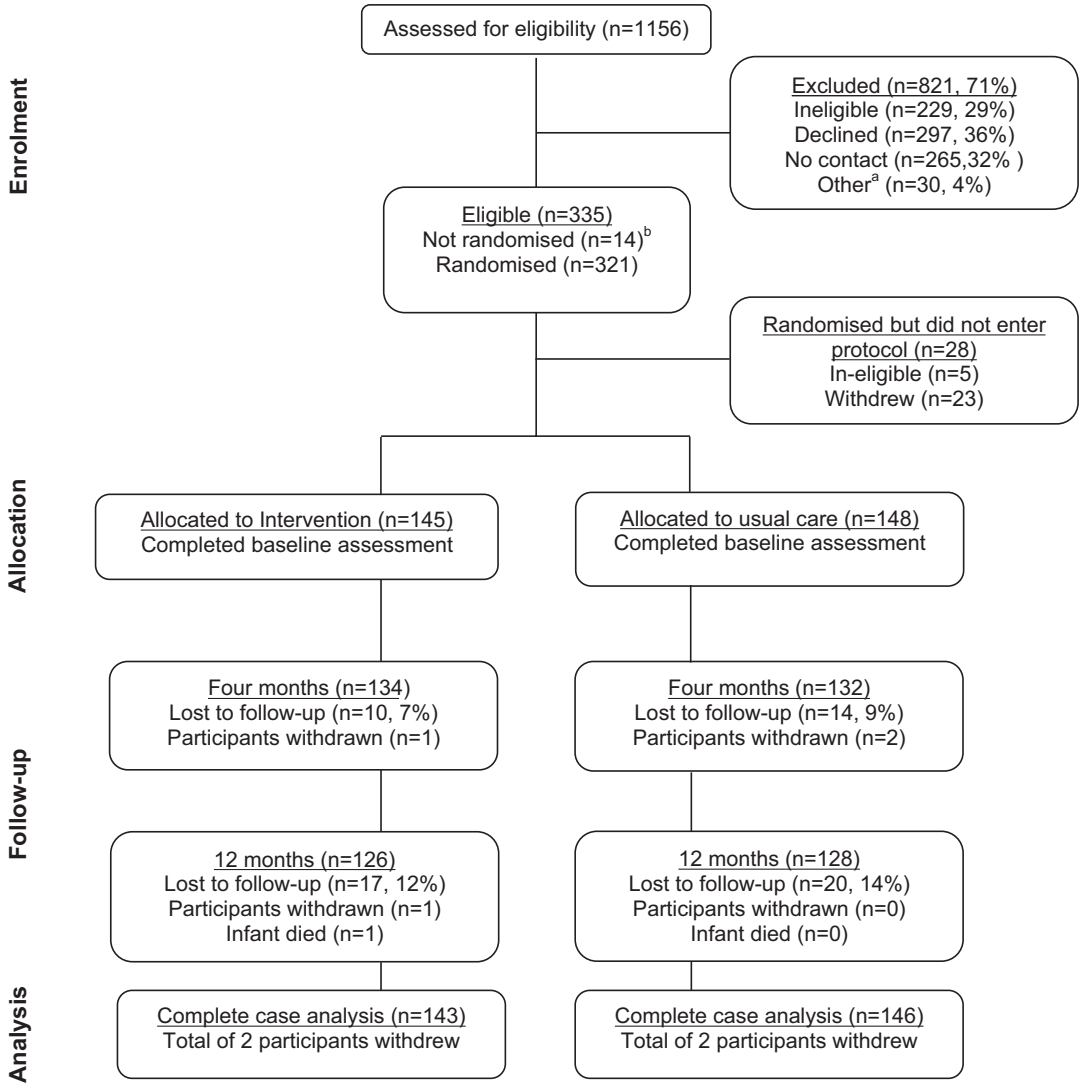
Overall, 228 mother/infant dyads were recruited in NZ (115 intervention, 113 usual care) and 93 in Australia (46 intervention, 47 usual care). Recruitment took 14 months in NZ and stopped at 28 months in Australia due to not reaching the recruitment target within the budgeted timeframe. More potential participants in Australia declined to participate (56%) than those in NZ (31%, $p < .001$), and more were noncontactable in NZ (39% NZ vs. 7% Australia, $p < .001$). Rates of withdrawal and ineligibility after randomization and before baseline assessment did not differ between country or by treatment group (Figure 1).

A total of 293 participants were available for follow-up, with a total 12-month loss-to-follow-up (including withdrawal and death) of 13% (39/293) and with no significant difference by treatment group, country, or their interaction. No significant differences in baseline data between the two countries were found, and thus the two data sets were combined (Table 1), with results presented hereafter.

In the intervention group at baseline, 2 months, and 3 months, all but 2, 8, and 17 households, respectively, received the intervention. Not all parts of the program were implemented in every household, due to the mother and/or family members wanting to move the conversation on or stop the interview. Across the baseline, 2-month and 3-month visits, only 2%–7% of the mothers/family members failed to agree to smoking restrictions inside the home and car.

Primary Outcome

The rate of health provider presentations for new primary episodes of ARI in the first year of life did not differ significantly between the groups (intervention: 471 events; usual care: 438 events; IRR = 1.10, 95% CI = 0.88–1.37, $p = .40$). Results were similar for ITT analysis (IRR = 1.10, 95% CI = 0.88–1.36, $p = .41$) and after adjusting for infants' birth weight, mother's age at baseline, education, smoking status, breast feeding status, and crowding index (IRR = 1.07, 95% CI = 0.86–1.34, $p = .53$). No differences in the primary outcome were found according to new episodes of upper respiratory tract infection (intervention: 315 events; usual care: 278 events; IRR = 1.16, 95% CI = 0.92–1.46, $p = .22$),



[a] One infant died at birth, four were placed into care/adopted, 12 mothers were excluded due to socio-emotional issues (e.g. Domestic violence, substance abuse etc) and 13 did not proceed in Australia after recruitment was stopped.

[b] We do not know why the 14 women/infants (all from New Zealand) did not go onto to get randomised, despite being eligible.

Figure 1. Flowchart of recruitment and retention of participants throughout the trial (New Zealand and Australia combined).

lower respiratory tract infection (intervention: 147 events; usual care: 167 events; IRR = 0.90, 95% CI = 0.65–1.25, p = .53), otitis media (intervention: 105 events; usual care: 95 events; IRR = 1.13, 95% CI = 0.74–1.73, p = .58), or rate of hospitalizations for ARI (intervention: 53 events; usual care: 44 events; IRR = 1.23, 95% CI = 0.70–2.15, p = .47). Between country, interrater agreements for lower respiratory tract infection, upper respiratory tract infection, and otitis media were high (κ = 0.84, 0.78, and 0.79, respectively).

Secondary Outcomes

Mothers' Report on Infants' Exposure to SHS

Three quarters of mothers smoked during their pregnancy, and two thirds were current smokers at baseline (Table 1). No significant change in smoking prevalence and intensity was seen by group over the first year of the infants' lives (Table 2). Of the 238 mothers (80%) who had a partner at baseline, 164 (69%) reported that their partner smoked, with no difference in this proportion by group or over time (Table 2). The geometric mean CCRs were significantly higher if

Table 1. Baseline Characteristics of Infant and Mother (New Zealand and Australia Combined)

Variables	Intervention group, *N* = 145 (%)	Usual care group, *N* = 148 (%)
Infants: female	58 (40)	68 (46)
Infants: mean age at baseline, weeks (*SD*)	6.3 (2.7)	6.0 (2.7)
Infants: country		
New Zealand	108 (74)	108 (73)
Australia	37 (26)	40 (27)
Infants: mean gestational age at birth, weeks (*SD*)	39.3 (1.3)	39.3 (1.5)
Infants: mean birth weight, kilograms (*SD*)	3.3 (0.5)	3.3 (0.6)
Infants: unwell since birth[a]	44 (30)	36 (24)
Infants: coughing		
No daytime cough	111 (77)	119 (80)
No nighttime cough	123 (85)	128 (87)
Mothers: mean age at baseline in years (*SD*)	26.8 (6.5)	25.3 (5.8)
Mothers: highest level of education		
≤Secondary school	104 (72)	114 (77)
TAFE/polytechnic/university	41 (28)	34 (23)
Mothers: marital status[b]		
Married/defacto/living with partner	72 (50)	91 (62)
Divorced/separated/widowed	8 (6)	20(14)
Never married	44 (30)	25 (17)
Refused to answer[c]	21 (15)	12 (8)
Mothers: breast feeding state		
Yes—exclusive	43 (30)	49 (33)
Yes—full	29 (20)	31 (21)
Yes—partial	42 (29)	40 (27)
No	31 (21)	28 (19)
Mothers: smoked during pregnancy	114 (79)	105 (71)
Mothers: reduced amount smoked during pregnancy[e]	74 (65)	71 (68)
Mothers: current smoking status		
Current smoker	105 (72)	88 (60)
Ex-smoker	22 (15)	37 (25)
Never smoked	18 (12)	23 (16)
Mothers: frequency of smoking[f]		
At least weekly	95 (90)	83 (94)
Less than weekly	10 (10)	5 (6)
Mothers: number of cigarettes smoked per day[f]		
≤10	54 (51)	63 (72)
11–20	38 (35)	20 (23)
21–30	6 (6)	2 (2)
≥31	1 (1)	1 (1)
Missing data	6 (6)	2 (2)
Mothers: time to first cigarette[f]		
≤30 min of waking	48 (49)	31 (37)
>30 min of waking	50 (51)	53 (63)
Mothers: quit attempt in last 12 months[f]	49 (47)	44 (50)
Mothers: mean self-efficacy score (*SD*)[g]	3.3 (0.8)	3.3 (0.8)
Household: mean crowding index (*SD*)[d]	2.0 (0.7)	2.0 (0.7)
Household: mean number of children in house aged under 5 years (*SD*)	1.9 (1.0)	1.9 (1.0)

SD = standard deviation; TAFE = Technical and Further Education Institution.

[a]The specific question was "Has your infant been unwell and needed to go to the health clinic, general practitioner or hospital since he/she was born?"

[b]X^2; $p = .002$.

[c]All but three participants were from New Zealand. In New Zealand, different levels of social support are offered depending on marital status. It is likely that some women in the study did not wish to disclose their marital status, in case their access to certain social support was jeopardized.

[d]Defined as the number of people currently sleeping in the house divided by the number of rooms in the house where people were sleeping

[e]In those that smoked during pregnancy.

[f]In current smokers.

[g]Belief in their ability to quit this time, measured on a scale of 1–5, where one was very low and five was very high.

parents smoked (mother and partner smoked: 365 ng/mg; mother only smoked: 245 ng/mg; partner only smoked: 27 ng/mg) compared with neither parent smoking (25 ng/mg; $p < .0001$). Breast feeding rates were high at baseline, with this proportion declining over time and no difference noted by group (Table 2). Mean CCRs were significantly higher at baseline and 4 months in those mothers breast feeding their infants compared with artificial feeding (both $p < .001$), with no difference at 12 months ($p = .09$; Figure 2). Overall, mean CCRs declined

Table 2. Secondhand Smoke Exposure Over Time (New Zealand and Australia Combined)

	Baseline		4 Months		12 Months	
	Intervention, *n*/*N* (%)	Usual care, *n*/*N* (%)	Intervention, *n*/*N* (%)	Usual care, *n*/*N* (%)	Intervention, *n*/*N* (%)	Usual care, *n*/*N* (%)
Full smoking ban in home	136/145 (94)	141/148 (95)	125/134 (93)	123/132 (93)	117/126 (94)	120/128 (95)
Smoking ban in car	140/145 (97)	143/148 (97)	130/134 (97)	129/132 (98)	119/126 (95)	123/128 (97)
Mother is current smoker	105/145 (72)	88/148 (60)	94/134 (70)	78/132 (59)	83/126 (66)	70/128 (55)
Mother smokes ≤20 per day	92/105 (88)	83/88 (94)	87/94 (93)	74/78 (95)	73/83 (88)	64/70 (91)
Partner is current smoker[a]	83/116 (72)	81/122 (66)	75/109 (69)	72/108 (67)	59/99 (60)	72/109 (66)
Infant is breast fed[b]	114/145 (79)	120/148 (81)	83/134 (62)	87/132 (66)	34/126 (27)	34/128 (27)
Members of the household smoke inside	13/145 (9)	11/148 (7)	10/134 (8)	13/132 (10)	10/126 (8)	7/128 (5)
In last seven days, infant has been cared for in another place where people smoke[c]	16/145 (11)	26/148 (18)	10/134 (8)	11/132 (8)	13/126 (10)	10/128 (8)
In last seven days, infant has been around tobacco smoke[c, d]	17/145 (12)	20/148 (14)	14/134 (10)	10/132 (8)	23/126 (18)	15/128 (12)
In last seven days, infant has been near (within arm's length) of an open fire for cooking or heating or camp fire[c]	5/145 (3)	6/148 (4)	11/134 (8)	5/132 (4)	2/126 (2)	7/128 (5)
In last seven days, infant has been near (within arm's length) of people smoking cannabis[c]	0/145 (0)	3/148 (2)	3/134 (2)	1/132 (1)	0/126 (0)	1/128 (1)
Geometric mean cotinine/creatinine ratio (ng/mg)[e]	157.6	112.2	84.8	47.5	25.0	21.8

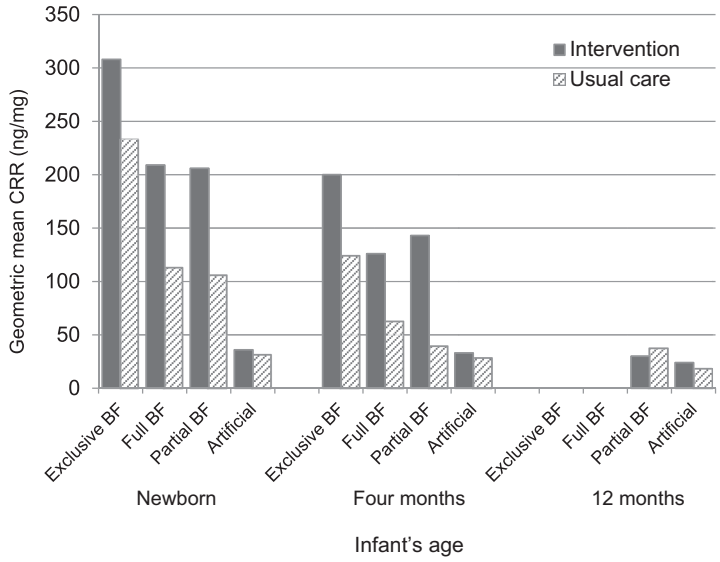
[a]In those mothers with partners.

[b]Defined as breast fed exclusively, fully, or partially.

[c]Defined as number of infants with ≥1 day of exposure.

[d]Defined as in the same room with someone that is smoking, in a car with someone smoking, or sitting outside within arm's length of someone who is smoking—includes when holding the infant.

[e]Readings of ≥30 ng/mg indicate secondhand smoke exposure.



BF: Breastfeeding

Exclusive BF: Baby has never received anything else apart from breast milk since birth, except for prescription medication; Full BF: Breast milk in the last 48 hours; Partial BF: Some breast milk and some formula or other food in the last 48 hours; Artificial: no breast milk in the last 48 hours.

Figure 2. Cotinine/creatinine ratio in infants' urine, according to feeding status over time.

over time to <30 ng/mg by 12 months of age, indicative of little SHS exposure (Table 2). After adjusting for baseline variables (including baseline CCR), there was no significant difference in mean CCRs between the groups at 4 months (ratio of geometric mean difference = 1.39, 95% CI = 0.99–1.94, p = .06) or 12 months of age (ratio of geometric mean difference = 0.97, 95% CI = 0.69–1.37, p = .87).

Smoking bans were reportedly well established with ≥95% smoke-free homes/cars at baseline, and mothers' report of infants' exposure to SHS in the last 7 days was relatively low. Both variables did not differ by group and did not significantly change over time (Table 2). Overall, in the last 7 days ≤18% of infants were potentially exposed to SHS through being cared for in other places where people smoke, being in the same room in a house with someone that was smoking, being in a car with someone that was smoking, or sitting outside within arm's length of someone who was smoking (Table 2). In the last 7 days, ≤8% of infants had been near an open fire for cooking/heating, or a camp fire, and ≤2% of infants had been near people smoking cannabis (Table 2).

Quitting Behavior

At baseline, almost half of the 193 mothers that were current smokers had tried to quit smoking in the last 12 months (Table 1). When asked about their belief in their ability to quit smoking this time, measured on a scale of 1–5, where one was very low and five was very high, mothers had a mean self-efficacy score of 3.3 (SD = 0.8; Table 1). Across the baseline, 2-month, and 3-month visits, 24%–30% of the mothers in the intervention group that smoked agreed to quit smoking (p = .55), 65%–87% were offered free NRT (70% acceptance at baseline, which was significantly more than at 2 and 3 months: 32% and 41%, respectively, p = .0004), and 20%–43% were offered Quitline referrals (2-month data were significantly greater than those at baseline and 1 month: p = .0006, all refused the offer at baseline and 2 months; 7% accepted the offer at 3 months). Follow-up of infants at 4 and 12 months of age showed no difference between the groups in mothers' quitting behavior over time. At 4 months, 24% (41/172) of the current smokers had made a quit attempt, and 33% (51/153) at 12 months. No difference in 7-day point prevalence abstinence for mothers was seen between the two groups when their infants were 4 months (n = 25, 19% intervention vs. n = 34, 26% usual care, p = .16) or 12 months of age (n = 22, 18% intervention vs. n = 33, 26% usual care, p = .10).

As part of the intervention program, other family members that smoked were also supported to quit smoking. When the infants were ~1 month old, in 37 families other members of the household (N = 58, mean = 1.6 people/household, SD = 0.8) were given a brief cessation intervention or more intensive counseling to stop smoking, of which 32 people in 17 (50%) families agreed to quit. From 53 households, a total of 73 family members were offered free NRT (of which 61 people or 84% accepted). Thirteen people from 12 households were offered a referral to Quitline (of which none accepted). Similar findings were found when the infants were seen at 2 and 3 months of age (data not reported).

Other Outcomes

Across the baseline (Table 1), 4-month, and 12-month visits, 77%–88% of infants had no parent-reported daytime cough and 75%–93% had no nighttime cough, with no statistical differences over time or by group. The majority (n = 126, 93%) of the 135 mothers in the intervention group who were interviewed when their infant was 3 months old felt that the program was "helpful" or "very helpful" for reducing SHS exposure in their child. Almost all (n = 131, 97%) were "satisfied" or "very satisfied" with the program.

Discussion

This trial tested whether a family-based SHS intervention focusing predominately on the health of infants, as opposed to smoking cessation in adults, had any effect on the number of health provider

presentations by infants for ARI over a 12-month period. Although previous research suggested that such an intervention would lead to reduced exposure to SHS for infants ([17,36]), we found no effect on SHS exposure, parental smoking, or ARI. Mothers reported exposure of the infant to SHS was low. However, mean urinary CCRs in the infants at baseline and at 4 months of age were consistent with SHS exposure. A recent U.S. trial (n = 138) looking at the effectiveness of a community-based motivational intervention to reduce SHS exposure in children under six in low-income communities reported similar findings ([37]).

Possible explanations for why the trial infants had high CCRs in the first 4 months of life are addressed below and include exposure to maternal nicotine/cotinine via breast milk, and/or unreported or underestimated SHS exposure by mothers. It is unlikely that the CCRs observed can be solely explained by high smoking rates in pregnancy, as prenatal exposure to nicotine is not measureable 3–6 weeks after birth ([38]), which was when the first urine samples were collected. Elevated CCRs in breast-fed infants has previously been noted ([39–42]) and is likely due to the transmission of nicotine metabolites in the milk of mothers who smoked and/or were exposed to high levels of SHS.

Breast milk cotinine levels have been shown to peak 30–60 min after smoking 1–2 cigarettes and dissipate after 3 hrs ([43]). Cotinine does not appear to have any pharmacological or toxicological properties of concern and thus is unlikely by itself to cause any adverse health effects for infants ([42]). However, it is unknown whether carcinogenic substances present in SHS are transferred to breast milk and thus to infants. Breast feeding has been shown to modify the effect of maternal smoking, such that the risk of ARI is decreased ([44]). As the infants aged and became increasingly mobile, their mean CCRs declined. Use of NRT by mothers trying to quit smoking may also have resulted in increased nicotine exposure for infants via breast milk ([45]), leading to increased CCRs. Approximately 50% of mothers in the intervention arm were offered NRT, but we did not record whether they used it, and it is unknown how many mothers in the usual care arm were offered and used NRT as part of usual care.

Unreported SHS exposure may also be an explanation for the elevated CCRs. Previous research has shown that the association between infant cotinine levels and parental smoking is in part due to cosleeping and minimum room temperature ([46]). We did not consider these variables and thus are unable to comment on their influence. Underestimating SHS exposure is another possibility. Some infants may have been exposed to SHS in spite of families having smoke-free rules, or without their mother recalling this exposure.

Some of the trials' strengths are that it was conducted in line with CONSORT guidelines, assessment of the primary outcome was blinded, and family members were involved. The complex reasons for the slow recruitment (especially in Australia) and high participant retention rate are discussed in a recent paper by Glover et al. ([47]). A number of limitations should be acknowledged: (a) mothers' reported infant SHS exposure and breast feeding status may be affected by social desirability bias. Advertising campaigns ran in both countries before and during the study period promoting the importance of breast feeding, smoking cessation, and not exposing children to SHS. Consequently, breast feeding may have been over-reported and SHS exposure under-reported, biasing the results toward the null (although not differentially). No measurements of home/car air quality were taken as verification of reported infant SHS exposure; (b) the number of ARI events was considerably lower than previously reported in a remote indigenous community setting ([48]), meaning our findings may not be generalizable to populations with higher rates of ARI. In Darwin, we hand-searched all hospital records and the primary care records of the largest Aboriginal Medical Service. However, we may have missed cases that attended mainstream primary care services and were not reported by mothers. In NZ, we electronically searched primary care and hospital records within the region for ARI cases but with limited data linkage cases of ARI that occurred outside of the region may have been missed (again, this bias was unlikely to be differential); (c) the mothers that we were unable to contact or those that declined to participate may have been the ones who did not have strong smoke-free policies in place and thus were the ones this intervention could have helped the most; (d) the total number of cigarettes smoked in the home is reported as a key predictor of cotinine levels in children ([49]). However, we did not assess this variable as indigenous Australians report significant sharing of cigarettes ([23]), making accurate measurement of "cigarettes smoked per day" difficult. In hindsight, we should have asked this question and acknowledged the potential misclassification; (e) as part of the intervention, information was provided to mothers about the negative health effects (including ARI) of SHS exposure on children. It can be hypothesized that mothers increased awareness of ARI may have increased their engagement with health care specialists about their infants' health, thereby reducing any differences between the groups; (f) the proportion of smoke-free homes and cars reported in this study were much higher than reported by adults in population surveys ([3,15,16,50,51]), which raises some concerns about the generalizability of our findings to other populations; (g) we did not record use of NRT by mothers trying to quit smoking, so are unable to discuss the effect of NRT use on CCRs; and (h) we did not record "partner" versus "other family member" involvement in the study, so are unable to report findings according to these two groups.

Research has shown that having smoke-free homes ([17,36]) and smoking outside or away from infants can reduce SHS exposure but does not offer complete protection as the dust, air, and surfaces within homes remains contaminated ([52]). Future research therefore needs to focus on not only supporting mothers, their partners, and other family members to stop smoking but also on how to reduce children's exposure to third-hand smoke. Personalized feedback on indoor air quality and CCR levels to families also has the potential to increase the effectiveness of future interventions ([19,53]). Future research should also investigate why these populations had no interest in Quitline support, although qualitative research from NZ (n = 168, 53% Māori) suggests awareness of the service, time required, and personal relevance may play some role ([54]).

In summary, our family-centered intervention to reduce exposure to SHS had no effect on rates of ARI in indigenous infants or on smoking and quitting behavior. These findings suggest that simply having smoke-free homes and cars is not sufficient to protect children from exposure to SHS—all household members who smoke should stop smoking from the time of conception and should continue to be smoke-free after the child is born ([55]). Furthermore, breast feeding should continue to be encouraged, but smoking while breast feeding should be discouraged.

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Contributors

DT, AC, and VJ conceived the original idea for the trial; NW, DT, VJ, MG, CB, AT, AC, PM, NB, RB, VP, CS, DF, Toni Mason, and Kane Ellis sought funding and wrote the protocol. VJ, TvB, DW, and EH managed the day-to-day running of the trial, including management of staff involved in participant follow-up. CB, AT, VJ, and DT acted as medical reviewers of the clinical records data in the study. VP provided statistical advice for the trial and carried out all data analyses. The paper was written by NW with input from all coauthors. NW will act as guarantor for the paper.

Declaration of interests

All authors declare that (1) no authors have received support from any companies for the submitted work; (2) CB has previously undertaken research on behalf of NicoNovum, but prior to the purchase of the company by RJ Reynolds. NW has provided consultancy to the manufacturers of smoking cessation medications, received honoraria for speaking at a research meeting, and received benefits in kind and travel support from a manufacturer of smoking cessation medications. MG has provided consultancy to the manufacturers of smoking cessation medications; (3) their spouses, partners, or children have no financial relationships that may be relevant to the submitted work; and (4) all authors have no non-financial interests that may be relevant to the submitted work. NW, CB, MG, and VP have also undertaken two trials of very low nicotine content cigarettes, which were purchased from two different tobacco companies. The companies concerned had no role in development of the study design, data collection, data analysis, data interpretation, or writing of the trial publications.

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