Original Investigation

Reduction of Secondhand Smoke Exposure Among Healthy Infants in Iran: Randomized Controlled Trial

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Abstract

Introduction: The objective of this study was to assess whether counseling both mothers and fathers reduces their infants’ exposure to secondhand smoke (SHS).

Methods: Participants were 130 nonsmoking children aged less than 1 year, exposed to their fathers’ or mothers’ smoking, and recruited from a health center in southern Tehran. Eligible families were randomly assigned to intervention or control group. Infant urine samples were collected, and parents were interviewed at baseline and at a 3-month follow-up in each of the 2 groups. Mothers of the intervention group were provided 3 counseling sessions, one of which was face to face and 2 of which were by telephone. Fathers were provided 3 counseling sessions by telephone. Parents were also given an educational pamphlet and a sticker depicting a smoke-free home. The control group received usual care. Changes in infant urinary cotinine levels, parental cigarette consumption in the presence of the child, and home- and car-smoking bans were assessed.

Results: The intervention was effective in reducing infant urinary cotinine levels (1-tailed p = .029). There was a greater decrease in the total daily cigarette consumption in the presence of the child in the intervention group compared with the control group, and the differences between the 2 groups were statistically significant (1-tailed p = .03). While the differences between home-smoking bans in the 2 groups were statistically significant (1-tailed p = .049), the differences between car-smoking bans did not reach significance.

Conclusion: Counseling similar to that employed in other countries can reduce infant exposure to SHS, suggesting generalizability.

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Introduction

Childhood exposure to secondhand smoke (SHS) is a major public health concern (Boyaci, Etiler, Duman, Basgigit, & Pala, 2006). Infant exposure to SHS increases the risk of respiratory tract infections, otitis media, asthma, allergy, and sudden infant death syndrome (U.S. Department of Health and Human Services, 2006). The World Health Organization has reported that exposure to SHS threatens the health of almost half of the world’s children (World Health Organization, Division of Noncommunicable Diseases, 1999). Approximately 47% of Eastern Mediterranean children and 58% of Southeast Asian children are exposed to SHS in the home (Gordon, Mackey, & Rehfues, 2004).

In Iran, the prevalence of current tobacco smoking is 26.1% for men and 3.2% for women. While cigarette smoking is the most common form of tobacco smoking among Iranian men, it is less popular among women (23.4% vs. 1.4%). In addition, the daily number of cigarettes smoked by men is more than women (Meyssamie et al., 2010). In Iranian families, cigarette smoking is not the cultural norm for women. Thus, children are mainly exposed to SHS through the father. Women who normally care for very young children must do so under conditions where the male smoker is not likely to be comfortable with instructions on protecting the child. In this cultural context, it is important to directly impact the father to protect the children. One study in Tehran found that 40.6% of children lived in homes with fathers who smoked, and approximately 35% of the smokers did not avoid smoking at home, and the SHS exposure caused by paternal smoking led to adverse health outcomes in the children (Shiva, Nasiri, Sadeghi, & Padyab, 2003).

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While smoking is banned in some public and work places, these restrictions are not enforced in the home and car (U.S. Department of Health and Human Services, 2006). SHS exposure is a preventable problem in children. If smoker parents avoid smoking in the presence of their infants, they could reduce exposure to SHS (Blackburn et al., 2005; Hovell & Hughes, 2009). Previous studies suggest that children’s exposure can be reduced through interventions (Gehrman & Hovell, 2003; Hovell et al., 2009; Priest et al., 2008). A recent study reported that interventions are effective in reducing childhood SHS exposure (Tyc, Hovell, & Winicoff, 2008). Most of the current interventional programs target smoking mothers (Emmons et al., 2001; Fossum, Arborelius, & Bremberg, 2004; Hovell et al., 2000); few studies have provided counseling to fathers who smoke, in spite of the fact that most household smoking is due to the fathers (Blackburn et al., 2005; Chan & Lam, 2006).

The effects of previous interventions designed to reduce childhood SHS exposure were assessed based on parents’ reports of their smoking habits, nicotine levels in the household air, and cotinine in the mother’s milk, saliva, or the child’s urine (U.S. Department of Health and Human Services, 2006). The sensitivity and specificity of parental reports have been questioned in previous studies (Boyaci et al., 2006; Wilkinson, Arheart, & Lee, 2006; Willers et al., 2000). However, Hovell et al. (2000) have shown relatively high association among cotinine, nicotine, smoking levels, and reported SHS exposure, suggesting equal degree of validity for both “objective and reported” measures. Thus, to determine the effects of intervention, measurements of cotinine levels were suggested (Benowitz, 1996). The ratios of cotinine to creatinine are often used to account for dilution (U.S. Department of Health and Human Services, 2006).

To the best of our knowledge, there are no interventional studies on the reduction of infant SHS exposure in Iran. In the only interventional study on the topic in Iran, 40 parents and children aged 8–12 years were educated about the harmful effects of passive smoking, and the study found that educating children was more effective than educating parents, which resulted in change in SHS exposure (Kelishadi, Moghtaderi, Khavarian, & Famouri, 2007). The aim of this study was to assess the effects of providing counseling to Iranian smoking parents to reduce their infants’ SHS exposure.

**Methods**

**Recruitment**

Families attending a health center in southern Tehran for routine infant health checks were approached to identify whether they were eligible and willing to participate in the study. After having provided informed consents, 130 families were recruited and randomly assigned to the control or intervention group. Considering the nature of the intervention, blinding the participants and researchers to the group assignment was not possible. The statistical analyst and outcome assessors were blinded to the group assignment, the control group was uniformed of the counseling processes, and the researchers did not know the results of the study until all the data were collected. All the laboratory tests were performed with no knowledge of group assignment and parental smoking status. Eligible families were those with healthy infants aged less than 12 months of age and at least one smoking parent who smoked at least 1 cigarette/day; the parents also had to be able to speak Persian and have a telephone number. Parents who reported the use of other addictive substances or being under a smoking cessation treatment program were excluded from the study. Ethical approval was obtained from the Ethics Committee of the Tehran University of Medical Sciences, Tehran, Iran (code number 87-01-28-6870).

**Intervention and Control Conditions**

In the intervention group, motivational interviewing (Miller & Rollnick, 2002) was used to reduce household SHS exposure. The motivational intervention was designed to raise parents’ motivation and perceived self-efficacy to change their present smoking habits by investigating and resolving their ambivalence about the change. Counseling was based on the “Smoke-free children” method (Arborelius & Bremberg, 2001; Fossum et al., 2004; Greenberg et al., 1994). The method included elements such as finding out what the parents themselves know about the risks of infant SHS exposure, suggesting to mothers that they record how much cigarette smoke there is in the presence of the infant, discussing current smoking habits and whether parents have suggestions about possible changes, and supporting parental efforts to change smoking habits and resolving difficulties faced in trying to change. Mothers were provided three counseling sessions, one of which was face to face and two of which were by telephone, and fathers were provided three counseling sessions by telephone. The counselor was a supervised master’s level student. The mean duration of counseling varied between participants, with the maximum duration of 20 min. In addition, parents were given an educational pamphlet about reducing infant exposure to SHS and a sticker depicting a smoke-free home where the father chooses to smoke outside to protect the infant. The control group received usual care but had the opportunity, if desired, to receive the intervention after completion of the study. The usual care included usual health visits for checking the infants’ growth and developmental milestones.

**Outcome Measures**

The main outcome of this study was the changes in infant exposure to SHS as measured by urinary cotinine, a major metabolite of nicotine. A secondary outcome of this study was the change in infant exposure to SHS based on parents’ reports of alternations in cigarette smoking.

**Measures of Infant Exposure to SHS**

**Infant Urinary Cotinine**

Urine samples collected from the children at baseline and at a 3-month follow-up using a urine collection bag were immediately frozen and transported to the Department of Toxicology at Tarbiat Modares University and stored at −20 °C until analysis. Urinary cotinine was analyzed using gas chromatography based on the methods of Feyerband and Russell (1990) with some modifications. The cotinine in the urine was adjusted for creatinine to account for dilution effects. The detection limit was 1–3 ng/mg.

**Parental Reports**

Parents were interviewed at baseline and at a 3-month follow-up. A questionnaire consisting of three parts was completed.
through a face-to-face interview. It consisted of 14 questions on demographic characteristics, 5 questions on house characteristics, and 15 questions on smoking status. The questionnaire was adapted from the tool used by Wakefield et al. (2000), with the permission of the principal investigator. An acceptable 1-week reliability test with a minimum kappa coefficient of 0.88 for qualitative variables and a minimum Pearson correlation coefficient of 0.82 for quantitative variables was obtained. The social status of the families was determined according to the Registrar General Model of Social Class (Currie, Elton, Todd, & Platt, 1997). An index was calculated to describe the population density of the households by dividing the total number of family members by the total number of rooms in the home. The index was scored as low (scores 0–1), moderate (scores 2–3), or high (score >3; Nurgalieva et al., 2002).

The participants were questioned about all smokers in the household and the total number of cigarettes smoked per day by each one. To estimate the rate of infant SHS exposure, daily cigarette consumption in the presence of the infant on weekdays and weekends was ascertained. Weekday daily cigarette consumption was multiplied by the number of weekdays in the previous week, and weekend daily cigarette consumption was multiplied by the number of weekend days in the same week. The two consumption figures were then added and divided by 7 to yield the weighted daily exposure (Wakefield et al., 2002). The total daily cigarette consumption of smoking parents in general as opposed to total cigarette consumption in the presence of an infant in both intervention and control groups was assessed.

**Home- and Car-Smoking Ban Status**

To compare changes in smoking ban status in the home and car, the variables at baseline and follow-up were dichotomized as a complete ban or partial/no ban.

**Statistical Methods and Sample Size**

Since urinary cotinine was not normally distributed, log adjustments were made and geometric means were employed to reduce skewness. Intervention and control groups were compared at baseline using the independent *t* test, chi-square test, and Fisher’s exact test. Changes in infant’s urinary cotinine-to-creatinine ratios (CCRs) and parental cigarette consumption in the presence of the child were compared between the two groups using the independent *t* test and the Mann–Whitney *U* test respectively. Spearman’s rank was used to estimate correlations. The McNemar test was used to compare changes from baseline in home-smoking ban status and car-smoking ban status in each group. These variables were then compared between the two groups using the chi-square test and Fisher’s exact test. The data were analyzed using SPSS version 16.0, and *p* values <.05 were considered as statistically significant. The sample size calculation was based on study outcomes. In agreement with the study of Wilson et al. (2001) to detect a 35% reduction in infants’ SHS exposure at the 3-month follow-up with a power of 0.80 and 5% significance level (one-tailed), 110 participants (55 in each group) were needed, given an expected dropout rate of 20%. A total of 130 participants (65 in intervention and 65 in control groups) were planned to allow for larger effect size of the secondary outcomes. Because many of the community trials designed to lower SHS exposure among children have observed reduced exposure and reduced smoking in both the control and the experimental conditions and as this is the first study of its kind in Iran; we adopted a one-tailed test, to avoid Type II error, for a pilot testing generalizable to the population of Iran (Gehrman & Hovell, 2003; Hovell & Hughes, 2009).

**Trial Registration**

The study protocol was approved by the Ethics Committee of the Tehran University of Medical Sciences in Tehran, Iran (code number: 87-01-28-6870). This trial was registered at www.ClinicalTrials.gov (NCT00821639) and with the Iranian Registry of Clinical Trials at www.irct.ir (IRCT138801171791N1).

**Results**

Participants were recruited between July 2008 and May 2009. Figure 1 shows the participant flowchart through the trial. Overall, 130 families were eligible and consented to participate in the study. Follow-up data after 3 months were incomplete for nine participants (6.9%). Table 1 shows baseline characteristics of the intervention and control groups. The two groups were comparable and did not differ with respect to any of the baseline variables (all two-tailed *p* values >.05), indicating successful randomization. The study sample was predominantly of low social class. There were no families in the first and second classes, only 40.8% had a car, and a high proportion of the infants (90%) did not have separate rooms. In 46.2% of the households, the household density index was two or three persons per room. Most mothers were housewives (98.5%), and only 3%–4% of the parents had a university education. Most of the families only had one child, and 80.8% of the families lived in rental accommodations. In 96.9% of the families, only the father smoked; in 0.8%, only the mother smoked; and in 2.3%, both parents smoked. There were no other smokers in the households.

**Outcome Measurements**

**Infant Urinary Cotinine**

The CCRs ranged from 3 to 330 ng/mg, with a median of 43.5 ng/mg; the first quartile was 29 ng/mg, and the third quartile was 66.5 ng/mg. Figure 2 shows that infant’s geometric mean urinary cotinine concentrations decreased from 48.72 ng/mg at baseline to 28.68 ng/mg at the 3-month follow-up in the intervention group. In the control group, mean urinary cotinine concentrations decreased from 40.43 to 36.32 ng/mg. The differences between the two groups varying over the course of the 3-month follow-up were statistically significant (one-tailed *p* = .029).

**Parental Reports**

There was a greater decrease in the total daily cigarette consumption in the presence of the child in the intervention group (median = 0, interquartile range: 0, 2.71) than in the control (median = 1, interquartile range: 0, 3.21) at the 3-month follow-up. The differences between the two groups were statistically significant (one-tailed *p* < .03). There was no significant correlation between the CCRs and the reported level of exposure (*r* = −.01, two-tailed *p* = .84) and the CCRs and the reported level of smoking in the home and car (*r* = .07, two-tailed *p* = .47).

**Home- and Car-Smoking Ban Status**

In the control group, home-smoking bans increased from 11.5% at baseline to 19.7% at the 3-month follow-up, but this
increase was not statistically significant (one-tailed $p = .13$). In the intervention group, the ban on smoking in the home increased from 15% at baseline to 33.3% at the 3-month follow-up, reaching significance (one-tailed $p < .001$). The differences between the two groups were statistically significant (one-tailed $p = .049$). Table 2 shows the change in car-smoking ban status for the intervention and control households who had a car. In the control group, the ban on smoking in the car did not change. In the intervention group, bans increased from 4% at baseline to 8% at the 3-month follow-up, but this increase was not statistically significant (one-tailed $p = .5$). The differences between the two groups were not statistically significant (one-tailed $p = .5$).

### Discussion

This is the first trial in Iran to test the benefits of an intervention consisting of in-person and phone counseling and information provided directly to both the father and the mother of infants known to be exposed to SHS at baseline. With a significant differential reduction in cotinine and reported infant exposure in 3 months, we believe that our brief counseling program is responsible for these short-term changes. Unfortunately, the power of the intervention and limited sample size did not enable definitive detection of significant differences in car-smoking bans. Here too, the relatively large effects seen for bans suggest that more emphasis on bans in future studies might show greater differential effects. These design issues are in the context of recognized reactivity from measures in previous studies of SHS exposure that artificially results in change in a therapeutic direction for control families (Hovell et al., 1994). This observation suggests that counseling interventions may be more powerful than usually observed in the context of apparent measurement reactivity.

In this study, infant exposure to SHS, as measured by urinary cotinine concentrations, decreased from baseline to follow-up in the intervention group compared with the control ($p = .029$), consistent with previous studies (Fossum et al., 2004; Gehrman & Hovell, 2003; Hovell et al., 2009; Tyc et al., 2008). However, one of the first trials, the “Smoke-free children” intervention, reported by Greenberg et al. (1994) showed a decrease of infant exposure to SHS based on parental reports but did not obtain a significant decrease in infant urinary cotinine levels. This study was published before the potential confounding due to thirdhand smoke (THS) exposure was recognized, and most studies to date have not established complete bans in private homes to offset this risk. Thus, reliance on cotinine as the only standard for judging the effects of counseling may result in false-negative conclusions. Furthermore, the main limitation of cotinine as a biomarker of exposure is the short half-life, which limits the duration of exposure.

Infants of smoking parents are at a particular risk of THS exposure through contaminated house surfaces. The present study does not offer a measure of THS exposure or contamination separate from that of SHS. The fact that these homes had been contaminated for some time suggests that children were exposed to off-gassing and particle exposures in dust and on surfaces. Thus, even when the parents reduced or eliminated all
smoking in home, we would expect some residual exposure that could sustain relatively high cotinine levels even if the parents smoked only when the child was not at home or even if they always smoked outdoors. Thus, the fact that cotinine remains relatively high may mean that even more powerful interventions must be employed to reduce SHS exposure and that interventions might need to include decontamination procedures to reduce THS exposure (Hovell & Hughes, 2009; Matt et al., 2004, 2008; Winicoff et al., 2009). Thus, reduction in SHS and THS exposures may require cultural change to motivate legislators to establish policies that protect the children and the public from SHS and THS exposures (Hovell & Hughes, 2009).

Although the difference in car-smoking bans between the intervention and control groups was not statistically significant, home-smoking bans increased significantly between the two groups ($p = .049$). With respect to methodological factors that may have influenced the results, it is noteworthy that the groups were comparable at baseline, the loss to follow-up rate was acceptable (6.9%), and there were no significant differences between the two groups in the loss to follow-up rate or smoking ban status at baseline. Therefore, differences in group composition or loss to follow-up were unlikely to explain the study outcomes.

It seems that a larger sample and/or a somewhat more powerful intervention might result in clear establishment of bans in the intervention condition and that future studies should emphasize bans in order to reduce both SHS and THS exposures. A small study of 72 households reported a slight

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intervention group$^a$ ($n = 65$)</th>
<th>Control group$^a$ ($n = 65$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant’s age (months)</td>
<td>4 ± 3.9</td>
<td>4.3 ± 3.6</td>
</tr>
<tr>
<td>Infant’s gender (% male)</td>
<td>61.5</td>
<td>60</td>
</tr>
<tr>
<td>Mother’s age (years)</td>
<td>28.1 ± 5.2</td>
<td>26.4 ± 5.4</td>
</tr>
<tr>
<td>Father’s age (years)</td>
<td>33.2 ± 6.3</td>
<td>31.4 ± 5.3</td>
</tr>
<tr>
<td>Mother’s education (years)</td>
<td>9.2 ± 3.3</td>
<td>9.6 ± 2.7</td>
</tr>
<tr>
<td>Father’s education (years)</td>
<td>8.6 ± 3</td>
<td>8.6 ± 2.8</td>
</tr>
<tr>
<td>Mother’s job (other than housewife)</td>
<td>1 (1.5)</td>
<td>1 (1.5)</td>
</tr>
<tr>
<td>Social class of household</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clerical or skilled nonmanual</td>
<td>7 (10.8)</td>
<td>10 (15.4)</td>
</tr>
<tr>
<td>Manual skilled</td>
<td>32 (49.2)</td>
<td>31 (47.7)</td>
</tr>
<tr>
<td>Unskilled or semiskilled</td>
<td>26 (40)</td>
<td>24 (36.9)</td>
</tr>
<tr>
<td>Housing status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Owner occupied</td>
<td>12 (18.5)</td>
<td>13 (20)</td>
</tr>
<tr>
<td>Rented/other</td>
<td>53 (81.5)</td>
<td>52 (80)</td>
</tr>
<tr>
<td>Having a car (yes)</td>
<td>26 (40)</td>
<td>27 (41.5)</td>
</tr>
<tr>
<td>Crowding index$^c$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>10 (15.4)</td>
<td>10 (15.4)</td>
</tr>
<tr>
<td>Moderate</td>
<td>30 (46.2)</td>
<td>30 (46.2)</td>
</tr>
<tr>
<td>High</td>
<td>25 (38.5)</td>
<td>25 (38.5)</td>
</tr>
<tr>
<td>Access to outdoor area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>5 (7.7)</td>
<td>7 (10.8)</td>
</tr>
<tr>
<td>Yard</td>
<td>45 (69.2)</td>
<td>46 (70.8)</td>
</tr>
<tr>
<td>Back yard, balcony, or roof</td>
<td>15 (13.5)</td>
<td>12 (18.5)</td>
</tr>
<tr>
<td>Separate room for infant (yes)</td>
<td>7 (10.8)</td>
<td>6 (9.2)</td>
</tr>
<tr>
<td>Home-smoking ban (yes)</td>
<td>9 (13.8)</td>
<td>7 (10.8)</td>
</tr>
<tr>
<td>Car-smoking ban (yes)</td>
<td>1 (3.8)</td>
<td>2 (7.4)</td>
</tr>
<tr>
<td>Infant’s nutrition type (breast feeding)</td>
<td>65 (100)</td>
<td>62 (95.4)</td>
</tr>
<tr>
<td>Mean number of cigarettes smoked per day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>2.6 ± 1.9</td>
<td>3 ± 1.4</td>
</tr>
<tr>
<td>Father</td>
<td>12 ± 10</td>
<td>11.6 ± 8.3</td>
</tr>
<tr>
<td>Total daily cigarette consumption in presence of the infant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (interquartile range)</td>
<td>0.6 (0, 3.5)</td>
<td>1 (0, 3.4)</td>
</tr>
<tr>
<td>Low (&lt;10)</td>
<td>58 (92)</td>
<td>55 (87.3)</td>
</tr>
<tr>
<td>Medium/high (≥10)</td>
<td>5 (8)</td>
<td>8 (12.7)</td>
</tr>
<tr>
<td>Urinary cotinine (ng/mg)</td>
<td>48.7 ± 1.1</td>
<td>40.4 ± 1.0</td>
</tr>
</tbody>
</table>

Note. $^a$Values are numbers (%), means ± SD, or median (interquartile range); urinary cotinine values are geometric means ± geometric SDs. $^b$For categorical variables, chi-square and Fisher’s exact tests showed no statistically significant differences ($p$ values > .05) between the two groups. For continuous variables, independent $t$ tests and Mann–Whitney $U$ test showed no statistically significant differences ($p$ values > .05) between the two groups. $^c$Crowding index was calculated by dividing the total number of family members by the total number of rooms in the home. The index was scored as low (scores 0–1), moderate (scores 2–3), or high (score >3).
increase in home-smoking bans in the intervention group at follow-up, although the difference was not statistically significant relative to the control group; this may, however, have been due to the limited sample size and resulting limited power to detect small to moderate effects assessed in that study (McIntosh, Clark, & Howatt, 1994). Others have also reported nonsignificant results raising questions about the conditions and sample sizes required to detect true effects from counseling and other interventions (Wakefield et al., 2002). It is important to note that a community social norm may affect home policy (Winickoff et al., 2009). In Iran, smoking is banned in all enclosed public places, workplaces, and public transportation vehicles; however, nonsmokers are still exposed to cigarette smoke outside the home and in social settings that necessitates public health interventions and enhancement of the existing legislations (Kelishadi et al., 2007; Warren, Sinha, Lee, Lea, & Jones, 2009).

Few studies have assessed the effects of intervention to reduce children’s SHS exposure in low- and middle-income countries where the prevalence of smoking among women was low compared with the rates of smoking by men (Oncken et al., 2010). In agreement with the present study, these studies reported a reduction of smoke exposure among children (Abdullah, Mak, Loke, & Lam, 2005; Chan, Leung, Wong, & Lam, 2008; Chan et al., 2005). Further research is required in developing countries including Iran to ascertain culturally sensitive interventions to protect children from exposure to SHS. Future studies in developed and developing countries should emphasize establishment of complete smoking home bans in order to reduce SHS and THS exposures.

The majority of the participants in the present study were from low socioeconomic classes. Therefore, participants may have had other pressing problems that compete with lowering children’s SHS exposure. To achieve greater reductions in children’s urinary cotinine levels in such high-risk families, even more intensive interventions may be required (Hovell et al., 2009).

In this study, there was a lack of association between report ed exposures and infant urinary cotinine levels. Some studies indicated that parental reports were reliable (Matt et al., 2000; Seifert, Ross, & Norris, 2002), but other studies questioned the accuracy of parental reports (Boyaci et al., 2006; Wilkinson et al., 2006; Willers et al., 2000). This issue probably reflects the questionnaire, conditions under which answers were provided, the timing of the reports in relation to the timing of the urine samples, and possible bias on the part of the respondents. Culture could play a role in the degree of accurate disclosure about smoking and SHS exposure.

Study procedures conducted in the control group might affect their behaviors, which may have underestimated the true effects of counseling due to reactivity to measures and other nonspecific features true for both conditions. Several studies have demonstrated that interviewing and sampling of body fluids in the control group may influence study outcomes (Hovell et al., 2009). In this study, infant urinary cotinine levels and total daily parental cigarette consumption in the presence of the infant decreased and home-smoking bans increased in both the control and the intervention groups, which may have masked the true magnitude of the effect of the intervention. Because of the urinary collection procedures, parents in the control group knew that the study was related to their smoking behavior and infant exposure to SHS. However, further contamination of the control group was unlikely because no further contact was made with them until the 3-month follow-up interview. This study did not provide a formal attention control procedure for the control condition. However, as shown in Hovell et al. (1994), standard measurement procedures are reactive and tend to reduce exposure in the control condition. Thus, measures served as a partial attention (and feedback) control process.

In summary, this is the first study of the reduction of infant exposure to SHS in Iran and found significant differential reduction in cotinine for experimental families compared with controls as measured by cotinine and parental reports. We conclude that it is possible to reduce infants’ SHS exposure in Iranian homes by counseling both fathers and mothers and that future studies should test more refined and powerful interventions with greater emphasis on establishing complete home bans. This study highlights the need for future studies that can employ interventions of greater duration and intensity to encourage parents, particularly fathers, to take action to protect their children from exposure to SHS.

### Table 2. Changes in Home- and Car-Smoking Bans in the Intervention and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>Intervention group</th>
<th>Control group</th>
<th>p value*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Home bans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>9</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>3-month follow-up</td>
<td>20</td>
<td>33.3</td>
<td>12</td>
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<tr>
<td>3-month follow-up</td>
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<td>8</td>
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Note. Differences between the two groups were compared using chi-square and Fisher’s exact tests.
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Declaration of Interests

None declared.

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References


