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Pediatrics; originally published online December 13, 2010;
DOI: 10.1542/peds.2010-2046

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American Academy of Pediatrics

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Tobacco-Smoke Exposure in Children Who Live in Multiunit Housing

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KEY WORDS

secondhand smoke, passive smoking, environmental tobacco smoke, multiunit housing, apartment

ABBREVIATION

NHANES—National Health and Nutrition Examination Survey

Dr Wilson participated in all aspects of the study, including study conception and design, interpretation of data, drafting and revising of the manuscript, and supervising the statistical analysis; Dr Klein made substantial contributions in the design of the study, interpretation of the data, critical revision of the manuscript, and supervision; Mr Blumkin provided the statistical analyses for the study, acquired the data, and assisted with the drafting and revision of the manuscript; Dr Gottlieb provided important information on the medicolegal and policy implications of the study, assisted in the interpretation of the data from the policy perspective, contributed to the revision of the manuscript, and supervision; and Dr Winickoff provided overall supervision for the direction of the manuscript and was involved in the conception and design, analysis and interpretation of data, and critical review of the manuscript.

www.pediatrics.org/cgi/doi/10.1542/peds.2010-2046

doi:10.1542/peds.2010-2046

Accepted for publication Sep 29, 2010

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.



WHAT'S KNOWN ON THIS SUBJECT: Exposure to secondhand tobacco smoke is an important cause of morbidity and mortality among children, even at low levels of exposure. In a recent national sample, 54% of children who did not live with a smoker showed measureable amounts of cotinine.



WHAT THIS STUDY ADDS: Children who live in homes in which no one smokes inside have a 45% increase in cotinine levels if they live in apartments compared with detached homes. Multiunit housing may be a significant source of secondhand tobacco-smoke exposure for children, at levels associated with morbidity.

abstract

OBJECTIVE: There is no safe level of secondhand tobacco-smoke exposure, and no previous studies have explored multiunit housing as a potential contributor to secondhand tobacco-smoke exposure in children. We hypothesized that children who live in apartments have higher cotinine levels than those who live in detached homes, when controlling for demographics.

METHODS: We analyzed data from the 2001–2006 National Health and Nutrition Examination Survey. The housing types we included in our study were detached houses (including mobile homes), attached houses, and apartments. Our study subjects were children between the ages of 6 and 18 years. Cotinine levels were used to assess secondhand tobacco-smoke exposure, and those living with someone who smoked inside the home were excluded. χ^2 tests, *t* tests, and Tobit regression models were used in Stata. Sample weights accounted for the complex survey design.

RESULTS: Of 5002 children in our study, 73% were exposed to secondhand tobacco smoke. Children living in apartments had an increase in cotinine of 45% over those living in detached houses. This increase was 212% ($P < .01$) for white residents and 46% ($P < .03$) for black residents, but there was no significant increase for those of other races/ethnicities. At every cutoff level of cotinine, children in apartments had higher rates of exposure. The exposure effect of housing type was most pronounced at lower levels of cotinine.

CONCLUSIONS: Most children without known secondhand tobacco-smoke exposure inside the home still showed evidence of tobacco-smoke exposure. Children in apartments had higher mean cotinine levels than children in detached houses. Potential causes for this result could be seepage through walls or shared ventilation systems. Smoking bans in multiunit housing may reduce children's exposure to tobacco smoke. *Pediatrics* 2011;127:85–92

Tobacco-smoke exposure causes illness in children, including asthma^{1,2} and respiratory infections,³ and has been associated with sudden infant death syndrome,⁴ metabolic syndrome,⁵ and otitis media.⁶ There is no safe level of exposure to tobacco smoke.⁶ Very low levels of tobacco-smoke exposure have been associated with attenuated endothelial function in children,⁷ as well as decreased scores on reading, math, and block-design tests of cognitive function.⁸ Morbidity has been documented in those with the lowest levels of cotinine (0.015–0.5 ng/mL), and these children have greater rates of conduct disorder.⁹ Even brief exposure to ambient tobacco smoke can decrease lung function and cause persistent elevations in inflammatory cytokines.¹⁰

Parental smoking is the most common source of secondhand tobacco-smoke exposure for children. In 1 study,⁶ 25% of children aged 3 to 11 years reportedly lived with at least 1 smoker. However, 60% of the children in the study had detectable levels of cotinine,⁶ a metabolite specific to tobacco smoke. Other known exposures do not explain all of the 54% of children with elevated cotinine levels who had no identified smoker in the home.^{11,12} These children, therefore, must have been exposed to other sources of tobacco smoke that are not being captured by parent report.

Tobacco smoke can migrate through walls, ductwork, windows, and ventilation systems of multiunit dwellings and potentially affect residents in other units far removed from the smoking area.^{11,13} In addition to the dissemination of this secondhand smoke into other apartments, tobacco toxins may persist on and be absorbed from surfaces in the indoor environment well beyond the period of active smoking.^{14–16} This “thirdhand smoke”¹⁷ may re-emit deposited volatile compounds

and particulate matter on indoor surfaces, and particulate matter in dust may be resuspended into the air as respirable suspended particulate matter.^{14,18,19} In addition to inhalation, there are other potential exposure routes, such as ingestion, that are particularly likely in children.¹⁵

Recent public health efforts to reduce tobacco smoke exposure have concentrated on banning smoking in public places outside of the home, including workplaces, restaurants, and bars, leading to improved air quality in those locations.²⁰ However, in New York City, where the prevalence of cigarette smoking is lower than the national average and there are strict smoking bans in bars and restaurants, a recent study²¹ found that the prevalence of elevated cotinine levels among non-smoking adults was higher than the national average. The authors speculated that contamination of multiunit buildings with tobacco smoke from other units may contribute to these surprisingly high cotinine levels, although no direct measurement of nicotine in the air was performed. Some municipalities have proposed legislation to reduce or ban smoking in apartment buildings,^{22,23} and some public-housing authorities have implemented smoke-free policies.²⁴ In 2009, the Department of Housing and Urban Development encouraged public-housing authorities to ban smoking in low-income multiunit housing.²⁵ There also have been reports of privately owned housing units that have banned smoking because of the potential health risks, increased costs associated with removing tobacco residue from apartments after smoking tenants leave, and the need to relocate tenants disturbed by neighbors who smoke.²⁶ A recent study²⁷ of low-income apartments in Boston found that 94% had detectable air nicotine levels, includ-

ing 89% of apartments inhabited by nonsmokers.

There still is a lack of scientific evidence about whether smoking in multiunit housing accounts for the presence of tobacco-smoke biomarkers in children who live in a home with no adult smokers. In the current study, we used data from the 2001–2006 National Health and Nutrition Examination Survey (NHANES) to examine the association between types of housing and cotinine levels in children. We hypothesized that children who live in apartments have a higher cotinine level than children who live in detached homes and that this relationship persists when controlling for poverty and race/ethnicity.

METHODS

The NHANES

The NHANES used a multistage sampling design that included a questionnaire (parent and teen reports), physical examination, and blood and urine samples. Survey components were administered to a proxy respondent for children up through 15 years of age, whereas children aged 16 to 18 years completed the survey responses themselves unless they were cognitively unable. Demographic variables included age, gender, and self-report of race and ethnicity. In addition, the federal poverty-level ratio was calculated. A federal poverty-level ratio below 1 means the family lives below the poverty level, whereas a ratio above 1 means they live above the poverty level.

Housing Type

Interviewers assessed housing type and asked respondents to verify their impressions. The response categories included detached house (“a one-family house detached from any other house”), apartment, attached house (“a one-family house attached to one

or more houses”), mobile home, dormitory, or other. For these analyses, mobile homes were combined with detached houses. The other 2 categories analyzed were attached houses and apartments. Subjects reported to be living in dormitory or other settings were excluded.

Tobacco-Smoke Exposure: Survey

The NHANES assessed household smoking with the question, “Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?” For those households in which no one was reported to smoke inside the home, no other information was available about smoking status, home or car smoking bans, other sources of exposure, or outside smoking behaviors. Preliminary analyses on the full sample showed that children who lived in a house where anyone smoked inside had exposure levels that overwhelmed any relationship between cotinine level and housing type; we therefore limited the sample in this study to children who lived in a household in which no member was reported to smoke inside the home. Likewise, we excluded any child who admitted to smoking.

Tobacco-Smoke Exposure: Biochemical Verification

Serum cotinine was measured using isotope dilution-high-performance liquid chromatography/atmospheric-pressure chemical ionization tandem mass spectrometry; the detectable limit in the NHANES is 0.015 ng/mL, and the coefficient of variability is 2.5%.²⁸ Tobacco-smoke exposure was defined as a cotinine level of ≥ 0.015 ng/mL, although comparisons also were made at cotinine cutoff levels of 0.05, 1.0, and 2.0. A sensitivity analysis was done to determine whether the results were affected by the testing variability. In addition, because plants from the nightshade family contain low levels of

natural nicotine,²⁹ we tested the model controlling for intake of tomatoes, eggplant, and potatoes for 96.3% of the sample for whom these data were available. For this subsample, there were no significant differences in cotinine levels when vegetable intake was included; therefore, we continued our analysis with the full sample.

Analysis

χ^2 and *t* tests were conducted to analyze bivariate data. Analyses that included cotinine level as a continuous dependent variable used Tobit regression models to account for the censoring of the data at the lower cutoff of 0.015 ng/mL.³⁰ Race/ethnicity by housing-type interactions were tested using Tobit regression for cotinine levels as the outcome and logistic regression when using tobacco exposure as the outcome. Cotinine levels were analyzed using log transformations and geometric means to normalize the skewed distribution. The assumption of linearity among all continuous covariates was checked. Stata was used to control for the complex sample weighting and design.³¹ This secondary analysis of NHANES data was approved as exempt by the University of Rochester Research Subjects Review Board.

RESULTS

There were 5002 children surveyed in the NHANES who were living in a home in which no one smoked inside (81%). Compared with the children who were living in a home in which someone smoked, those who were living in a home in which no one smoked were more likely to live in a detached house (81.4% vs 73.4%) and less likely to live in an apartment (11.6% vs 16.7%; $P < .02$). They also were more likely to be over 12 years of age (46.9% vs 38.8%; $P < .01$), male (52.2% vs 46.7%; $P < .01$), and Hispanic (20.5% vs 8.8%) rather than black (14.0% vs 22.1%) or white (59.1% vs 63.7%; $P < .001$ for all

comparisons). In addition, children who were living in a home in which no one smoked inside were more likely to be more than 400% of the federal poverty-level ratio (28.5% vs 11.3%) and less likely to be 100% or lower (17.8% vs 34.1%; $P < .001$).

The remaining results pertain to those children who were living in a home in which no one smoked inside. The demographic characteristics of this sample are presented in Table 1. The overall geometric mean cotinine level among these children was 0.036 ng/mL (95% confidence interval: 0.030–0.043); cotinine levels were higher among children under 12 years of age, black children, and those living below the federal poverty levels. Mean cotinine levels among those who were living in apartments (0.075 ng/mL) were higher than in those who were living in detached houses (0.053 ng/mL; $P < .01$) and detached houses (0.031 ng/mL; $P < .001$). Overall, using the detectable limit of 0.015 ng/mL as the tobacco-exposure cutoff, 84.5% of children who were living in apartments had a cotinine level that indicated recent tobacco-smoke exposure, compared with 79.6% of children who were living in attached houses and 70.3% who were living in detached houses ($P < .001$) (Fig 1). Sensitivity analysis, using the higher cutoff of 0.05 ng/mL, showed exposure rates of 56.4% for children who were living in apartments, 47.0% for children who were living in attached homes, and 36.1% for children who were living in detached homes ($P < .0001$). Figure 2 shows the proportion of children by housing type who were unexposed at different cotinine levels, demonstrating the persistent and consistent decrease in the percentage exposed for those who live in detached homes.

The percentage of children who were exposed to tobacco smoke in different housing types varied significantly by

TABLE 1 Demographic Characteristics of the Sample

Variable	Weighted % (n = 5002)	Geometric Mean of Cotinine (95% Confidence Interval)	P
Housing type			<.001
Detached house	81	0.031 (0.026–0.038)	
Attached house	7	0.053 (0.035–0.079)	
Apartment	12	0.075 (0.062–0.091)	
Gender			.037
Male	52	0.039 (0.031–0.048)	
Female	48	0.033 (0.028–0.040)	
Age			.014
<12 y	53	0.040 (0.033–0.048)	
≥12 y	45	0.032 (0.026–0.039)	
Race/ethnicity			<.001
Black	14	0.105 (0.090–0.122)	
Hispanic	21	0.026 (0.022–0.031)	
White	59	0.031 (0.025–0.040)	
Other	6	0.033 (0.020–0.0501)	
Federal poverty-level ratio			<.001
≤100	18	0.085 (0.068–0.105)	
101–200	21	0.054 (0.041–0.072)	
>200–300	18	0.031 (0.023–0.043)	
>300–400	15	0.028 (0.021–0.036)	
>400	28	0.020 (0.016–0.025)	

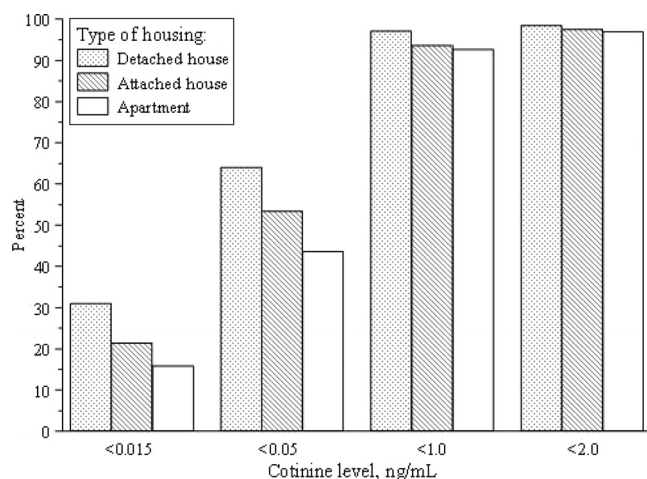


FIGURE 1

Percentage of children who are unexposed by housing type and cotinine cutoff. The y-axis shows the proportion of children who are unexposed at 3 different cotinine cutoff levels. These levels, displayed on the x-axis, are <0.015, <0.05, <1, and <2 ng/mL cotinine. The types of bars for each of the different housing types: detached house, attached house, and apartment.

race/ethnicity (Table 2). The highest level of exposure was found in white children who were living in apartments (99%), followed by black children who were living in apartments (96%); Hispanic and other race/ethnic groups had much lower levels of exposure (73% and 64%, respectively; $P < .001$). Black children who were living in attached houses had exposure rates similar to those who were living in

apartments (92%), whereas the rates were much lower for white (76%), Hispanic (70%), and other (80%) children ($P < .05$). Black children who were living in detached houses also had higher rates of exposure (89%) than white (68%), Hispanic (66%), and other (74%) children ($P < .001$). When we performed a stratified analysis of children in the wealthiest category (those more than or equal to 4 times the fed-

eral poverty level), we found that the relationship between exposure and multiunit housing persisted (data not shown).

In the unadjusted Tobit regression model, with the natural log of cotinine as the dependent variable, the percentage increase in cotinine levels for children who were living in apartments compared with children who were living in detached homes was 140% (95% confidence interval: 87–301); for children living in attached homes compared with those living in detached homes, the percentage increase was 69% (95% confidence interval: 21–135). In the Tobit model adjusted for age, gender, and federal poverty-level ratio, including race/ethnicity and housing-type interactions (Table 3), white children who were living in apartments had a 212% increase in cotinine levels over those who were living in detached houses ($P = .003$); black children who were living in apartments had a 46% increase ($P < .05$) in cotinine levels. Differences for other race/ethnic categories were not significant.

DISCUSSION

The majority of US children who live in homes where no one smokes inside have biochemical evidence of tobacco-smoke exposure, and cotinine levels are significantly higher in children who live in apartments, compared with those who live in detached houses. Although it is likely that some of this excess exposure is from family members who smoke only outside of the home but carry in tobacco residue on their clothes, this is unlikely to explain all of the discrepancy. In addition, our data are consistent with the findings from Kraev et al,²⁷ which showed that 89% of low-income apartments with no smokers had detectable air nicotine concentrations.

The finding that children are at risk for tobacco-smoke exposure in apart-

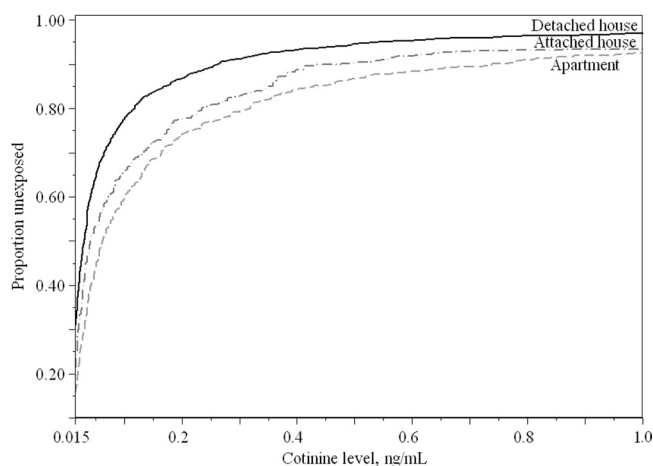


FIGURE 2

Percentage of children unexposed by housing type and cotinine level. The y-axis shows the proportion of children who are unexposed at different cotinine levels, which are displayed on the x-axis. The 3 lines represent each of the different housing types: detached house, attached house, and apartment (dashed line).

TABLE 2 Percentage of Children Exposed to Tobacco Smoke According to Race/Ethnicity and Housing Type

Variable	Race/Ethnicity (n)	Percentage Exposed (95% Confidence Interval)	P
Detached house	Black (885)	89 (85–92)	<.001
	Hispanic (1356)	66 (60–71)	
	Other (149)	74 (60–86)	
	White (1170)	68 (61–74)	
Attached house	Black (226) ^a	92 (83–96)	<.05
	Hispanic (133)	70 (52–83)	
	Other (22) ^{a,b}	80 (54–94)	
	White (64)	76 (61–86)	
Apartment	Black (385) ^a	96 (92–98)	<.001
	Hispanic (473)	73 (64–81)	
	Other (34) ^a	64 (40–82)	
	White (49) ^{a,b}	99 (91–99)	

^a Relative SE is >30%.

^b Inadequate sample size.

TABLE 3 Tobit Regression Model Predicting the Percentage Change in the Geometric Mean of Cotinine

Variable	Housing Type	Percentage Change (95% Confidence Interval)	P
Federal poverty-level ratio		−28.3 (−34.6 to −21.5)	<.001
White	Detached house	0.0	
	Attached house	−5.5 (−45.4 to 63.6)	.838
	Apartment	212.2 (50.3–548.7)	.003
Black	Detached house	0.0	
	Attached house	40.0 (−0.03 to 96.8)	.052
	Apartment	45.6 (5.4–101.1)	.024
Hispanic	Detached house	0.0	
	Attached house	4.7 (−38.1 to 76.9)	.863
	Apartment	7.8 (−23.0 to 50.9)	.656
Other	Detached house	0.0	
	Attached house	12.7 (−75.9 to 427.1)	.877
	Apartment	−18.5 (−71.1 to 130.2)	.694

Other variables included gender and age; includes the housing-by-race interaction.

ments may accelerate the current trend of limiting smoking in multiunit housing. One of the public health benefits seen from the restriction of smoking in the workplace has been a reduction in smoking rates and number of cigarettes smoked. Restrictions in multiunit housing may have a similar effect on residents; however, implementing these restrictions without providing smoking-cessation assistance for residents also might create a significant burden for low-income smokers. Adult residents of Department of Housing and Urban Development–funded housing who are uninsured will need access to free cessation programs, such as those offered by the national network of quitlines.³²

Banning smoking in multiunit dwellings by property owners or by regulation would be the obvious way to mitigate contamination and children's exposure to tobacco toxins. Concern has been raised that dictating what can be done in a private dwelling is an infringement on personal privacy and liberty; however, this argument holds only if smoking in an adjacent apartment has no impact on one's neighbors. Legal doctrine supports restrictions on private behavior if there are consequences for others, such as noise levels, noxious odors, or release of toxic chemicals.^{33,34} Tobacco smoke can be categorized both as a noxious odor and a toxic chemical. In addition, there is a strong probability that exposure may result in physical harm, particularly for children with underlying illnesses such as asthma. A recent analysis³⁴ addressing smoke-free public housing argued that phasing in such a policy as new leases were signed and existing ones renewed would be justified on legal and social justice grounds. The association between living in an apartment and child cotinine levels provides additional

support to this exposure-reduction strategy. Smoke-free policies should recognize that tobacco smoke drifts and can be measured in high quantities more than 20 feet from an outdoor source.³⁶ Because restriction inside apartments may encourage increased smoking in common areas where exposure to nonsmokers still may occur, these policies should include smoking restrictions for balconies, common porch areas, and entrances.

Our overall prevalence of children exposed to tobacco smoke is significantly higher than that reported in the 2006 Surgeon General's report.⁶ This difference is most likely because the NHANES now uses high-sensitivity cotinine testing that allows the detection of low levels of smoke exposure. Identifying those at risk for these low levels of exposure is important because there is increasing evidence that even small or brief exposure to tobacco smoke can cause physiologically significant cardiovascular effects.³⁷ Low-level exposure to tobacco smoke also has been associated with lower scores on cognitive testing.⁸

Although there was a significant association between living in an apartment and cotinine levels for white and black children, this was not the case for those of Hispanic ethnicity or other races. Overall, Hispanic and Asian adults have much lower smoking rates (13.3% and 9.6%, respectively) than black (19.8%) or white (21.4%) adults. This difference particularly is striking for women (8.3% of Hispanic and 4.0% of Asian women smoke compared with 15.8% of black women and 19.8% of white women).³⁷ Because Hispanic and Asian immigrants are more likely to be found in high-density ethnic enclaves where multiunit housing is common,³⁸ it is possible that the lower smoking prevalence among some ethnic groups reduces the overall tobacco-smoke burden in some multiunit housing.

There are other potential sources of exposure that need to be considered. Potential sources may include daycare centers or child-care arrangements³⁹ as well as smoke residue from a parent or caregiver who smokes outside. Other studies have found significantly increased house dust and air nicotine levels in households with a mother who smokes outside, with corresponding increases in children's urine cotinine level.¹⁵ There also is an increase in air and surface nicotine found in used cars previously owned by smokers.⁴⁰ This is an important issue for families who may believe that they are protecting their children by smoking outside. However, because smoking prevalence is much lower than exposure prevalence,³⁷ this does not explain all of the excess exposure.

There are limitations to these data. First, we only were able to examine the association between apartment living and tobacco-smoke exposure; there are other unmeasured potential confounders. Population density and current smoke-free housing legislation are 2 factors that likely play a role; these will need to be examined in future research. In addition, the NHANES data set has no information about home smoking bans or outside smoking behavior, so we cannot know how many of these children have parents who smoke outside or if they are exposed at daycare centers or relatives' homes. We hope that future research will be able to separate out the individual contributions of apartment smoke drift, outside-smoker "off-gassing" and thirdhand smoke, occasional inside smoking by visitors, or exposures outside of the home.

Finally, people who smoke may inaccurately report whether they smoke anywhere inside the home. If underreporting rates varied between those in apartments versus single-family homes, our results may be biased. As-

suming no differential in inaccurate reporting, children in apartments also might be expected to have higher cotinine levels because of the smaller square footage in apartments versus single-family homes. In general, however, people who smoke have demonstrated low rates of underreporting smoking behaviors in nonintervention trials.⁴¹ Finally, a growing number of buildings are smoke-free already,³⁷ leading to an underestimation of the exposure rate in multiunit dwellings where smoking still is allowed.

CONCLUSIONS

Most children in the US continue to be exposed to tobacco smoke, even with the growing knowledge of its damaging effects at low levels of exposure. It is vital to understand the contribution of all potential sources of exposure for children: parents smoking outside, daycare, visiting homes where smoking is allowed, and from connected dwellings. However, significant tobacco-smoke contamination in the air of nonsmoking units of multiunit housing already has been shown. This study is the first to document through human biological sampling that disseminated tobacco smoke from multiunit apartments may contribute to the actual exposure of children. In addition, there are likely to be many adult nonsmokers who also are exposed to tobacco smoke by this mechanism. Biochemical data demonstrating the increased risk of involuntary tobacco-smoke exposure posed by living in apartments may change public opinion and policies about smoke-free multiunit housing for those who live in low-income housing, and for those who live in apartments owned by private companies. These results provide direct evidence for a background level of tobacco-smoke contamination in multiunit housing at levels associ-

ated with childhood morbidity. Ultimately, smoke-free multiunit housing could improve health status by reducing nonsmokers' exposure to tobacco smoke in their own units.

ACKNOWLEDGMENTS

This study was funded by the Julius B. Richmond Center of Excellence of the American Academy of Pediatrics

through a grant from the Flight Attendant Medical Research Institute. Drs Wilson, Klein, Winickoff, and Gottlieb receive support from the Julius B. Richmond Center of Excellence, American Academy of Pediatrics; the Flight Attendant Medical Research Institute; and from the Legacy Foundation. All of the authors contributed substantially to this work and approved of the final

draft as submitted. Mr Blumkin is the programmer/analyst for the study and had full access to all the data provided by the NHANES; and Dr Wilson is the lead author of the study. They both take full responsibility for the accuracy of the data analysis.

We acknowledge the help of Sarah Gates, MS, in the preparation of this manuscript.

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INKED-UP: *When my daughter, who is 15 years old, announced that she intended to get a tattoo, I, like many parents, was emphatic in replying, “Not until you are 18.” As reported in The Wall Street Journal (September 29, 2010: Work and Family), teens and parents often don’t see eye to eye about tattoos. Teens may view tattoos as an accessory while parents view tattoos as a permanent mark likely to be regretted. Tattooing certainly has become commonplace. One of the best selling Barbie Dolls, Tattoo Barbie, comes complete with multiple stickers to attach to her body suggesting interest in tattoos begins early. Almost 40% of youth between the ages of 18 and 29 are tattooed compared to 1/3 of adults born in the 1960s and 1970s, and 15% of baby boomers. In most states, teens need to be 18 years old to be able to obtain a tattoo without parental consent. That, of course, is not an insurmountable barrier. As many as one in six teens gets a tattoo from a friend or an unlicensed parlor. Regulation of tattoo parlors is often minimal. Only nine states require tattoo parlors to comply with infectious disease guidelines such as using sterilized needles and individual pigment cups. Interestingly, while teens like the idea of a permanent mark, the most common reason for regretting getting a tattoo is that the person made the decision at too young an age followed by the permanence of the tattoo. Removing a tattoo is considerably more difficult and expensive than getting one, and rarely completely effective. A tattoo is like a photo on Facebook; easy to post, hard to permanently remove. I am hoping she waits.*

Noted by JFL, MD and WWR, MD

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Pediatrics; originally published online December 13, 2010;

DOI: [10.1542/peds.2010-2046](https://doi.org/10.1542/peds.2010-2046)

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