ORIGINAL ARTICLE

Secondhand Smoke and Sensorineural Hearing Loss in Adolescents

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Objective: To investigate the hypothesis that secondhand smoke (SHS) exposure is associated with sensorineural hearing loss (SNHL) in adolescents.

Design: A complex, multistage, stratified geographic area design for collecting representative data from the noninstitutionalized US population.

Participants: Cross-sectional data from National Health and Nutrition Examination Survey (2005-2006) were available for 1533 participants 12 to19 years of age who underwent audiometric testing, had serum cotinine levels available, and were not actively smoking.

Main Outcome Measures: SNHL was defined as an average pure-tone level greater than 15 dB for 0.5, 1, and 2 kHz (low frequency) and 3, 4, 6, and 8 kHz (high frequency).

Results: Secondhand smoke exposure, as assessed by serum cotinine levels, was associated with elevated pure-tone hearing thresholds at 2, 3, and 4 kHz, a higher rate of unilateral low-frequency SNHL (11.8% vs 7.5%; $P<.04$), and a 1.83-fold increased risk of unilateral low-frequency SNHL in multivariate analyses (95% confidence interval, 1.08-3.41). The prevalence of SNHL was directly related to level of SHS exposure as reflected by serum cotinine levels. In addition, nearly 82% of adolescents with SNHL did not recognize hearing difficulties.

Conclusions: Secondhand smoke is associated with elevated pure-tone thresholds and an increased prevalence of low-frequency SNHL that is directly related to level of exposure, and most affected individuals are unaware of the hearing loss. Thus, adolescents exposed to SHS may need to be closely monitored for early hearing loss with periodic audiologic testing.

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Secondhand smoke (SHS) exposure is a profound public health problem, with more than half of children in the United States exposed.[1] While exposure rates may vary across regions, by socioeconomic status, race/ethnicity, and sex, its detrimental effects have been demonstrated across all demographic groups. Specifically, prenatal tobacco or childhood SHS exposure has been linked to low birth weight,[2] sudden infant death syndrome,[3-5] upper and lower respiratory infections,[6,7] increased asthma severity,[6] behavioral problems,[8,9] cognitive problems,[10] and otitis media (OM).[11] A variety of mechanisms have been proposed to explain the detrimental effects of SHS, including disruption of normal in utero development,[12] alterations of the immune system,[13] postnatal deterioration of body function, and altered hemodynamics.[14]

In the auditory system, SHS is a known risk factor for OM.[11] Recurrent acute OM is more common in the nearly 60% of children exposed to SHS in the United States.[11] While the exact mechanism remains unclear, the increased risk of OM may be through suppression or modulation of the immune system, enhancement of bacterial adherence factors, the consequence of exposure to toxins within SHS, and impairment of the respiratory mucociliary apparatus, leading to Eustachian tube dysfunction.[15]

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Secondhand smoke may also have the potential to have an impact on auditory development, leading to sensorineural hearing loss (SNHL) because of its negative impact on in utero development of the fetus and low birth weight or may lead to decrease in hearing loss due to cochlear, vestibulocochlear nerve VIII damage, or brain damage secondary to childhood and adolescent exposure. To our knowledge, no previous study has examined the possible association between SHS and SNHL among children or adolescents. The pres-

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ent study examines risk factors for SNHL in different age, sex, race/ethnicity, and income or poverty groups among adolescents ages 12 to 19 years and investigates the independent association between SHS and SNHL in this nationally representative sample.

METHODS

PARTICIPANTS

Data from 2288 adolescents, 12 to 19 years of age, from the National Health and Nutrition Examination Survey (NHANES 2005 to 2006) were examined.[16] The survey was conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention and was reviewed and approved by the NCHS institutional review board. NHANES 2005-2006 is a cross-sectional health survey that used a complex, multistage design to achieve a nationally representative sample of the noninstitutionalized civilian population in the United States.[16]

Participants were evaluated during a home interview to determine family medical history, current medical conditions, medication use, self-report of the presence of any smokers in the household, and socioeconomic and demographic information. In addition, each person was randomly assigned to undergo a morning, afternoon, or evening examination at a mobile examination center consisting of physical examinations and laboratory testing using blood and urine samples.

AUDIOMETRIC MEASURES

The NHANES 2005-2006[16] protocol for audiometry included otoscopic examination. Audiometry was limited to those ages 12 to 19 years; no comparable data were available for younger children. The ears were examined with a Welch-Allyn otoscope (model 25020; Skaneateles Falls, New York) with rechargeable handle and disposable specula. The Micro Audiometrics Earscan Acoustic Impedance Tympanometer (Murphy, North Carolina) was used to perform tympanometry to evaluate the functional health of the middle ear system. Tympanometry was performed from –312 daPa to 200 daPa. The quality of the tympanogram was noted along with the peak response. Audiometry was conducted in a special sound booth (model Delta 143; Acoustic Systems, Cedar Park, Texas) built into the mobile examination center by trained examiners using a standardized protocol. An audiometer (model AD226; Interacoustic, Assens, Denmark) with standard TDH-39P headphones (Telephonics Corp, Farmingdale, New York) and insert earphones (model EARTone 3A; Etymotic Research, Elk Grove Village, Illinois) was used for testing hearing. The audiometer was calibrated with the same specifications at the start and end of testing at each field location using an acoustic simulator (model BA-201-25; Quest, Oconomowoc, Wisconsin). Additional information regarding the methods, calibration equipment, and calibration protocol is available at http://www.cdc.gov/nchs/data/nhanes/nhanes_05_06/AU.pdf. Air-conduction thresholds were measured for each ear at 0.5, 1, 2, 3, 4, 6, and 8 kHz, with testing repeated at 1 kHz across an intensity range of –10 to 120 dB. The correlation of the threshold for the 1-kHz first test with the retest was 0.9 ($P < .001$) for the left and right ears of each child. The 1-kHz first test was the value used for this analysis.[17]

The response to the following question was also used to determine the self-recognition of hearing impairment: "Which statement best describes {your/Sample Person's} hearing (without a hearing aid)? Would you say {your/his/her} hearing is excellent, good, that {you have/she or he has} a little trouble, moderate trouble, a lot of trouble, or {are you/is she or he} deaf?"

DEFINITION: HEARING LOSS

Hearing loss can be categorized by where or what part of the auditory system is damaged. There are 3 basic types of hearing loss: SNHL, conductive hearing loss (CHL), and mixed hearing loss (MHL). SNHL was inferred when findings from the otoscopic examination were normal and there were adequate or good-quality results from a tympanogram with a peak of more than 0.3 mL. Thus, individuals with abnormal otoscopic results, poor-quality tympanogram results, or a peak response of less than 0.3 mL were excluded from further analyses because these individuals may have had CHL or MHL. Among the 2288 available adolescents ages 12 to 19 years, only 32 were excluded based on these criteria.

Low- and high-frequency hearing threshold was defined as the average of pure-tone hearing levels at 0.5, 1, and 2 kHz, and 3, 4, 6, and 8 kHz, respectively. Low- and high-frequency hearing loss was defined as low- and high-frequency hearing threshold above 15 dB.[18-22] Unilateral low- or high-frequency hearing loss was defined as a pure-tone average greater than 15 dB HL in the worse ear. The hearing loss was deemed to be bilateral when the pure-tone average for the better ear was greater than 15 dB HL.

DEFINITION: ACTIVE SMOKING AND SHS EXPOSURE

Cotinine, a metabolite of nicotine, was used as a biomarker for both active smoking and exposure to SHS. In 424 individuals, cotinine levels were not available and, thus, they were excluded from further analyses. There were no significant differences between these 424 individuals and those included in the study with respect to either social demographic characteristics, or rates of hearing loss ($P > .05$ for all comparisons). In addition to cotinine, NHANES includes adolescent self-report of smoking status. Consistent with previously published studies, active smokers were defined as those with cotinine levels 15.0 µg/L or higher or those who reported smoking in the past 5 days. Those with serum cotinine levels that were detectable but less than15.0 µg/L and who did not report smoking in the past 5 days were defined as exposed to SHS.[23] A cotinine level of less than 0.05 µg/L was below the detection limit. Those with undetectable serum cotinine levels and without self-reported smoking were defined as unexposed. In the analyses assessing the association of exposure level and hearing loss, the observed cotinine levels were grouped into quartiles: quartile 1, 0.0876 µg/L or less; quartile 2, more than 0.0876 µg/L and up to 0.217 µg/L; quartile 3, more than 0.217 µg/L and up to 0.858 µg/L; quartile 4, more than 0.858 µg/L but less than 15.0 µg/L). Duration of exposure to SHS was not available in the database. To study the association of SHS and SNHL, 299 active smokers (either self-report of smoking in past 5 days or cotinine levels of 15.0 µg/L or higher) were excluded from the study. (To convert cotinine to nanomoles per liter, multiply by 5.675.)

SOCIODEMOGRAPHIC VARIABLES AND HEARING-RELATED COVARIATES

The adolescent participants were divided into 2 age groups: 12 to 15 years and 16 to 19 years. Race/ethnicity was classified as non-Hispanic black, non-Hispanic white, or Mexican American. The number of patients in the "all other" race/ethnicity category (eg, other Hispanics, Asians, and Native Americans), 40, was too small to be analyzed separately but was included

Table 1. Prevalence of Hearing Loss (HL) With and Without Secondhand Smoke (SHS) Exposure Among Adolescents (NHANES 2005-2006[a])

Hearing Loss	Participants, No.	≤15 dB, %	>15 dB, %				P Value[b]
			Overall Rate of HL, >15 dB	>15 dB and ≤25 dB	>25 dB and ≤40 dB	>40 dB	
Unilateral low-frequency							.03
Total	**1533**	90.45	9.55	7.33	1.17	1.06	
Nonexposed	754	92.47	7.53	6.15	0.86	0.52	
SHS-exposed	799	88.18	11.82	8.65	1.51	1.65	
Bilateral low-frequency							.18
Total	**1533**	97.81	2.19	2.07	0.04	0.07	
Nonexposed	754	98.35	1.65	1.65			
SHS-exposed	799	97.20	2.80	2.54	0.09	0.17	
Unilateral high-frequency							.29
Total	**1533**	84.62	15.38	11.18	2.53	1.67	
Nonexposed	754	86.14	13.86	10.10	2.69	1.07	
SHS-exposed	799	82.91	17.09	12.39	2.36	2.33	
Bilateral high-frequency							.04
Total	**1533**	96.32	3.68	3.18	0.33	0.17	
Nonexposed	754	97.46	2.54	2.31	0.16	0.07	
SHS-exposed	799	95.03	4.97	4.17	0.51	0.29	

[a] See Analytic and Reporting Guidelines: The National Health and Nutrition Examination Survey (NHANES).[16]
[b] *P* value is for the Cochran-Armitage trend test.

in all totals. These 4 race/ethnicity groups are mutually exclusive. The poverty-income ratio (PIR) was defined as the total family income divided by the poverty threshold, as determined by the US Bureau of the Census, for the year of the interview. Income was classified as poverty (PIR≤1) and not poverty (PIR>1).

Prematurity, diabetes mellitus, more than 3 episodes of OM, allergy, and eczema are each recognized risk factor for hearing loss.[24-27] In NHANES 2005-2006,[16] the Early Childhood section of the Sample Person Questionnaire provides personal interview data for children (age <15 years), including the age of the biological mother when the child survey participant was born, smoking habits of the mother while she was pregnant with the participant, birth weight, and whether the participant received care in an neonatal intensive care unit (NICU), premature nursery, or any other type of special care facility. Moreover, the NHANES 2005-2006 survey included questions about OM, asthma, allergy, and eczema. However, noise exposure history was not available for participants.

STATISTICAL ANALYSIS

After excluding potential participants because of inadequate smoking history, active smoking status, and findings suggestive of CHL/MHL, the sample available for the study comprised 1533 adolescents ages 12 to 19 years. χ^2 Tests were used for bivariate analyses to test for associations between independent characteristics investigated and unilateral or bilateral low- or high-frequency hearing loss (unilateral low-frequency hearing loss, bilateral low-frequency hearing loss, unilateral high-frequency hearing loss, and bilateral high-frequency hearing loss). The Cochran-Armitage trend test was used to test for trends. Two additional analyses were performed: one on SHS exposed quartile levels and the other on SHS exposure and its association with different speech-frequency levels (using *t* tests for 0.5, 1, 2, 3, 4, 6, and 8 KHZ). Logistic regression analysis was used to test for independent associations between hearing loss and variables of interest. In addition to sex, age, race/ethnicity, and socioeconomic demographic characteristics, all variables significant at $P<.20$ in bivariate analysis were included in the multivariate logistic regression model. All analyses were conducted with SAS statistical software (version 9.2; SAS Institute Inc, Cary, North Carolina). SUDAAN statistical software[28] was used to account for the complex sample design of the NHANES and to apply sampling weights to produce national estimates by adjusting for the oversampling of young children, older adults, Mexican Americans, and blacks.

RESULTS

Table 1 shows the prevalence of given levels of auditory thresholds (≤15 dB, >15 and ≤25 dB, >25 and ≤40 dB, and >40 dB) for individuals with and without SHS exposure. For example, among adolescents aged 12 to 19 years, using 15 dB as the threshold for hearing loss, the overall rates of unilateral low-frequency hearing loss, bilateral low-frequency hearing loss, unilateral high-frequency loss, and bilateral high-frequency loss were 9.55%, 2.19%, 15.38%, and 3.68% respectively. The prevalence of hearing loss was greater among individuals exposed to SHS for both unilateral low- and high-frequency hearing and bilateral low- and high-frequency hearing compared with those who were nonexposed. Among these, SHS was associated with a significantly increased rate of elevated auditory thresholds for unilateral low-frequency hearing loss and bilateral high-frequency hearing loss ($P=.03$ and .04; Cochran-Armitage trend test *P* value, respectively).

Most adolescents with unilateral low- or high-frequency hearing loss were unaware of their hearing impairment. Only 18.43% with low-frequency and 11.43% with high-frequency hearing loss reported "a little trouble, moderate trouble, or a lot of trouble" with hearing (**Table 2**).

The association of SHS and hearing levels at 0.5, 1, 2, 4, 5, 6, and 8 kHz was evaluated using both the average threshold for the right and left ears, as well as the hear-

Table 2. Self-reporting[a] of Hearing Difficulty Among Adolescents With and Without Sensorineural Hearing Loss (NHANES 2005-2006)[b]

	Self-Report of Hearing Loss					
	Low Frequency[c]			High Frequency[d]		
Pure-Tone Frequency Hearing	Participants, No.	Excellent or Good, %	A Little, Moderate, or Lot of Trouble, %	Participants, No.	Excellent or Good, %	A Little, Moderate, or Lot of Trouble, %
Normal	1833	95.41	4.59	1701	95.23	4.77
Hearing loss	185	81.57	18.43	309	88.57	11.43

[a] Which statement best describes {your/Sample Person's} hearing (without a hearing aid)? Would you say {your/his/her} hearing is excellent, good, that {you have/she or he has} a little trouble, moderate trouble, a lot of trouble, or {are you/is she or he} deaf?
[b] See Analytic and Reporting Guidelines: The National Health and Nutrition Examination Survey (NHANES).[16]
[c] Low-frequency hearing loss: average of hearing thresholds at 0.5, 1, and 2 kHz greater than 15 dB HL.
[d] High-frequency hearing loss: average of hearing thresholds at 3, 4, 6, and 8 kHz greater than 15 dB HL.

Table 3. Pure-Tone Hearing Level (dB) at Each Frequency, With and Without Secondhand Smoke (SHS) Exposure, Among Adolescents (NHANES 2005-2006)[a]

	Average, Right and Left Ears, Mean (SD)			Worse Ear, Among Right and Left Ears, Mean (SD)		
Frequency, kHz	SHS-Exposed	Unexposed	*P* Value[b]	SHS-Exposed	Unexposed,	*P* Value[b]
0.5	9.87 (0.51)	8.86 (0.51)	.13	12.72 (0.65)	11.55 (0.57)	.15
1	5.23 (0.58)	4.43 (0.36)	.23	8.37 (0.78)	7.16 (0.37)	.14
2	5.99 (0.35)	3.50 (0.41)	<.001	9.11 (0.53)	6.16 (0.41)	<.001
3	5.56 (0.48)	4.11 (0.35)	.03	8.65 (0.65)	6.87 (0.44)	.04
4	6.20 (0.54)	4.28 (0.50)	.03	9.71 (0.69)	7.41 (0.51)	.01
6	12.10 (0.73)	11.59 (0.55)	.53	16.48 (0.89)	15.91 (0.64)	.55
8	8.46 (0.82)	7.64 (0.60)	.28	12.37 (0.92)	11.75 (0.74)	.54

[a] See Analytic and Reporting Guidelines: The National Health and Nutrition Examination Survey (NHANES).[16]
[b] For independent *t* test.

ing level of the worse ear (**Table 3**). Across all frequencies, the mean pure-tone hearing level was noted to be higher in adolescents exposed to SHS when compared with individuals without SHS exposure; the thresholds were statistically significantly elevated at 2, 3, and 4 kHz both when investigating average thresholds or thresholds for the ear with worse hearing.

Results of bivariate analyses conducted to investigate the relationship of hearing loss with a wide variety of variables are shown in **Table 4**. Compared with those adolescents not exposed, SHS exposure was associated with a significantly higher prevalence of unilateral low-frequency hearing loss (11.82% vs 7.53%; $P=.04$); although rates of unilateral high-frequency hearing loss were greater in the group exposed to SHS (17.09% vs 13.86%), the difference was not statistically significant ($P=.34$). Greater SHS exposure, as reflected by higher cotinine levels, was associated with higher rates of hearing loss (see the discussion of dosage analysis at the end of this section). Eczema and black race were significantly associated with bilateral low-frequency hearing loss and bilateral high-frequency hearing loss, respectively. Adolescents who had received care in the NICU were found to have a significantly higher prevalence of low-frequency hearing loss. Despite a higher prevalence, low birth weight was not statistically significantly associated with hearing loss, likely reflecting the small number of participants (76) who had been of low birth weight in the study sample. Similarly, although the data suggest an association between both prenatal smoking and ear infections and low-frequency unilateral hearing loss, these associations were not significant at the $P<.05$ level.

Multivariable logistic regression analyses were used to investigate if SHS was independently associated with low-frequency SNHL when controlling for age group, sex, race/ethnicity, and poverty; because history of receiving NICU care was available only for adolescents younger than 15 years, it was not included in this analysis (**Table 5**). Only SHS exposure was significantly associated with unilateral hearing loss. Adolescents with exposure to SHS were 1.83 times more likely to have low-frequency hearing loss (95% confidence interval [CI], 1.08-3.41) compared with nonexposed individuals.

To assess the possibility of a dose-response relationship between exposure levels and unilateral low-frequency hearing loss, the exposed group was categorized into 4 levels of exposure based on the serum cotinine quartiles. Higher serum cotinine levels were associated with a greater prevalence of unilateral low-frequency hearing loss: the prevalence of hearing loss was 7.53% for nonexposed participants, and 7.71%, 10.54%, 12.08%, and 17.05% for those with exposure levels of 1, 2, 3, and 4, respectively ($P=.02$) (Table 4). In multivariable logistic regression analyses, controlling for other covariates, a significant association was found for the group with level 4 exposure (odds ratio, 2.72; 95% CI, 1.46-5.06) (Table 5).

Table 4. Bivariate Analysis for Risk Factors for Sensorineural Hearing Loss (SNHL) Among Adolescents (NHANES 2005-2006)[a]

		Low-Frequency SNHL (>15 dB)[b]				High-Frequency SNHL (>15 dB)[c]			
Risk Factor	N	Unilateral SNHL, %	*P* Value[d]	Bilateral SNHL, %	*P* Value[d]	Unilateral SNHL, %	*P* Value	Bilateral SNHL, %	*P* Value[d]
	1533*	9.55		2.19		15.38		3.68	
SHS[e]			.04		.36		.3428		.10
Nonexposed	754	7.53		1.65		13.86		2.54	
Exposed	779	11.82		2.80		17.09		4.97	
SHS exposure level, quarter[f]			.02		.16		.37		.16
Nonexposed	754	7.53		1.65		13.86		2.54	
1	190	7.71		0.42		15.64		3.79	
2	188	10.54		1.56		13.95		7.60	
3	201	12.08		4.14		19.85		1.49	
4	200	17.05		5.14		18.80		6.90	
Prenatal smoking[g]			.11		.24		.34		.48
Yes	129	16.78		4.10		18.48		5.73	
No	703	6.40		1.63		13.47		3.13	
BW[g]			.53		.41		.58		.72
Low, ≤2.5 kg	76	16.48		5.87		17.89		3.94	
Normal, >2.5 kg	709	7.77		1.88		13.98		3.06	
Very low BW[g]			.52				.45		.46
Very low, ≤1.6 kg	12	42.32		42.32	.41	60.36		15.41	
Low, ≤2.5 kg	64	12.95		0.89		12.08		2.37	
Normal, >2.5 kg	709	7.77		1.88		13.98		3.06	
Received NICU care[g]			.01		.22		.40		.82
Yes	111	16.30		2.41		18.07		3.04	
No	722	7.35		2.16		13.74		3.50	
Sex			.69	2.73	.42		.14		.21
Male	737	8.56		1.64		14.66		3.66	
Female	796	10.56				15.49		3.70	
Age group, y			.60		.99		.33		.99
12-15	847	8.47		2.18		14.35		3.52	
16-19	686	11.08		2.20		16.83		3.63	
Race/ethnicity			.33		NA		.52		.02
Mexican	539	7.75		1.56		13.92		2.87	
Other Hispanic	40	3.37		0.00		12.78		4.59	
White	359	10.36		2.14		15.99		3.46	
Black	523	10.10		3.80		17.24		6.06	
Poverty income ratio			.61		.65		.81		.94
Poor (<1)	432	9.74		1.92		14.77		3.52	
Not poor (≥1)	1043	8.54		2.28		15.67		3.63	
OM[h]			.23		.48		.64		.49
Yes	408	12.95		2.63		16.54		4.25	
No	1114	7.29		1.90		14.68		3.22	
Allergy			.78		.63		.76		.21
Yes	395	9.88		2.74		14.66		2.73	
No	1136	9.21		1.98		15.49		4.08	
Eczema			.85		<.01		.98		.86
Yes	132	10.28		2.40		15.49		3.37	
No	1397	9.49		0.29		15.39		3.72	

Abbreviations: BW, birth weight; HL, hearing loss; NA, not applicable; NICU, neonatal intensive care unit; OM, otitis media; SHS, secondhand smoke.
[a] See Analytic and Reporting Guidelines: The National Health and Nutrition Examination Survey (NHANES).[16]
[b] Low-frequency SNHL: average of hearing thresholds at 0.5, 1, and 2 kHz >15 dB HL.
[c] High-frequency SNHL: average of hearing thresholds at 3, 4, 6, and 8 kHz >15 dB HL.
[d] χ^2 Test.
[e] Defined as serum cotinine levels lower than 15.0 μg/L, and the individual did not report smoking in the previous 5 days.
[f] The SHS exposure levels are defined as follows: 1, to 0.0876 μg/L; 2, more than 0.0876 μg/L up to 0.217 μg/L; 3, more than 0.217 μg/L up to 0.858 μg/L; 4, more than 0.858 μg/L but less than 15.0 μg/L. To convert cotinine to nanomoles per liter, multiply by 5.675.
[g] Data regarding prenatal smoking, low birth weight, very low birth weight, history of NICU care are available only for adolescents younger than 15 years.
[h] More than 3 episodes of OM.

COMMENT

Exposure to SHS has been linked to diverse disease in humans, affecting the unborn to the elderly. In this study, based on a large, nationally representative sample with an objective biochemical marker of tobacco exposure, SHS is found to be associated with hearing loss in US adolescents. Furthermore, this risk to auditory function is directly related to serum cotinine level, a biomarker of tobacco exposure. Specifically, SHS is associated with

Table 5. Risk Factors Independently Associated With Unilateral Low-Frequency Sensorineural Hearing Loss[a] for Adolescents Aged 12 to 19 Years (NHANES 2005-2006)[b]

Risk Factor	OR (95% CI)
SHS[c]	
Nonexposed	
Exposed	1.83 (1.08-3.41)
SHS exposure level, quarter[d]	
Nonexposed	
1	1.10 (0.59-2.04)
2	1.69 (0.77-3.72)
3	1.89 (0.76-4.72)
4	2.72 (1.46-5.06)
Sex	
Male	1.35 (0.77-2.37)
Female	
Age group, y	
12-15	
16-19	1.46 (0.95-2.26)
Race/ethnicity	
Mexican American	0.9 (0.45-1.79)
Other Hispanic	0.59 (0.18-1.94)
Non-Hispanic white	
Non-Hispanic black	0.79 (0.48-1.31)
Poverty income ratio	
Poor (<1)	0.82 (0.48-1.38)
Not poor (≥1)	

Abbreviations: CI, confidence interval; OR, odds ratio; SHS, secondhand smoke.

[a] Unilateral hearing loss: worse ear hearing thresholds at [(0.5 kHz + 1 kHz + 2 kHz)/3 > 15 dB].[b] See Analytic and Reporting Guidelines: The National Health and Nutrition Examination Survey (NHANES).[16]

[c] Defined as serum cotinine levels lower than15 μg/L, and the individual did not report smoking in the previous 5 days.

[d] The SHS exposure levels are as follows: 1, ≤0.0876 μg/L; 2, more than 0.0876 μg/L up to 0.217 μg/L; 3, more than 0.217 μg/L up to 0.858 μg/L; 4, more than 0.858 μg/L but less than 15.0 μg/L. The SHS level was tested in a separate multivariate model adjusted by age, sex, race/ethnicity, and poverty income ratio. To convert cotinine to nanomoles per liter, multiply by 5.675.

elevated pure-tone thresholds at 0.5, 1, 2, 3, 4, 6, and 8 kHz (although not statistically significant at every frequency), suggesting that the injury to the inner ear is global. The elevated threshold for pure tones is significantly higher at 2, 3, and 4 kHz in individuals with SHS exposure when compared with those without exposure. These mid to high frequencies are critical for hearing in humans and are responsible for the clarity of hearing that allows us to discriminate between similar sounding words. Smoking has been previously associated with accelerated hearing loss in adults.[29] The finding of elevated auditory thresholds in adolescents in this study suggests that the injury to the inner ear responsible for smoking-related hearing loss in adults may begin at a very early age and may also include those who do not actively smoke but who are passively exposed.

In this study, the rates of unilateral and bilateral low-frequency, and unilateral and bilateral high-frequency SNHL were found to be greater in adolescents exposed to SHS, and this hearing loss was significantly higher for unilateral low-frequency hearing loss. The unilateral hearing loss identified in adolescents exposed to SHS likely reflects an early stage of injury; with continued exposure, we hypothesize that it would progress to involve both ears and all frequencies. Also, there was a relationship between the prevalence of hearing loss and exposure level: higher serum cotinine levels were associated with higher rates of hearing loss. In multivariate analyses, controlling for sex, age, race/ethnicity, and poverty, exposure to tobacco smoke was associated with a 1.83-fold increased risk of unilateral low-frequency hearing loss among adolescents. The risk of hearing loss increase is the greatest for the highest exposure level: the increased risk is 2.72-fold higher for cotinine quartile level 4 (>0.858 μg/L and <15.0 μg/L). Findings of the bivariate and multivariate analyses are consistent with the hypothesis that the deterioration in auditory function observed is independently associated with SHS exposure.

In children, untreated hearing loss above 35 dB in the better ear has a considerable negative impact on speech, language, and cognitive development; these children also go on to have difficulties with academic and vocational achievement.[30-32] While still somewhat controversial, mild hearing loss (>15 dB and <35 dB) also has been shown to be similarly detrimental to children's development. As a result of their hearing loss, they may miss up to 10% of speech and respond inappropriately. Mild hearing loss in the sensitive speech and language acquisition period has been associated with delays similar to children with more severe hearing loss.[33] Children with mild hearing loss have been shown to have academic problems (Bess et al[34]). Similarly, their social interactions may be affected. Children with hearing loss are often labeled as troublemakers owing to their inappropriate responses based on misunderstood verbal instructions.[35] The effects of mild SNHL during adolescence remain to be elucidated.

A variety of mechanisms may be implicated in how SHS affects auditory function, including its effect on the microvasculature,[36] endocrine function,[37] and oxidative stress.[38] Tobacco smoke is known to have a detrimental effect on the microvasculature. As an active energy producing and consuming organ, the inner ear is dependent on adequate blood supply and thus may be susceptible to tobacco-mediated alterations in blood flow. Smoking's effect on the cardiovascular system[39] and diabetes mellitus[40] may both also affect hearing; both are independently associated with hearing loss in adults.[41] Hypoxemia due to SHS may be deleterious to the inner ear. Direct injury to the inner ear by nicotine or other chemicals in SHS could also be implicated.

Because more than 50% of adolescents are exposed to SHS,[1] the association of the mild SNHL with SHS has significant implications for public health in the United States. While there is active screening for hearing loss in newborns and young children, adolescents are not routinely evaluated for hearing loss and are screened for hearing loss only when risk factors are present.[42] Should the findings of this study be corroborated, adolescents exposed to SHS may be at risk for hearing loss, and prudent policy may dictate that they undergo hearing screening. Self-reporting of hearing loss alone is inadequate for identifying affected individuals because more than 80% were unaware of hearing difficulty in this national sample. Thus, adolescents who are exposed to SHS may need to be more closely monitored for hearing loss; in addition, they should be educated about risk factors for hearing loss, such as recre-

ational or occupational noise exposure and SHS. In homes where there is active smoking, parents and caretakers should be made aware of risks to hearing in their children. Health care providers (ie, physicians and nurse practitioners) should add SHS exposure to the list of risk factors for hearing loss and refer these young adults for complete audiologic evaluation to identify early hearing loss.

There are several limitations to these data and their analysis. While large and comprehensive, the NHANES data set is cross-sectional, and causal inferences cannot be made. Furthermore, prenatal SHS exposure history was available for some but not all of the participants and was based on retrospective report, thus limiting the validity of this measure. The duration of SHS exposure was also not available; thus, it was difficult to determine if earlier and/or long-term exposure is worse than later and/or short-term exposure. Similarly, these data do not enable one to disentangle prenatal from childhood or concurrent adolescent exposure. The data also do not provide the source of the SHS exposure, thus limiting the development of effective public health interventions to prevent SHS-mediated hearing loss. Another significant limitation of the NHANES data set is the absence of noise exposure history. Exposure to recreational noise is rising among young adults and may be an independent risk factor for hearing loss. The possibility that noise is a confounder cannot be excluded using the NHANES database. Finally, in this study, SNHL was inferred when findings from the otoscopic examination were normal and there were adequate or good-quality tympanogram results with a peak greater than 0.3 mL. In previous publications related to noise-induced hearing loss (in which the hearing loss was presumed to be SNHL) using various iterations of the NHANES database, the investigators used less rigorous criteria for defining the presumed noise-induced hearing loss: excluding those with compliance of 0.2 mL or less and without consideration of otoscopic findings.[22,43] Nonetheless, even with the more restrictive definition and the use of otoscopic findings in the current study, it is still possible that an indeterminate number of these individuals may have had CHL instead of SNHL. While this would not change the statistical outcome, the association of SHS would be with the more generic CHL and SNHL combined instead of SNHL alone.

In conclusion, this study demonstrates, to our knowledge for the first time, a relationship between tobacco smoke exposure and hearing loss among adolescents in the United States. These data come from a large, nationally representative sample with objective biochemical rather than exclusive self-reported, measures of smoking status. The findings indicate that exposure to tobacco smoke is independently associated with an almost 2-fold increase in the risk of hearing loss among adolescents. These findings may have profound implications in light of the high exposure rates among adolescents in the United States. Future studies need to investigate the adverse consequences of this early hearing loss on social development, academic performance, behavioral and cognitive function, and public health costs.

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