

Original Investigation

Secondhand smoke exposure among nonsmokers nationally and in New York City

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Abstract

Introduction: We describe smoking prevalence and secondhand smoke (SHS) exposure among adult nonsmokers in New York City (NYC) across key demographic strata and compare exposure estimates with those found nationally.

Methods: We used serum cotinine data from the 2004 NYC Health and Nutrition Examination Survey ($n=1,767$ adults aged 20 years or older) and the 2003–2004 National Health and Nutrition Examination Survey ($n=4,476$ adults aged 20 years or older) to assess and compare smoking prevalence and the prevalence of elevated cotinine levels (≥ 0.05 ng/ml) among nonsmokers. We conducted multivariate logistic regression to assess independent predictors of elevated cotinine levels in NYC.

Results: Although the smoking prevalence in NYC was lower than that found nationally (23.3% vs. 29.7%, $p < .05$), the proportion of nonsmoking adults in NYC with elevated cotinine levels was greater than the national average overall (56.7% vs. 44.9%, $p < .05$) and was higher for most demographic subgroups. In NYC, the highest cotinine levels among nonsmokers were among adults aged 20–39 years, males, and Asians.

Discussion: Although NYC enacted comprehensive smoke-free workplace legislation in 2003, findings suggest that exposure to SHS remains a significant public health issue, especially among certain subgroups. The finding of a higher prevalence of SHS exposure in NYC despite lower smoking rates is puzzling but suggests that SHS exposure in dense, urban settings may pose a particular challenge.

Introduction

Exposure to secondhand smoke (SHS), also called environmental tobacco smoke, has been associated with numerous health effects, including coronary heart disease, lung cancer, and ischemic heart disease among adult nonsmokers (Law, Morris, & Wald, 1997; U.S. Department of Health and Human Services [USDHHS], 2006; U.S. Environmental Protection Agency, 1992). The U.S. surgeon general has concluded that there is no risk-free level of exposure to SHS (USDHHS, 2006). Recent national data have suggested that SHS exposure among nonsmokers has decreased; however, disparities in progress exist, with less success documented among males and non-Hispanic Blacks (Pirkle, Bernert, Caudill, Sosnoff, & Pechacek, 2006).

States and municipalities throughout the United States are implementing smoke-free air laws to reduce SHS exposure. By the end of 2004, seven U.S. states had comprehensive smoke-free laws in place. Currently, such laws are in effect in 13 states. To measure progress in tobacco control activities, states and cities often rely on population-based telephone surveys that measure self-reported SHS exposure. However, such measures may underestimate exposure (Cummings et al., 1990) and may be particularly unreliable in estimating magnitude or duration of exposure (Brownson, Alavanja, & Hock, 1993; Coultas, Peake, & Samet, 1989; Pron, Burch, Howe, & Miller, 1988). Serum cotinine can provide a more accurate assessment of SHS exposure (Benowitz, 1996; Pérez-Stable, Benowitz, & Marin, 1995), which can be used to guide public health policy.

To reduce morbidity and mortality from tobacco, New York City (NYC) implemented a comprehensive tobacco control

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program that (a) made virtually all workplaces, including restaurants and bars, smoke free through the Smoke Free Air Act of 2002; (b) passed legislation that increased the price of cigarettes; (c) established an initiative to provide free nicotine replacement therapy directly to smokers; (d) and developed and launched an intensive local antitobacco advertising campaign. Since the inception of this comprehensive tobacco control program, self-reported smoking prevalence in NYC decreased by 15% between 2002 and 2004 (from 21.6% to 18.4%; New York City Department of Health and Mental Hygiene [NYCDHMH], 2008). Similarly, self-reported SHS exposure prevalence among nonsmoking adults declined by 23% between 2002 and 2004 (from 28.1% to 21.5%; NYCDHMH, 2008). Although these declining rates of self-reported smoking and SHS exposure demonstrate the effectiveness of the tobacco control program, biological measurements collected through cross-sectional surveillance tools can assess both known and unknown exposure to SHS and can provide data to compare with national measures.

We used the 2004 New York City Health and Nutrition Examination Survey (NYC HANES), a population-based sample of NYC residents, and the adult data from the 2003–2004 National Health and Nutrition Examination Survey (NHANES) to describe smoking and SHS exposure, as measured by serum cotinine. We estimated the prevalence of smoking and of SHS exposure among nonsmoking adults in NYC. We also identified demographic patterns in smoking and SHS estimates. Exposure levels, both overall and by demographic subgroup, were compared with national levels from the 2003–2004 NHANES.

Methods

NYC HANES survey design and study population

NYC HANES was a population-based, cross-sectional survey of noninstitutionalized adult NYC residents (aged 20 years or older) that was modeled after NHANES. Detailed information on the data collection components and protocols, as well as a detailed description of the study design, has been published elsewhere (Thorpe et al., 2006). We used a three-stage cluster sampling plan to recruit participants between June and December 2004. In the first stage of the sample design, we randomly selected 144 segments as primary sampling units from a sampling frame of 21,169 segments across the city. The segments were based on counts of households from the 2000 U.S. Census and consisted of a block or a group of proximal blocks within a given census tract; each segment had a required minimal total number of households. We selected the sample of segments with probability proportional to a measure of size. In the second stage of the sample design, we generated a sampling frame of households by sending field staff teams to enumerate all dwelling units located in the 144 segments. We randomly selected a sample of 4,026 households from the 144 segments. In the third stage of the sample design, we selected adults within households for inclusion in the study. We randomly selected eligible adults aged 20 years or older based on an a priori computer-generated sampling flag. The sampling procedure was designed to select zero, one, or two adults from each selected household, depending on the total number of adults residing in that unit.

The survey consisted of a physical examination, clinical and laboratory tests, a face-to-face fully automated computer-assisted personal interview, and a private audio computer-assisted self-interview. Eligibility screening questionnaires were completed in 3,388 (84%) of the 4,026 households selected for NYC HANES. A total of 3,047 eligible survey participants were identified; 1,999 individuals (66%) completed the face-to-face interview and at least one comprehensive examination measurement, yielding an overall survey response rate of 55%. The serum cotinine level was determined for 1,767 (88.4%) of the 1,999 participants.

NHANES survey design and study population

NHANES is a routinely conducted population-based, cross-sectional survey of noninstitutionalized U.S. residents aged 2 months or older. Information on data collection protocols, equipment, and study design is published elsewhere (National Center for Health Statistics, 2006b). We analyzed data for the NHANES cycle conducted during 2003–2004 (National Center for Health Statistics, 2006a). The NHANES 2003–2004 overall response rate for adults aged 20 years or older was 69% (4,742/6,916), based on those who were originally selected for participation (Ogden et al., 2006). All analyses were limited to adults aged 20 years or older.

Laboratory methods

Serum cotinine levels were determined for all study participants with an available blood specimen. NYC HANES serum cotinine samples were analyzed at the New York State Wadsworth Laboratories, using the technique consistent with the standardized protocol of NHANES, an isotope dilution, liquid chromatography/tandem mass spectrometry (LC/MS/MS) method. The current NHANES method for serum cotinine, described by Bernert, McGuffey, Morrison, and Pirkle (2000) and Bernert et al. (1997), was transferred to and validated at the New York State Wadsworth Laboratories after training in the laboratory of one of the coauthors (Aldous) at the National Center for Environmental Health at the Centers for Disease Control and Prevention. In summary, the NYC HANES serum samples were equilibrated with a trideuterated cotinine internal standard and then extracted using precleaned ChemElute solid-phase extraction cartridges (Varian, Palo Alto, CA). The extract was evaporated to dryness under vacuum, reconstituted in 100 μ l of isopropanol, and analyzed by LC/MS/MS using electrospray ionization. The instrumental system comprised an Agilent 1100 series LC and Applied Biosystems API 4000 triple quadrupole mass spectrometer. The limit of detection (LOD) for this method was 0.050 ng/ml cotinine in serum. Typical batches included 40 serum samples; at least two blanks; and quality control (QC) samples for high, medium, and low levels (15, 1.5, and 0.15 ng/ml, respectively). All final results were blank corrected using the mean batch blank value. The average blank for the NYC HANES serum cotinine project conducted from July 2004 to February 2005 was 0.018 ng/ml ($n=440$). Batch blanks typically had levels of less than 0.03 ng/ml throughout the analysis. QC charts for the three QC levels were evaluated to ensure that data were reported only when the analysis was within control limits and that signals did not exceed the calibration range; otherwise, the analysis was repeated.

Although the LOD for this method is 0.050 ng/ml, the NHANES serum samples were analyzed using a newer, more

sensitive mass spectrometer and thus had an LOD of 0.015 ng/ml (Pirkle et al., 2006).

Data analyses

We defined smokers as those having a serum cotinine level higher than 10 ng/ml; nonsmokers had a serum cotinine level of 10 ng/ml or lower (NYC HANES $n=1,330$; NHANES $n=3,285$), consistent with previous analyses (Pirkle et al., 1996). To assess a comparable measure of cotinine prevalence among nonsmokers across both surveys, we compared proportions of the population with a cotinine level of 0.05 ng/ml or higher; hereafter we refer to this level as “elevated.” We present elevated cotinine prevalence estimates for both NYC HANES and NHANES, overall and by age, sex, race/ethnicity, country of birth, education, and income. For country of birth, we included in the “U.S. born” category all those born in the 50 states and Washington, DC. We age-adjusted prevalence estimates to the year 2000 U.S. standard population. We also calculated geometric mean values of serum cotinine for all demographic indicators assessed in the NYC HANES; due to the difference in LOD between NYC HANES and NHANES, we do not present NHANES geometric means. For NYC HANES, we assigned a value of 0.035 to participants with serum cotinine levels below the LOD, determined by the formula $\text{LOD}/\sqrt{2}$ (Pirkle et al., 1996). We used multiple logistic regression to assess independent demographic predictors of elevated serum cotinine levels in NYC HANES. We included demographic variables (age, sex, race/ethnicity, country of birth, education, and income) in the model to assess risk factors that may explain SHS exposure. We classified respondents as “Asian” if they reported being either “Asian” or “Native Hawaiian/Other Pacific Islander.”

We weighted all analyses to adjust for the complex sampling design, nonresponse, and poststratification. We adjusted the weights further to address component- and item-level nonresponse (Mohadjer, Montaquila, & Waksberg, 1996). We used SAS version 9.0 for statistical analyses and SUDAAN version 10.0 to apply sample weights and to obtain *SE* estimates by Taylor series linearization. Statistical significance for differences in prevalence for univariate comparisons was determined at the $\alpha=.05$ level using the *t* statistic derived from the general linear contrast procedure. We calculated relative *SEs* and 95% *CI*s for means and percentages; relative *SEs* greater than 30% were noted as “unreliable.”

Results

Compared with national adult population characteristics (as reported in NHANES), more NYC adults were foreign born (51.3% vs. 15.2%), had less than a high school education (26.6% vs. 18.2%), and had an annual income of less than US\$20,000 (32.4% vs. 23.6%). Non-Hispanic Blacks and Hispanics comprised a greater proportion of the NYC population than was found nationally (23.1% vs. 11.4% and 26.1% vs. 11.4%, respectively). In NYC, Asians comprised 10.9% of the population, whereas nationally the sample size of Asian adults was insufficient for Asian-specific NHANES results to be reported. The category of “non-Hispanic other” in NHANES, which includes any Asians, comprised 4.4% of the population.

Smokers were defined as those having a serum cotinine level higher than 10 ng/ml. Based on this cutoff level, overall smoking

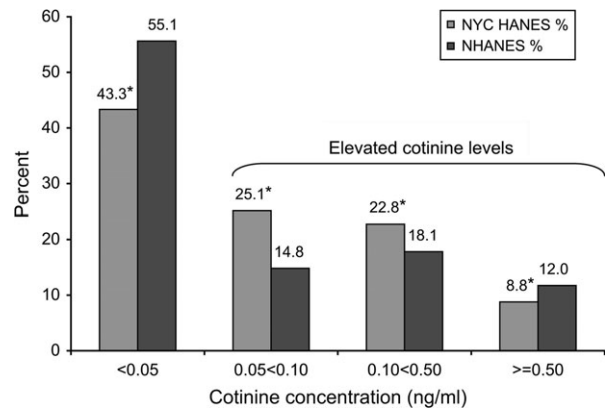


Figure 1. Cotinine levels among nonsmokers, New York City Health and Nutrition Examination Survey 2004 and National Health and Nutrition Examination Survey 2003–2004. Nonsmokers defined as those with a cotinine level of 10 ng/ml or lower; age adjusted to 2000 U.S. standard population. * $p < .05$.

prevalence was 23.3% in NYC, significantly lower than the national prevalence of 29.7% ($p < .05$; see Figure 1). Smoking prevalence by population subgroups demonstrated a generally consistent pattern: smoking prevalence in both the NYC and U.S. populations was higher in the youngest age group, males, those born in the United States, and those earning less than \$20,000 per year (Table 1). Nationally, those with less than a high school education had a significantly higher smoking prevalence than those with at least a high school education. In NYC, the effect of education did not reach statistical significance ($p < .10$).

In comparing NYC and U.S. smoking prevalence by demographic subgroups, we found that NYC had significantly lower estimates for respondents aged 20–39 years (26.6% in NYC vs. 35.7% nationally, $p < .01$), males (26.5% vs. 36.8%, $p < .01$), Whites (24.2% vs. 31.5%, $p < .05$), and across income and education levels examined ($p < .05$). Also, for White and Black males, NYC smoking prevalence was significantly lower than the national smoking prevalence ($p < .01$).

A higher proportion of NYC nonsmokers had an elevated cotinine level compared with nonsmokers nationally (56.7% vs. 44.9%, $p < .01$). Table 2 shows elevated cotinine levels by demographic subgroup for NYC and the United States. We found similar patterns in the percentage of nonsmokers with elevated cotinine levels across age and gender groups, with a few notable exceptions. In general, NYC nonsmokers were significantly more likely to have elevated cotinine levels than their U.S. counterparts, except for adults aged 60 years or older, White females, Black males, and those with an annual income below \$20,000. Although no national comparison was available for Asian adults, NYC Asian adults had the highest percentage of elevated cotinine (68.7%) of any demographic-specific strata analyzed in either survey. When these estimates were stratified by income level, Asians earning less than \$20,000 annually had a significantly higher prevalence of elevated cotinine (79.5%) than Asians earning a higher income ($p < .001$). A comparison of race-gender strata within the NYC nonsmoking population showed that the Asian nonsmoking population was the only racial/ethnic group in which males did not have a significantly higher prevalence of elevated cotinine than females.

Table 1. Smoking prevalence, overall and by demographic subgroup, New York City Health and Nutrition Examination Survey (NYC HANES 2004) and nationally (National Health and Nutrition Examination Survey [NHANES] 2003–2004)

Characteristics	NYC HANES			NHANES		
	Number of subjects	%	95% CI	Number of subjects	%	95% CI
Total*	1,767	23.3	20.6–26.1	4,476	29.7	26.3–33.2
Age (years)						
20–39*	885	26.6	23.1–30.4	1,554	35.7	31.1–40.7
40–59	653	26.4	22.1–31.1	1,273	31.5	27.1–36.3
60+	229	12.6	8.8–17.9	1,649	16.3	14.4–18.4
Gender						
Male*	752	26.5	23.1–30.2	2,170	36.8	31.6–42.3
Female	1,015	20.6	17.5–24.1	2,306	23.0	20.2–26.1
Race/ethnicity by gender						
White*	516	24.2	19.7–29.4	2,415	31.5	27.7–35.5
Male*	255	25.7	20.0–32.3	1,173	37.9	31.3–45.1
Female	261	22.7	17.1–29.4	1,242	25.3	22.1–28.8
Black	375	27.3	21.6–33.8	873	34.3	27.8–41.5
Male*	146	29.0	21.8–37.4	420	45.5	37.0–54.3
Female	229	26.1	19.0–34.8	453	25.6	18.8–33.8
Hispanic	621	19.2	16.2–22.6			
Male	238	23.7	18.8–29.3			
Female	383	16.9	13.3–21.3			
Non-Hispanic Asian	226	23.5	17.8–30.2			
Male	101	35.9	26.4–46.6			
Female	125	12.2	7.0–20.4			
Mexican American				900	18.2	15.5–21.4
Male				436	25.7	21.2–30.6
Female				464	10.3	7.2–14.4
Other includes multiple races				150	20.1	14.0–28.0
Male				78	31.9	22.9–42.5
Female				72	8.5	3.7–18.3
Other Hispanic				138	25.2	18.6–33.1
Male				63	33.4	20.3–49.7
Female				75	19.8	14.3–26.7
Nativity						
U.S. born	786	29.4	25.2–34.1	3,545	31.5	28.0–35.1
Foreign born	975	18.0	15.2–21.3	930	20.4	16.8–24.4
Education						
<High school*	507	26.4	22.2–31.1	1,306	37.4	31.8–43.4
≥High school*	1,252	22.2	19.5–25.1	3,163	28.1	25.0–31.4
Income						
<US\$20,000*	600	28.2	24.3–32.4	1,445	39.5	34.7–44.5
≥\$20,000*	1,115	21.7	18.5–25.2	2,909	26.9	23.7–30.3

Note. Smokers are defined as those with a cotinine level higher than 10 ng/ml; prevalences are age adjusted to the U.S. standard population.

*NYC HANES versus NHANES, $p < .05$.

The overall geometric mean for cotinine among nonsmokers in NYC was 0.086 ng/ml (Table 3). Males had a significantly higher geometric mean than females (0.099 vs. 0.078 ng/ml for females, $p < .000$). Asians had a significantly higher geometric mean for cotinine than Whites and Hispanics. Asian nonsmokers had the highest geometric mean estimate (0.110 ng/ml) of any demographic subgroup examined. Furthermore, when race and gender were considered, Asian males had the highest geometric mean (0.135 ng/ml). Among other demographic subgroups examined, we found no differences in geometric mean cotinine levels, including across nativity (U.S. born vs. foreign born), education levels, or

income levels. Because of differences in the LOD in the NYC HANES and NHANES cotinine laboratory analyses, we could not compare geometric means between the samples; however, a comparison of the distribution of cotinine levels among nonsmokers with elevated cotinine levels in each survey showed that NYC nonsmokers with elevated cotinine had lower levels of cotinine than those nationally (25.1% of NYC nonsmokers and 14.8% of nonsmokers nationally had a cotinine level between 0.05 and 0.10 ng/ml, $p < .05$). Compared with NYC nonsmokers, a significantly larger proportion of nonsmokers nationally had cotinine levels higher than 0.50 ng/ml (8.8% vs. 12.0%, $p < .05$; Figure 1).

Table 2. Nonsmokers with elevated cotinine levels (≥ 0.05 ng/ml), New York City Health and Nutrition Examination Survey (NYC HANES 2004) and nationally (National Health and Nutrition Examination Survey [NHANES] 2003–2004)

Characteristics	NYC HANES			NHANES		
	Number of subjects	%	95% CI	Number of subjects	%	95% CI
Total*	1,330	56.7	53.6–59.7	3,285	44.9	38.1–51.8
Age (years)						
20–39*	656	64.4	60.2–68.4	1,049	49.4	42.1–56.8
40–59*	478	55.1	50.1–59.9	867	45.1	37.2–53.2
60+	196	46.1	39.0–53.4	1,369	36.7	29.9–44.1
Gender						
Male*	537	64.3	60.0–68.3	1,422	50.8	43.9–57.6
Female*	793	50.9	46.9–54.9	1,863	40.4	33.0–48.3
Race/ethnicity by gender						
White*	388	54.5	49.8–59.0	1,742	43.3	34.8–52.2
Male*	185	61.3	54.7–67.6	767	50.9	42.4–59.4
Female	203	47.3	40.8–53.8	975	37.6	27.9–48.4
Black	259	57.4	50.8–63.6	565	62.6	53.6–70.8
Male	98	67.5	58.2–75.7	229	64.1	52.3–74.4
Female*	161	49.8	41.4–58.1	336	61.6	53.1–69.4
Hispanic	488	54.6	49.4–59.7			
Male	180	62.7	53.1–71.4			
Female	308	48.7	42.5–55.0			
Non-Hispanic Asian	173	68.7	57.6–77.9			
Male	65	71.4	52.5–84.9			
Female	108	66.9	55.3–76.7			
Mexican American				750	36.6	30.0–43.8
Male				327	45.7	39.2–52.3
Female				423	28.3	20.1–38.3
Other (includes multiple races)				121	45.5	31.8–59.9
Male				54	45.5	22.8–70.2
Female				67	45.9	33.4–59.0
Other Hispanic				107	44.2	30.3–58.9
Male				45	49.7	23.7–75.9
Female				62	42.7	33.5–52.5
Nativity						
U.S. born*	530	54.6	49.9–59.3	2,502	45.2	37.3–53.4
Foreign born*	797	58.5	54.5–62.4	783	43.8	37.6–50.1
Education						
<High school*	372	65.7	59.8–71.1	938	53.7	46.6–60.7
\geq High school*	953	54.0	50.5–57.5	2,342	43.4	36.3–50.7
Income						
<US\$20,000	428	63.3	57.4–68.9	972	57.1	50.7–63.3
\geq \$20,000*	861	54.1	50.7–57.4	2,231	41.9	35.2–48.9

Note. Nonsmokers are defined as those with a cotinine level of 10 ng/ml or lower; prevalences are age adjusted to the U.S. standard population.

*NYC HANES versus NHANES, $p < .05$.

Table 4 shows results from the logistic regression model examining factors associated with having elevated cotinine levels in the NYC population. We found that younger age, male gender, and low education were independently associated with having an elevated cotinine level. Specifically, persons aged 20–39 years were nearly 2.5 times more likely and those aged 40–59 years were about 60% more likely than those aged 60 years or older to have an elevated cotinine level. Men were nearly twice as likely as women and Asians were nearly two-and-a-half times more likely than Hispanics to have an elevated cotinine level. Those with less than a high school education were 64% more likely than

those with at least a high school education to have an elevated cotinine level.

Discussion

Findings from this population-based examination survey suggest that, despite having a lower smoking prevalence than the national average, a larger proportion of NYC residents may be experiencing SHS exposure compared with their national counterparts. This elevation existed nearly one-and-a-half years after

Table 3. Cotinine levels among New York City nonsmokers by selected characteristics

Characteristic	Number of subjects	Geometric mean (ng/ml)	95% Confidence interval
Total	1,330	0.086	0.080–0.093
Age (years)			
20–39	656	0.097	0.087–0.108
40–59	478	0.082	0.073–0.090
60+	196	0.077	0.064–0.089
Gender			
Male	537	0.099	0.088–0.110
Female	793	0.078	0.070–0.085
Race/ethnicity by gender			
White	388	0.081	0.072–0.091
Male	185	0.092	0.075–0.109
Female	203	0.071	0.059–0.084
Black	259	0.090	0.077–0.102
Male	98	0.099	0.079–0.118
Female	161	0.083	0.066–0.101
Hispanic	488	0.083	0.073–0.092
Male	180	0.097	0.078–0.117
Female	308	0.074	0.064–0.084
Non-Hispanic Asian	173	0.110	0.087–0.134
Male	65	0.135	0.082–0.188
Female	108	0.097	0.078–0.117
Nativity			
U.S. born	530	0.087	0.078–0.097
Foreign born	797	0.086	0.078–0.094
Education			
<High school	372	0.104	0.086–0.123
≥High school	953	0.081	0.074–0.087
Income			
<US\$20,000	428	0.098	0.083–0.112
≥\$20,000	861	0.082	0.075–0.088

Note. Nonsmokers are defined as those with a cotinine level of 10 ng/ml or lower.

comprehensive smoke-free workplace legislation was established in NYC. More than half of all NYC nonsmokers had elevated cotinine levels (57%), suggesting recent SHS exposure, a level significantly higher than national findings (45%). Asian adults appear to experience even more SHS exposure than other demographic groups. Nearly, three quarters of Asian adults had elevated cotinine levels, and they had the highest geometric mean cotinine level of any primary subgroup examined (0.110 ng/ml). However, a larger proportion of nonsmokers nationally had cotinine levels higher than 0.50 ng/ml, suggesting that, among nonsmokers exposed to SHS, the concentration of exposure is lower in NYC than it is nationally.

The higher proportion of the NYC nonsmoking population with elevated cotinine levels may be explained in part by the demographic composition of NYC's population. Whites, a relatively small proportion of which have elevated cotinine levels, represent a much smaller proportion of the NYC population

Table 4. Adjusted odds ratios (ORs) for elevated cotinine levels (≥0.05 ng/ml) among New York City nonsmokers for selected characteristics (logistic regression)

Characteristics	Elevated cotinine	
	Adjusted OR ^a	95% CI
Age (years)		
20–39	2.37	1.57–3.56
40–59	1.62	1.13–2.33
≥60	1.00	
Gender		
Male	1.96	1.50–2.55
Female	1.00	
Race		
White	1.29	0.91–1.83
Black	1.34	0.89–2.00
Asian	2.39	1.38–4.14
Hispanic	1.00	
Nativity		
U.S. born	1.00	
Foreign born	0.96	0.69–1.34
Education		
<High school	1.63	1.09–2.46
≥High school	1.00	
Income		
<US\$20,000	1.38	1.00–1.90
≥\$20,000	1.00	

Note. Nonsmokers are defined as those with a cotinine level of 10 ng/ml or lower.

^aORs are adjusted for all variables listed.

than they do nationally (38% vs. 73%; data not shown). Asians, who comprise more than one tenth of the NYC population and have the highest geometric mean cotinine values of any demographic subgroup examined in NYC, represent a small proportion of adults nationally. However, the prevalence of elevated cotinine levels is generally higher across many NYC sociodemographic strata when compared with national levels, suggesting that sociodemographic differences in population composition do not fully explain the higher prevalence levels of elevated cotinine among NYC nonsmokers.

A possible explanation for the higher prevalence of elevated cotinine in NYC overall and among most demographic subgroups may be that NYC residents face unique exposure to SHS due to the density of the urban environment (Matsukura et al., 1984). NYC has a population density of about 26,000 people and more than 10,000 housing units per square mile compared with national values of 80 people and 33 housing units per square mile (U.S. Census Bureau, 2000). Exposure to SHS at building and public transportation entrances may be more intensive in NYC compared with other urban areas and may have an effect on SHS exposure, particularly given evidence suggesting detectable levels of SHS in outdoor areas (California Environmental Protection Agency, 2005) and given that proximity to the source of smoke outdoors can produce SHS levels equal

to or exceeding those associated with indoor SHS exposure (Klepeis, Ott, & Switzer, 2007). Household crowding also may explain the greater SHS exposure in NYC. Whereas only 30% of U.S. residents live in housing that has more than 0.5 residents per room, nearly 50% of NYC residents live in such conditions (U.S. Census Bureau, 2005).

The analyses of smoking prevalence and SHS exposure presented here demonstrate that some subgroups not previously identified may be in need of targeted prevention efforts. For instance, although smoking prevalence was not higher in Asians in NYC, they had the highest prevalence of SHS exposure. Moreover, the disparity is explained almost entirely by the high prevalence of elevated cotinine levels among low-income Asians, who represent more than one third of the NYC Asian population. The fact that there were no gender differences in levels of elevated cotinine among Asians, in contrast to all other racial/ethnic strata, suggests that smoking may be more persistent in the homes of Asian adults. Although NHANES and other studies have demonstrated differences in cotinine levels by race (Pirkle et al., 2006), no analysis to date has provided Asian-specific estimates of cotinine exposure. Thus, no national comparisons are available. Nevertheless, at least one study has demonstrated that Chinese Americans had low awareness of the adverse health consequences of smoking (Shelley et al., 2004). Other research has indicated that Asian American populations are exposed to SHS at home (Ma, Tan, Fang, Toubbeh, & Shive, 2005). Education may be an effective strategy for changing the prevalence of smoking and SHS exposure among this population.

Assessing differences in self-reported and biologically measured SHS exposure helps to identify populations that may need targeted intervention to raise awareness about the dangers of even minimal SHS exposure (Otsuka et al., 2001). Although NYC HANES did not include a measure of self-reported exposure to SHS at home, data collected in the same year as NYC HANES from an annual local-level telephone-based survey suggest that only 5% of NYC nonsmoking adults report home SHS exposure and 13% report SHS at work; no increased rates of self-reported SHS at home or work were found in the Asian respondents to that survey (NYCDHMH, 2008). National comparison data are not available for these measures, but these data suggest that the reported practice of smoking at home in NYC is relatively uncommon. Our finding that 57% of nonsmoking NYC adults had an elevated SHS exposure level suggests that other exposure sources—including those that are not apparent to the respondent—may be significant in NYC. Furthermore, the finding that a greater proportion of NYC nonsmokers with an elevated cotinine level had a cotinine level at the lowest end of the distribution, compared with nonsmokers nationally, may provide some support for this hypothesis. Given the recent finding that smoke-free laws are associated with reduced SHS exposure particularly among nonsmoking adults with no SHS exposure at home (Pickett, Schober, Brody, Curtin, & Giovino, 2006) and the fact that NYC enacted more stringent smoking legislation than the rest of the United States (Centers for Disease Control and Prevention [CDC], 2005), the lower prevalence of SHS exposure would be expected.

A notable finding is that smoking prevalence, defined here as a cotinine level of 10 ng/ml or higher, was 23.3% in NYC

and 29.7% nationally. The NYC and national prevalences reported here are higher by similar magnitudes (27% higher in NYC and 39% higher nationally) than results obtained from a random-digit-dialed telephone survey conducted in the same year—18.4% and 21.4% adult smoking prevalence, respectively (CDC, 2008; NYCDHMH, 2008)—presumably reflecting a more accurate assessment than is possible with self-reported smoking status captured via telephone survey.

The present study's primary strength is that it is the first population-based assessment of SHS exposure conducted at the community level using a biologically measured indicator. The information gathered is being used to develop intervention strategies with the goal of reducing SHS exposure in NYC. However, the lack of a measure of self-reported SHS exposure at home prevented us from explaining differences in self-reported exposure between NYC and the United States. Second, the differences in sensitivity of the laboratory tests prevented direct comparison of geometric mean cotinine levels in each population. Finally, although the sample was designed to be representative of the NYC adult population aged 20 years or older, the overall NYC HANES study response rate was 55%. Thus, reported estimates may be biased. However, all data reported were weighted using information on age, gender, race/ethnicity, income, education, language spoken at home, and household size to correct for bias related to these factors. Data used for weighting were obtained from interview or from neighborhood census data.

In summary, we found, unexpectedly, that a greater proportion of NYC adults are exposed to SHS than are adults nationally, despite lower levels of smoking. Sociodemographic differences in the NYC population account only partially for the observed higher prevalence of SHS exposure. The higher prevalence across racial/ethnic and socioeconomic strata in NYC compared with nationally suggests that SHS exposure in dense, urban settings may be elevated, although the concentration of the SHS exposure may be lower than that found nationally. Despite NYC's comprehensive smoke-free air legislation, certain groups continue to be exposed to SHS, particularly those in the lower socioeconomic strata and Asians. More interventions may be needed to reduce home exposure, protect all workers, and reduce public exposure to SHS.

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Declaration of Interests

None declared.

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