How Should Parents Protect Their Children From Environmental Tobacco-Smoke Exposure in the Home?

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ABSTRACT. *Background.* **Children's exposure to tobacco smoke is known to have adverse health effects, and most parents try to protect their children.**

Objective. **To examine the effectiveness of parents' precautions for limiting their children's tobacco-smoke exposure and to identify variables associated to parents' smoking behavior.**

Design and participants. **Children, 2.5 to 3 years old, participating in All Babies in Southeast Sweden, a prospective study on environmental factors affecting development of immune-mediated diseases. Smoking parents of 366 children answered a questionnaire on their smoking behavior. Cotinine analyses were made on urine specimen from these children and 433 age-matched controls from nonsmoking homes.**

Results. **Smoking behavior had a significant impact on cotinine levels. Exclusively outdoor smoking with the door closed gave lower urine cotinine levels of children than when mixing smoking near the kitchen fan and near an open door or indoors but higher levels than controls.**

Variables of importance for smoking behavior were not living in a nuclear family (odds ratio: 2.1; 95% confidence interval: 1.1–4.1) and high cigarette consumption (odds ratio: 1.6; 95% confidence interval: 1.2-2.1).

An exposure score with controls as the reference group (1.0) gave an exposure score for outdoor smoking with the door closed of 2.0, for standing near an open door + outdoors of 2.4, for standing near the kitchen fan + outdoors of 3.2, for mixing near an open door, kitchen fan, and outdoors of 10.3, and for indoor smoking of 15.2.

Conclusion. **Smoking outdoors with the door closed was not a total but the most effective way to protect children from environmental tobacco-smoke exposure. Other modes of action had a minor effect.** ***Pediatrics*** **2004;113:e291–e295. URL: http://www.pediatrics.org/cgi/content/full/113/4/e291;** ***ETS, cotinine, children, smoking behavior, measures of precaution.***

ABBREVIATIONS. ETS, environmental tobacco smoke; ABIS, All Babies in Southeast Sweden; LLQ, lowest level of quantification; CCR, cotinine/creatinine ratio.

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Exposure to environmental tobacco smoke (ETS), both pre- and postnatal, is an important and well-known health hazard to children.[1–6] The World Health Organization[7] has estimated that ~700 million, or almost half of the children in the world, are exposed to ETS. This exposure occurs primarily in the home, although cigarette-consumption level and children's exposure outside the home must also be considered. In their homes, children depend on parents' good will and capacity to minimize their ETS exposure. Most smoking parents make efforts to protect their children. However, we cannot tell how meaningful different modes of action might be. In exceptional cases, as we reported recently,[8] a child can still be massively exposed to ETS in the home.

Numerous studies have been reported on parents' smoking and children's ETS exposure.[9–11] Most of these studies are based on invalidated questionnaires for estimation of children's ETS exposure in the home.[9] Earlier studies[12,13] raise the question of whether the effectiveness of precautions taken by parents could be confirmed with objective cotinine analyses of the children's urine.[14–16] Therefore, an instrument aiming to survey parents' smoking behavior in the home was developed and tested that demonstrated sensitivity to varying levels of exposure (A. K. Johansson, RN, MPH, A. Halling, G. Hermansson, MD, PhD, and J. Ludvigsson, MD, PhD, unpublished data, 2001).

Although cotinine measurements have their weaknesses (eg, the individual differences in the metabolism of nicotine and the relatively short half-life [~20 hours]), they have been regarded as the best available biomarker of ETS at present.[6,7] An assumption of steady state for cotinine levels is reasonable if there is a daily exposure.[17] Cotinine levels provide a valid and quantitative measure of average human ETS exposure over time.[18,19] Parenthood seems to influence smoking behavior but not smoking prevalence.[13,20] Thus, we need to determine the effectiveness of the modes of action used among smokers to protect their environment from tobacco smoke.

METHODS

Participants

We studied a subsample of 2.5- to 3 year-old children participating in a prospective cohort study, All Babies in Southeast Sweden (ABIS). The aim of ABIS is to study environmental factors affecting the development of immune-mediated diseases in children. The cohort comprises 17 055 of 21 700 (78.6%) of the children born in the southeast region of Sweden between October 1, 1997

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and September 30, 1999. Biological samples were taken at birth (cord blood, breast milk, and mothers' hair) and from the child at 1 year (blood and hair) and 2.5 and 5 years (blood, urine, stool, and hair) of age. At these time points, the parents also responded/will respond to comprehensive questionnaires.[21]

We used the questionnaires and urine specimens from the 2.5- to 3-year-old children. All parents who responded between April 2001 and January 2003 and reported that they themselves or others (ie, guests) smoke in their home (n = 687) were sent a new questionnaire on smoking behavior immediately after the ABIS questionnaire was received. The parents were asked to tell how often they smoked at the dinner table, at the television set, near an open door, at the kitchen fan, outdoors with the door closed, outdoors with the door closed and changing clothes after smoking, and using their own alternatives. They also were asked how much they smoked, how often the child was in smoky environments outside the home, and the length of time that the smoking had been performed in the described way and the level of cigarette consumption had been unchanged. Eighty-four percent (n = 578) responded. A urine sample from the child was delivered by 366 of 578 families. These 366 (47% girls) children, together with age-matched controls chosen among children in ABIS whose parents had denied smoking and smoking ever occurring in their homes (n = 433), constituted the study sample (n = 799).

The families were categorized into 5 smoking-behavior groups that were used in the analyses of the results. They were defined as:

- Outdoors: All smoking was performed outdoors with the door closed. (The group that smoked outdoors and changed clothes afterward [n = 10] are included here)
- Open door + outdoors: The group either smoked near an open door or outdoors with the door closed.
- Kitchen fan + outdoors: The group either smoked close to the kitchen fan or outdoors with the door closed
- Mixers: The group either smoked close to the kitchen fan or near an open door or outdoors with the door closed
- Indoor smoking: All who stated that smoking sometimes occurred at the dinner table and/or the television set and/or anywhere indoors and/or other places indoors. This behavior was sometimes combined with using the precautions listed above.

The urine specimens provided by the children were delivered by the parents to the local child health care centers, frozen, and then sent to the Department of Pediatrics at Linköping University. They were stored frozen (−20°C) until analyzed. Cotinine analyses were performed with capillary gas chromatography ("Method NM-018-8" by Pharmacia UpJohn, Pharmacia Consumer Healthcare, Helsingborg, Sweden). The analyses were made together with control samples of known values. The lowest level of quantification (LLQ) was based on the standard curve and was 6 ng/mL. However, we have also used, on our own responsibility, the less-reliable values between 2 and 6 ng/mL. Creatinine was measured on all samples ≥LLQ to correct for the dilution of the urine. The cotinine/creatinine ratio (CCR) was calculated as micrograms of cotinine/mole of creatinine, which then was related to cigarette consumption and smoking behavior of the parents.

Cigarette-consumption levels were recategorized into none, sporadic, 1 to 10, 10 to 20, and >20 cigarettes per day to avoid small numbers of cases in extreme groups. ETS exposure outside the home was categorized as "visiting grandparents," "visiting friends," "cafés and restaurants," "in father's home," "outdoors," or "other."

Statistics

The statistical program SPSS 11.0 for Windows (SPSS Inc, Chicago IL) was used. Because data were not distributed normally, equalities and differences between groups were shown with the Mann-Whitney U test, and correlation between variables were calculated with Spearman's correlation test. Most analyses were performed by using the CCR expressed as micrograms of cotinine/mole of creatinine. The natural logarithm of the cotinine values was used in some comparisons. Logistic regression models were used to show variables of importance for parents' smoking behavior and for children's ETS exposure. The dependent variables were dichotomized as smoking indoors or outdoors and urine CCR as above or below quantification level (6 ng/mL). $P < .05$ was considered as significant.

The study was approved by the regional Research Ethics Committee.

RESULTS

The birth of the 366 ETS-exposed children, as well as the parents responding to the tobacco questionnaire, was distributed evenly between the months of the year. Only 84% of the children lived with both their parents, 67% had a smoking mother, and 60% had a smoking father. Sixty-six percent of the fathers and 44% of the mothers had completed the lowest education level, 9-year compulsory education, or practical upper secondary school. Eleven percent of the fathers and 19% of the mothers had completed a university education. Fifteen percent of the children had 1 or both parents not born in Sweden.

Mean cigarette-consumption level on weekdays as well as on weekend days was 11 to 15 cigarettes per day. Eighteen percent of the homes reported a joint consumption of >20 cigarettes a day. Other smoked tobacco products were used sparsely (3%). Both the mother and the father smoked in the homes of 33% of the children, in 34% only the mother smoked, and in 27% only the father smoked. In 6% of the children's homes, only guests smoked.

Parents' smoking behavior as well as tobacco consumption seemed to be stable; 91% stated that they were unchanged for the past ≥12 months. Most children were seldom (70%) or never (16%) exposed to ETS in places other than the home. When exceptionally exposed outside home, the most common places were when visiting the grandparents (42%) and in cafés or restaurants (28%).

The most common precaution among the parents was smoking outdoors with the door closed: 56% (n = 206) of the parents stated that they always did so, and 3% (n = 10) always changed clothes after smoking outdoors. Three groups mixing different precautions were found: smoking near an open door combined with outdoor smoking (12%; n = 45); smoking near the kitchen fan combined with outdoor smoking (14%; n = 50); and mixing smoking near an open door, near the kitchen fan, and outdoors (7%; n = 27). One group, indoor smokers, stated that they smoked at the dinner table and/or the television set and/or anywhere indoors sometimes (8%; n = 28). Groups exclusively smoking near an open door (n = 7) or the kitchen fan (n = 9) were small and thus added to parents combining this with outdoor smoking.

Variables Associated to Smoking Behavior

Logistic regression models showed that high cigarette consumption and not living in a nuclear family

TABLE 1. Variables of Importance for Smoking Behavior

Independent Variables	Odds Ratio (95% Confidence Interval)	P Value
Cigarette consumption	1.6 (1.2–2.1)	<.01
Ethnicity	1.4 (0.9–2.2)	.11
Family situation*	2.1 (1.1–4.1)	.03
Exposure outside the home	1.5 (0.8–2.9)	.19
Size of dwelling	0.9 (0.6–1.2)	.36

* Nuclear family or broken home.

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were associated with more indoor smoking (Table 1). Other variables were of no significant importance.

The Association Between Smoking Behavior and Children's ETS Exposure

Reported smoking behavior was related to the mean logvalues of the urine/creatinine ratios (CCRs) of the children (Fig 1, bold lines). Significant differences between smoking-behavior groups were calculated. The control group had significantly lower values than all smoking groups no matter which precautions were used ($P < .03$). The "indoor-smoking" and "mixers" groups had significantly higher mean values than all other groups ($P < .02$). The same pattern was also seen if only families smoking >10 cigarettes a day were included.

In Fig 1, smoking-behavior groups were related to mean cotinine values (dashed lines). All values from 2 ng/mL, thus above the 55th percentile, were included, and the cotinine values here were unadjusted for creatinine. The extended use of low cotinine values made the differences between the groups more obvious, although it did not change the results.

A significant correlation was seen between CCR and ethnicity, size of dwelling, cigarette-consumption level, which of the parents smokes, and smoking behavior. In logistic regression models, smoking behavior was the only variable of importance for CCR level (Table 2). Cigarette consumption had no significant impact on the children's CCR levels, but the higher cigarette consumption, the less-extensive precautions the parents used.

Exposure Score

An exposure score illustrating the effect of different smoking behaviors was calculated. A logistic regression model was made with cotinine dichotomized as described above or <6 ng/mL as a dependent variable and smoking behavior as independent and categorical variables. Score levels were depicted as odds ratios in Table 3.

TABLE 2. Variables of Importance for ETS Exposure

Independent Variables	Odds Ratio (95% Confidence Interval)	*P* Value
Smoking behavior	1.5 (1.2–1.9)	<.001
Cigarette consumption	1.3 (0.9–2.0)	.13
Ethnicity	1.6 (1.0–2.6)	.06
Family situation*	1.0 (0.4–2.4)	.99
Which parent smokes	1.2 (0.8–1.8)	.31
Exposure outside the home	1.1 (0.4–2.7)	.86
Size of dwelling	0.8 (0.5–1.2)	.21

* Nuclear family or broken home.

DISCUSSION

The results in this study indicate that smoking behavior in the home is of significance for children's ETS exposure.

Most often, the urine specimens were delivered some weeks before the parents got the smoking-behavior questionnaire. This means that parents' smoking behavior was not influenced by the questionnaire before the urine sample was delivered. The described smoking behavior and cigarette consumption were reported to be stable.

Cotinine levels can be expected to reflect average group exposure[19] and have been shown to serve well as estimates for settings that do not change often.[9,11] The relatively high LLQ (6 ng/mL urine) in the cotinine analyses made some of the exposure groups small. This made it difficult to discriminate between all the smoking-behavior groups by using the calculated CCR values. Cotinine values of 2 to 6 ng/mL were used to illustrate mean values for exposure groups (Fig 1). (These values were not validated, and Pharmacia could not guarantee their reliability [J.

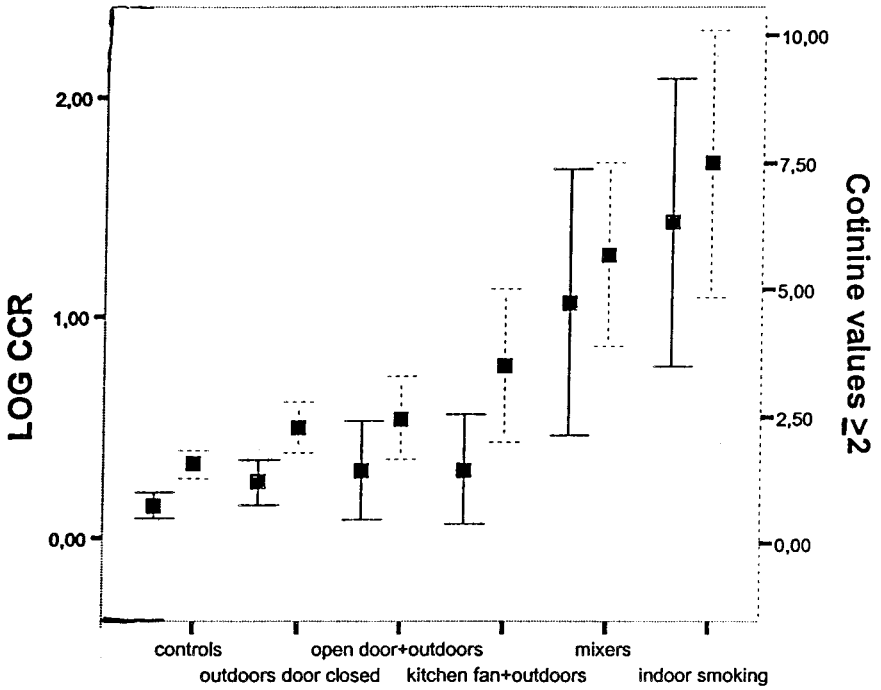

Fig 1. Children's cotinine/creatinine logvalues (bolded lines) and mean values of cotinine (dashed lines) related to parents' smoking behavior. Cotinine levels ≥ LLQ 6 ng/mL were included (82 children, 10.3%), and the remaining were given the value '1' in the calculation. Values ≥ 2 ng/mL were included (251 children, 56%), and the remaining were given the value '0' in the calculation. Cotinine was nonadjusted for creatinine.

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TABLE 3. Exposure Score for Different Smoking Behaviors

Smoking Behavior	Score, Odds Ratio	95% Confidence Interval	*P* Value
Controls (n = 433)	1		
Outdoors with door closed* (n = 216)	1.99	1.1–3.6	.015
Open door + outdoors (n = 45)	2.39	0.9–6.1	.069
Kitchen fan + outdoors (n = 50)	3.23	1.3–7.9	.010
Mixers (n = 27)	10.32	4.3–24.8	<.001
Indoor smokers (n = 28)	15.09	6.6–35.3	<.001

* The group of those smoking outdoors and always changing clothes afterward (n = 10) is included in "outdoors with door closed."

Birgersson and Pharmacia, verbal communication, 2003].) They do not alter results given by the CCR levels but make differences more obvious.

The children were all of the same age, 2.5 to 3 years, and differences in metabolism and proximity to the parents, related to age, should not have to be considered. There is no risk for confounding from nicotine through breast milk. The home as the major ETS source was obvious, because only 12% of the children were in smoky environments outside the home every week (and just 2% every day). This reflects the legislation in Sweden, which forbids smoking in most milieus where children are. The most common source of ETS exposure was reported to be the grandparents, results in concordance with Hopper and Craig.[22] This was a generation with higher smoking prevalence.[23]

Parents' cigarette consumption has been shown to influence children's ETS exposure.[1] In this study, consumption was shown to influence smoking behavior, which in turn influenced ETS exposure.

Because Sweden has the lowest smoking prevalence (20% among adults) in Europe,[7] and most parents were aware of the importance of protecting children from ETS, the level of exposure is probably lower than in many other countries. Almost 60% of the parents in this study stated that they always smoked outdoors with the door closed, which significantly decreases ETS exposure to their children. In countries in which smoking is less restricted in society, the smoking behavior in the home might be less important for the children's cotinine levels.

The groups with parents only smoking in an open door (n = 9) or only near the kitchen fan (n = 9) or always changing clothes after outdoor smoking (n = 10) were small, and the protection effect of these behaviors was difficult to evaluate. They were included in the groups combining these behaviors with smoking outdoors with the door closed. However, it was obvious that any deviation from smoking outdoors was reflected in the cotinine levels of the children, and the results indicated that smoking near an open door was somewhat better than smoking near the kitchen fan.

Although outdoor smoking with the door closed seemed to be the best precaution, there was still a significant difference between this group and the controls. There was no known "safe" level of ETS exposure. The results can be compared with the results of the study by Johansson et al,[12] in which 1- to 2-year-old children of only outdoor-smoking parents tended to have a higher prevalence of ear infections and respiratory symptoms than children of nonsmokers but less than children of indoor smokers.

CONCLUSIONS

The results of this study could contribute to a more-valid counseling of smoking parents. If parents cannot stop smoking, they should handle their smoking in a way that limits their children's ETS exposure. Nurses in child health care, as well as others meeting parents in consulting situations, should ask for smoking behavior when estimating the children's level of ETS exposure. They should also explain that smoking outdoors with the door closed is a meaningful way of ETS protection, whereas other precautions indoors have little or no effect. Whether outdoor smoking is as good as nonsmoking remains to be shown.

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