Exposure of Young Infants to Environmental Tobacco Smoke: Breast-Feeding among Smoking Mothers

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ABSTRACT

Objectives. This study examined the degree to which breast-feeding and cigarette smoking by mothers and smoking by other household members contribute to the exposure of infants to the products of tobacco smoke.

Methods. The subjects were 330 mother–infant pairs derived from a cohort of 1000 pairs enrolled in a longitudinal study of the pulmonary effects of prenatal and postnatal smoking. The main outcome measure was corrected urinary cotinine levels.

Results. Urinary cotinine levels were 10-fold higher in breast-fed infants of smoking mothers than among bottle-fed infants of smoking mothers. Among infants of nonsmoking mothers, urine cotinine levels were significantly increased in infants living in homes with other smokers; in this group there was no significant difference between bottle-fed and breast-fed infants. Infants whose mothers smoked in the same room as the infant had only nonsignificant increases in cotinine levels compared with infants whose mothers restricted their smoking to other rooms.

Conclusions. Breast-fed infants of smoking mothers have urine cotinine levels 10-fold higher than bottle-fed infants whose mothers smoke, suggesting that breast-feeding, rather than direct inhalation of environmental tobacco smoke, is the primary determinant of cotinine levels in infants whose mothers smoke. (*Am J Public Health.* 1998;88:893–896)

Introduction

Exposure to cigarette smoking, both in utero and by passive exposure to environmental tobacco smoke in infancy, has been linked to numerous adverse health outcomes in young children, from prematurity and increased infant mortality[1,2] to higher rates of asthma[3–5] and sudden infant death syndrome.[6,7] Breast-feeding is one form of passive exposure that has recently received close scrutiny. The presence of nicotine and cotinine in the breast milk of nursing mothers has been documented.[8–12] Infants exposed to environmental tobacco smoke and breast-fed by smoking mothers have been found to have urine cotinine levels ranging from 2 to 10 times as high as those of environmental tobacco smoke–exposed, bottle-fed infants.[11,13–17] Despite uncertainty about the magnitude of the effect, breast-feeding does contribute to the nicotine exposure of infants. Whether smokers in the home other than the mother might add to infants' exposure via lactating mothers' exposure to environmental tobacco smoke has not been explored.

In this study we compared urine cotinine levels from breast-fed and bottle-fed infants of smoking mothers. We also examined the contribution of breast-feeding to urine cotinine levels among infants of nonsmoking mothers who are exposed to environmental tobacco smoke by other household members, and whether restriction of location of smoking at home affects infants' exposure.

Methods

The subjects were derived from a cohort of 1000 mother–infant pairs who enrolled early in the mother's pregnancy in a longitudinal study of the effects of prenatal and postnatal smoking on the pulmonary function and respiratory illness experience of infants and children. This investigation includes 330 mother–infant pairs from whom an infant urine specimen was collected in the first 12 months of life.

Mother–infant pairs who enrolled in this study had obstetric and pediatric care at a single urban clinic in East Boston, Mass. Those presenting for prenatal care between March 25, 1986, and October 1, 1992, prior to the 20th week of gestation were eligible for enrollment. Women who did not speak either English or Spanish, whose age was less than 18 years, and who did not plan to have pediatric follow-up at the clinic were excluded. At enrollment of the mother, detailed data were obtained on age, race, last menstrual period, medical and obstetrical history, past and current smoking history, and environmental tobacco smoke exposure from other household members. Spirometry was performed, and urine specimens were obtained for determination of cotinine levels. Further information about smoking practices and urine specimens for cotinine analysis were collected at all subsequent

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prenatal visits and at delivery. Information was collected by trained interviewers using standardized questionnaires at the clinic.

Hospital delivery records were reviewed to collect information on infants' birthweight and length. At all well-baby pediatric visits, participating mothers were interviewed to elicit data on smoking patterns of the mother and other members of the household and any respiratory symptoms experienced by the infant. Urine was collected from a nonrandom sample of infants at 1 or more visits during the first 12 months of life via infant urine collection bags. Financial constraints limited testing to 330 infants. Urine specimens were stored at 4°C until analyzed. The specimens were analyzed for cotinine concentration by radioimmunoassay[18] in the order in which they were collected. Specimens were batched together by date of collection. The lower limit of detection was 3 ng/mL, and both interassay and intra-assay variation were less than 10%. Urine cotinine values were corrected by urine creatinine level (nanograms cotinine per milligram creatinine) to adjust for differences in urine concentration.

We defined smoking mothers as those who reported smoking at least 1 cigarette per day at the time of the visit or those whose corrected urine cotinine level exceeded 200 ng/mg creatinine at the visit, a level unlikely to represent environmental tobacco smoke exposure. Infants were considered breast-fed if the mother reported any current breast-feeding (supplemented or not) at the time of the urine collection. We categorized infants as environmental tobacco smoke–exposed by nonmaternal smokers in the household if they lived in a home or spent significant time (>2 hours, 2 times a week) with a smoker. Finally, we stratified infants of smoking mothers by whether these mothers smoked in the same room as the infant at any time.

We report median values and interquartile ranges for each group. Comparisons between groups are made with the Wilcoxon rank sum test because the distributions of urine cotinine values are positively skewed.

Results

Between March 1986 and October 1992, 1000 pregnant women were enrolled in the study. Prenatal follow-up was obtained for 927, with a mean of 6.1 visits, and infant follow-up was obtained for 817, with a mean of 5.5 visits. At least 1 urine specimen for cotinine measurement was obtained from 330 infants before the age of 12 months. Comparison of demographic characteristics of those mother–infant pairs from whom urine was collected vs those without urine specimens revealed no significant differences with respect to maternal age, race, education level, percentage of households with smokers, percentage who breast-fed, infant birthweight or length, or infant age at enrollment.

Urine cotinine levels ranged from 0 to 15 200 ng/mg creatinine (median = 102 ng/mg; interquartile range = 27, 492 ng/mg). Cotinine levels in infants of smoking mothers were significantly higher than levels in infants of nonsmoking mothers (400 ± 469 ng/mg vs 53 ± 31 ng/mg; $P < .0001$). There was no significant difference in urine cotinine levels among infants of nonsmokers with regard to type of feeding (Table 1). However, breast-fed infants of smoking mothers had median levels 10-fold higher than those of bottle-fed infants of smoking mothers.

We also evaluated the influence of exposure to nonmaternal household smokers on infant urine cotinine levels. Infants of nonsmoking mothers who were exposed to environmental tobacco smoke by another household member had significantly higher cotinine levels compared with their unexposed counterparts, regardless of type of feeding; among infants in this group, there was no significant difference in urine cotinine levels between breast-fed and bottle-fed infants. When we compared infants whose mothers were smokers, the presence of another smoker in the home resulted in a nonstatistically significant increase in the infants' cotinine levels. The magnitude of this increase was nearly 2-fold and approached statistical significance for bottle-fed infants. The small number of breast-fed infants of smoking mothers (n = 13) was not adequate to permit a precise estimation of the influence of other smokers in the home on infant urine cotinine levels, although point estimates were higher in homes with other smokers.

Finally, recognizing that many mothers restrict their smoking in the home to areas away from the infant, we attempted to determine whether the location of maternal smoking in the home significantly affected exposure. Studying only infants whose mothers were smokers, we found that infants whose mothers smoked in the same room had nonstatistically significant increases in urine cotinine levels, regardless of feeding type, compared with infants whose mothers always smoked in rooms away from the infant (Table 2).

Discussion

Data from this investigation indicate that for infants of smoking mothers, breast-feeding (rather than environmental tobacoo smoke exposure by direct inhalation) is the most important determinant of urine cotinine levels. Thus, the question is raised of whether the deleterious constituents of

TABLE 1—Infant Urine Cotinine Levels, by Maternal Smoking Status, Type of Feeding, and Presence of Other Smokers in the Home: East Boston, Mass, March 1986 through October 1992

Maternal Smoking Status	Type of Feeding	Infant Urine Cotinine, Median (IQR), ng/mg Creatinine	*P*	Other Smokers in the Home	Infant Urine Cotinine, Median (IQR), ng/mg Creatinine	*P*
Nonsmoker (n = 224)	Bottle (n = 117)	60 (22, 140)	NS[a]	No (n = 83)	42 (18, 125)	<.0001
				Yes[b] (n = 34)	127 (47, 263)	
	Breast (n = 107)	50 (29, 98)		No (n = 76)	41 (13, 74)	<.0001
				Yes[b] (n = 31)	65 (39, 194)	
Smoker (n = 103)	Bottle (n = 90)	361 (239, 593)	<.0001	No (n = 39)	250 (167, 400)	.07
				Yes (n = 51)	464 (291, 619)	
	Breast (n = 13)	4207 (1350, 4700)		No (n = 6)	3025 (1333, 4796)	.83
				Yes (n = 7)	4207 (1740, 4667)	

Note. IQR = interquartile range.
[a]Not significant at $P = .05$.
[b]Comparison between bottle-fed and breast-fed infants of nonsmokers exposed to another smoker in the home, $P = .15$.

TABLE 2—Infant Urine Cotinine Levels and Maternal Smoking in Same Room (Smoking Mothers Only): East Boston, Mass, March 1986 through October 1992

Type of Feeding	Mother Smokes in Same Room	Infant Urine Cotinine, Median (IQR), ng/mg Creatinine	*P*
Bottle (n = 83)	No (n = 34)	350 (239, 486)	
	Yes (n = 49)	413 (280, 608)	.09
Breast (n = 13)	No (n = 6)	2018 (1038, 4207)	
	Yes (n = 7)	4667 (1740, 4797)	.43

Note. IQR = interquartile range.

tobacco smoke are transferred directly to the nursing infant, and whether breast-feeding by infants of smoking mothers may be responsible for some of the adverse health outcomes in these infants that have been attributed to environmental tobacco smoke inhalation.

Earlier investigations suggested that breast-feeding by smoking mothers contributes to elevation of urine cotinine levels in their infants.[9,11–17] However, the present study suggests that the degree of exposure via breast milk is much more substantial than previously appreciated. This difference cannot be attributed to heavier smoking by breast-feeding mothers, since number of cigarettes smoked per day was no different between bottle-feeding and breast-feeding mothers. It is interesting that in all categories, urine cotinine levels in infants in our study are 2- to 8-fold higher than in previous reports. The contrasting results may be explained in part by the use of less sensitive methods to determine urine cotinine levels[11] or by failure to adjust for urine creatinine concentration[14] in these previous studies, but they may also reflect higher smoking rates in the communities where our subjects lived. Also, the average age of breast-fed infants at the time of urine collection was 2 weeks younger than that of bottle-fed infants (6 weeks vs 8 weeks), possibly influencing cotinine levels.

Nonmaternal household smokers have also been reported to affect infant nicotine exposure.[7] We found that infants exposed to nonmaternal household smokers had increased urine cotinine levels. These increases were highly significant in infants of nonsmoking mothers and approached statistical significance in infants of smokers who were bottle-fed. There were too few breast-fed infants of smoking mothers to assess the impact of additional household smokers with any precision. We further investigated the related but separate issue of whether the tobacco exposure of infants in households with nonmaternal smokers was augmented by breast-feeding via exposure of the lactating mother. No evidence of an increase in urine cotinine levels in these breast-fed infants was observed; point estimates were lower in breast-fed than in bottle-fed infants. One possible explanation for this finding may be that the level of household environmental tobacco smoke exposure for women who breast-feed is less than that for those who bottle-feed (family members may smoke less or be restricted in terms of location of smoking in the home); another may be chance, given the small number of subjects in several of these categories.

This study failed to identify any significant difference in urine cotinine levels for infants whose mothers did or did not smoke in the same room as the child, although small increases were found for both bottle-fed and breast-fed infants whose mothers did not restrict their smoking to rooms away from the child. Other studies have reported that location of maternal smoking in the home is a significant factor in children's exposure.[14,15] The results of the present study suggest that efforts to restrict smoking location in the home may have only a small or negligible impact on a child's exposure to environmental tobacco smoke. Alternative explanations could be the lack of power to detect a significant association or inaccuracy of reporting by the mothers.

It is important to recognize that cotinine is only a quantitative biomarker for smoking, and it is unlikely that cotinine or its parent compound, nicotine, is responsible for all of the adverse outcomes associated with smoking. The identity of the compounds in tobacco smoke that are actually responsible for the adverse health impact on infants and children and the degree to which their concentrations in breast milk are correlated with cotinine are largely unknown. In addition, in our study population, the category of smoking mothers who breast-fed their infants comprised only 13 women, and as a group they may differ in other, unrecognized ways that may account for some of the differences we found among our groups.

In summary, we found that infants of smoking mothers have significantly more exposure to the products of tobacco smoke than do infants whose mothers do not smoke and that breast-feeding dramatically increases this exposure. It is possible that adverse health consequences in children previously attributed only to environmental tobacco smoke exposure by inhalation may also result from exposure to both environmental tobacco smoke and the breast milk of smoking mothers. The relative importance of inhalational environmental tobacco smoke exposure vs ingestion via breast milk in predicting respiratory illness and other conditions in infants associated with early-life smoking exposure is an important public health issue that merits further exploration. Health care providers need to be as diligent in encouraging mothers to stop smoking after birth as in the prenatal period, especially those mothers who intend to breast-feed, and those mothers unable to stop smoking should be informed of the possibility that harmful chemicals derived from tobacco smoke may be transmitted to their infants via breast milk. □

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