Advising parents of asthmatic children on passive smoking: randomised controlled trial

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Abstract

Objective To investigate whether parents of asthmatic children would stop smoking or alter their smoking habits to protect their children from environmental tobacco smoke.
Design Randomised controlled trial.
Setting Tayside and Fife, Scotland.
Participants 501 families with an asthmatic child aged 2-12 years living with a parent who smoked.
Intervention Parents were told about the impact of passive smoking on asthma and were advised to stop smoking or change their smoking habits to protect their child's health.
Main outcome measures Salivary cotinine concentrations in children, and changes in reported smoking habits of the parents 1 year after the intervention.
Results At the second visit, about 1 year after the baseline visit, a small decrease in salivary cotinine concentrations was found in both groups of children: the mean decrease in the intervention group (0.70 ng/ml) was slightly smaller than that of the control group (0.88 ng/ml), but the net difference of 0.19 ng/ml had a wide 95% confidence interval (– 0.86 to 0.48). Overall, 98% of parents in both groups still smoked at follow up. However, there was a non-significant tendency for parents in the intervention group to report smoking more at follow up and to having a reduced desire to stop smoking.
Conclusions A brief intervention to advise parents of asthmatic children about the risks from passive smoking was ineffective in reducing their children's exposure to environmental tobacco smoke. The intervention may have made some parents less inclined to stop smoking. If a clinician believes that a child's health is being affected by parental smoking, the parent's smoking needs to be addressed as a separate issue from the child's health.

Introduction

The adverse effects of passive smoking on the respiratory system of children has been shown in infancy[1] and throughout childhood.[2] Asthmatic children have more severe disease if their parents smoke.[3]

Many asthmatic children are exposed to high levels of tobacco smoke at home.[4] Exposure mainly depends on proximity to smokers, and young children who spend much of their time with parents that smoke are particularly vulnerable.

The harmful effects of active smoking are now well known through campaigns,[5] but whether the risks from passive smoking are appreciated is unproved. Clinicians have been advised to counsel parents about the harmful effects of passive smoking on their children.[6] It is not clear whether this advice encourages parents to reduce their children's exposure to tobacco smoke.

We aimed to investigate whether a brief intervention informing parents about the harmful effects of smoking on childhood asthma encouraged them to stop smoking or to modify their smoking habits to protect their children.

Participants and methods

Recruitment

We invited 123 general practices in Tayside and Fife to take part in our study: 73 (59%) agreed to participate, and these practices identified 1047 potential families for our study. Families were considered eligible if they had a child aged 2-12 years with documented asthma who lived with a parent or guardian who smoked. Children were identified from asthma registers or from repeat prescribing of asthma drugs. We chose the lower age limit of 2 years to ensure that asthma had been definitely diagnosed, and we chose the upper age limit of 12 years to minimise the number of children who were actively smoking.

We selected the index parent as the parent of an asthmatic child who was registered with the same general practitioner as the child and who was currently a smoker. We ascertained smoking status from case notes or computerised records of the parent's lifestyle, and this status was confirmed at interview. When both parents were eligible, we invited the main carer to take part in our study.

We invited the 1047 families to take part in our study. We excluded 246 families (23.5%) as they did not meet the entry criteria: 121 parents (11.6%) reported being non-smokers; 76 children (7.3%) were not taking asthma drugs; 27 children (2.6%) had not been diagnosed with asthma; 9 parents (0.9%) were seldom at home; 6 children (0.6%) were unable to provide saliva samples; and 7 families (0.7%) were excluded for

other reasons. We could not contact 97 families (9.3%). Of the remaining 704 families (67.2%), 501 (47.9%) agreed to take part. We obtained written consent from all parents and from those children who were old enough to complete the form. Our study was approved by the two local research ethics committees.

Sample size

We aimed to reduce the proportion of children with high salivary cotinine concentrations. Cook et al[7] showed that 86% of children exposed to adults who smoke have salivary cotinine concentrations greater than 0.6 ng/ml. To detect a decrease in cotinine concentrations from 86% to 74% in children with concentrations greater than 0.6 ng/ml as being significant at the 5% level, with a power of 90%, we would require 248 children in both the control group and the intervention group.

Data collection

The families were visited at home on two occasions—at baseline and then about 1 year later—by two research nurses (LI and KG). At baseline each research nurse recruited half of the study population. At the second visit each nurse visited those families recruited by her colleague, thus the nurses remained blind to baseline information. A questionnaire was completed by the index parents at both visits. Information was collected on family socioeconomic factors, the child's asthma, smoking habits of the index parent, and overall exposure of the child to tobacco smoke. Saliva samples were obtained from the parents and the children on both occasions to measure cotinine concentrations, the major metabolite of nicotine.[8] Our methods have been reported.[4]

Intervention

On giving their written consent, the families were randomised to either an intervention group or a control group. The intervention was designed to be brief, on the basis of a method reported by Russell et al.[9] At the baseline visit, parents in the intervention group were given information on passive smoking. This was followed by a discussion on asthma, passive smoking, the effects of environmental tobacco smoke, and the potential benefits to the child when tobacco smoke is avoided. Financial and health benefits were also discussed. The parents were *(a)* given information on how to seek help to stop smoking, *(b)* advised that if they could not stop smoking then smoking in a different room or outside the home could help to protect their child, and *(c)* advised that their child's exposure to tobacco smoke could further be reduced by discouraging visitors from smoking in the home. The parents were given a leaflet (the first in a series of three) that was specifically designed to reinforce the information given and that included information on seeking help to stop smoking. The parents were also given a commercially available leaflet by The Advisory Council on Drug and Alcohol Education (TACADE). At 4 and 8 months after the baseline visit, they were sent the second and third leaflets by post with a letter encouraging them to stop smoking.

Parents in the control group were given the commercial leaflet on smoking but they were not given the additional information on passive smoking and asthma, and they were not advised to stop smoking to protect their child. No further contact was made with the parents until the follow up visit at home.

Follow up

Families were revisited at home about 1 year after the initial visit. We chose a 1 year follow up to assess the long term effects of the intervention.

Data analysis

We analysed the data with SPSS for Windows. As the children's cotinine concentrations were highly skewed, we used the conventional logarithmic transformation.[7] However, as the difference in cotinine concentrations (baseline minus follow up) was approximately normally distributed, we made no transformation for the analyses of change in cotinine concentration. To compare the intervention and control groups at baseline and to detect differences between the groups at follow up, we used the χ^2 test and *t* tests.

We obtained complete datasets (questionnaire data and salivary cotinine concentrations at baseline and follow up) from 435 families (86.8%). Analysis was on the basis of 213 families in the intervention group and 222 families in the control group. Overall, 14 families (2.8%) refused to participate at the second visit (6 intervention, 8 control), 13 (2.6%) had moved out of the two regions (9 intervention, 4 control), 9 (1.8%) no longer had the index parent and child living together (8 intervention, 1 control), 10 (2.0%) had children with high cotinine concentrations suggesting active smoking (3 intervention, 7 control), 12 (2.4%) had missing data for salivary cotinine concentrations (7 intervention, 5 control), 6 (1.2%) failed to get a follow up appointment despite repeated attempts (3 intervention, 3 control), one (0.2%) had a mother with poor health (intervention), and one (0.2%) had a father who frequently worked away from home (control).

Results

Randomisation

We compared those factors we had identified[4] as having an influence on cotinine concentrations in children (table 1). These were: the child's age, smoking habits of the index parent, contact with other smokers, and the home environment. The groups were similar for age, sex, socioeconomic factors, and smoking status of the parents. The children's mean cotinine concentrations showed that both groups had been similarly exposed to tobacco smoke: 2.83 ng/ml in the intervention group and 2.91 ng/ml in the control group (geometric mean).

At follow up, 12 parents (2.8%) reported that they had stopped smoking (7 intervention, 5 control). This was confirmed by measurement of salivary cotinine concentrations in all but two parents: 57.9 ng/ml in the intervention group and 78.9 ng/ml in the control group. Overall, 101 parents (47%) in the intervention group and 97 parents (44%) in the control group reported trying to stop smoking during the previous year, with similar numbers reporting several attempts. Ten parents (5%) from each group reported trying to stop on more than five occasions. Sixty seven parents (31%) from the intervention group and 66 parents

Table 1 Characteristics of intervention and control groups at baseline. Values are numbers (percentages) unless stated otherwise

Characteristics	Intervention group (n=213)	Control group (n=222)
Children		
Mean age (years; range)	7.7 (2.0-12.8)	7.5 (2.1-12.9)
Male	146 (68)	139 (63)
Salivary cotinine concentration (ng/ml; geometric mean)	2.83	2.91
Regular contact with smokers in addition to parents	114 (54)	118 (53)
Parents		
Mean age (years; range)	32.7 (19.5-49.1)	33.3 (20.5-53.8)
Male	42 (20)	48 (22)
Living with partner	160 (75)	170 (77)
Socioeconomic status		
Completed higher education	31 (15)	38 (17)
Non-manual employment	44 (21)	47 (21)
Owner occupied accommodation	81 (38)	89 (40)
>1 person per room	88 (41)	88 (40)
Have a garden	181 (85)	176 (79)
Smoking status of index parents		
Salivary cotinine concentration (ng/ml; arithmetic mean)	344.8	365.3
>20 cigarettes per day	64 (30)	74 (33)
>10 cigarettes per day in home	70 (33)	70 (32)
Smoke in room with child every day	107 (50)	118 (53)
With partner who smokes	90 (42)	89 (40)
Strong desire to stop smoking	116 (54)	129 (58)

(30%) from the control group reported at least one period of smoking cessation, although only 19 parents from each group managed to stop for more than 1 month. None of these findings was statistically significant.

Cotinine concentrations

The children showed a small decrease in cotinine concentrations at the second visit: the mean decrease in the intervention group (0.70 ng/ml) was slightly smaller than in the control group (0.88 ng/ml), but the net difference of 0.19 ng/ml had a wide 95% confidence interval (−0.86 to 0.48). Parental cotinine concentrations had increased marginally in both groups by the second visit. Although the mean increase was slightly larger in the intervention group (3.1 ng/ml) than in the control group (1.8 ng/ml), the net difference of 1.3 ng/ml again had a wide confidence interval (−26.4 to 23.9).

At baseline, 409 of 435 children (94.0%) had cotinine concentrations greater than 0.6 ng/ml: 202 of 213 children (95%) in the intervention group and 207 of 222 children (93%) in the control group. At follow up, 398 children (91.5%) had cotinine concentrations greater than 0.6 ng/ml: 196 children (92%) in the intervention group and 202 children (91%) in the control group.

Table 2 Changes in reported levels of parental smoking at follow up. Values are numbers (percentages) unless stated otherwise

Variable	Intervention group (n=213)	Control group (n=222)	χ^2 for trend*	P value
Reported difference in total amount smoked by index parent				
Smokes less†	59 (28)	55 (25)		
Smokes same amount	96 (45)	120 (54)	0.21	0.65
Smokes more	58 (27)	47 (21)		
Reported difference in amount smoked in home by index parent				
Smokes less†	104 (49)	84 (38)		
Smokes same amount	47 (22)	65 (29)	3.25	0.07
Smokes more	62 (29)	73 (33)		
Reported difference in smoking in same room as child				
Smokes less†	59 (28)	49 (22)		
Smokes same amount	131 (61)	139 (63)	3.05	0.08
Smokes more	23 (11)	34 (15)		

*df=1. †Includes parents who have stopped smoking (7 intervention, 5 control).

Changes in reported smoking

We assessed the changes in smoking habits by comparing the difference in responses to identical questions at baseline and follow up. At follow up, more parents in the intervention group (59, 28%) reported smoking less frequently in the same room as their child than parents in the control group (49, 22%; table 2). Similarly, 104 parents (49%) in the intervention group and 84 parents (38%) in the control group reported smoking less in the home at follow up. These differences were non-significant. However, more parents in the intervention group (58, 27%) smoked more cigarettes per day at the end of the study period than parents in the control group (47, 21%).

Impact of the study

We asked the parents if our study had encouraged them to think about stopping smoking or changing their smoking habits: 114 parents (54%) in the intervention group and 122 parents (56%) in the control group said that it had. When asked how much they wanted to stop smoking at follow up, however, only 31 parents (15%) in the intervention group compared with 51 parents (24%) in the control group expressed a greater desire to stop smoking than at baseline (P = 0.06).

Discussion

Our study showed that informing parents of the harmful effects of passive smoking was ineffective in persuading them to reduce the exposure of their children to tobacco smoke. Cotinine concentrations in the children had decreased by the end of follow up in both groups, but by the same margin. The decrease was of the order we would expect from the ageing of the children by 1 year.[4] The intervention also failed to increase either the number of attempts by parents to stop smoking or the numbers who had stopped at 1 year. Slightly more parents in the intervention group reported smoking less frequently in the presence of their child. The effect of this was weak as the cotinine concentrations were not correspondingly changed. Fewer parents in the intervention group (30/206; 15%) reported an increased desire to stop smoking at the end of the study than parents in the control group (51/217; 24%), but this difference was non-significant (P = 0.06). Similarly, more parents in the intervention group (58/213; 21%) than in the control group (47/222; 27%) reported smoking more overall at the end of the study than they had at baseline. These findings are consistent with the theory that patients are resistant to information or advice when it is not being sought.[10 11] As Butler said "telling patients what to do can make them feel challenged and provoke them to assert control by continuing their unhealthy behaviours with renewed vigour. Patients often erect barriers in response to the attempted imposition of a medical agenda."[12]

Ours is the first study to report the effect of a brief intervention on parents of asthmatic children in which

Key messages

- Many asthmatic children are exposed to high levels of environmental tobacco smoke
- A brief intervention informing parents of asthmatic children on the harmful effects of passive smoking did not lead to a reduction in exposure of their children to tobacco smoke
- Low rates of smoking cessation were found in both the intervention group and the control group
- Some parents may have been less inclined to stop smoking after the intervention
- Brief interventions requesting smokers to stop for another person's health seem ineffective

an objective measure of exposure to tobacco smoke—salivary cotinine concentrations—was used. A small scale study on asthmatic children did not report changes in cotinine concentrations.[13] Other trials have studied non-asthmatic children. One study of newborn infants found a non-significantly higher cotinine concentration in the intervention group than in the control group.[14] Another small study advising parents of ways to reduce the exposure of their children to environmental smoke showed no significant effect on the children's cotinine concentrations,[15] but this was flawed because follow up measurements of continine concentrations were not available for half of the children. A larger study that monitored passive exposure of preschool children to tobacco smoke by self report rather than by cotinine concentrations, found no effect on parental smoking behaviour.[16]

Our intervention was designed to be brief—that is, a package that could be easily delivered to parents in a clinical setting. Possibly a more intensive intervention repeated on several occasions could have been more effective. However, a recent systematic review showed that more intensive advice was no more successful in encouraging smoking cessation than brief advice.[17]

The overall cessation rate of 3% in our study was slightly lower than that of unaided smoking cessation (7%) reported in two recent meta-analyses.[17 18] Several explanations may apply. We recruited smokers who were not seeking help to stop smoking.[19] Several factors that are associated with poor success at quitting were apparent in our study. These were young age (mean age 33 years),[20] being female,[21] having a partner who smoked,[22] and low social class.[23] Finally, parents may regard the home as the only place where they are free to make choices about their smoking as more restrictions on smoking in public places are enforced.

Conclusion

Our study has shown that a brief intervention focusing on children's health is not sufficient to achieve a long term change in parental smoking. The intervention may have made some parents less inclined to stop smoking. Brief interventions on smoking cessation targeted at the smoker's health may have a modest impact,[24] but interventions aimed at the health of a third party—in this case the parent's child—seemed ineffective.

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Endpiece

More than reason

Many things escape the reason, and a person who should attempt to understand life by merely using his reason would be like a man trying to take hold of a flame with the tongs. Nothing remains but a bit of charred wood, which immediately stops flaming. (André Gide)

Alice Heim, *Intelligence and Personality* (1970)

Submitted by Nicholas Steel, health services research fellow, University of East Anglia