Deaths From Secondhand Smoke Exposure in the United States: Economic Implications

Wendy Max, PhD, Hai-Yen Sung, PhD, and Yanling Shi, MS

Exposure to secondhand smoke (SHS) has been linked to several fatal illnesses among infants and adults.¹ Worldwide, 603 000 deaths have been attributed to SHS exposure.² In the United States, the Centers for Disease Control and Prevention reported that 46 000 adults died from ischemic heart disease (IHD) and 3400 adults died from lung cancer annually between 2000 and 2004 as a result of SHS exposure.^{1,3} In addition, 776 infants were reported to have died as a result of maternal exposure in utero each year.^{1,3} Premature death results in years of productive life lost as well as economic losses.

Active smoking has been shown to place a disproportionately high burden on communities of color,⁴ including Blacks and Hispanics.^{5,6} Blacks have also been shown to be more likely to be exposed to SHS.^{7–9} However, the economic impact of SHS exposure on different racial/ethnic groups has yet to be examined.

Previous studies have estimated the impact of SHS exposure on mortality using selfreport exposure measures or assuming that those who live with smokers are exposed, but these measures yield much lower exposure estimates than biomarker-determined exposure. In 2003 to 2004, 14.8% of adults reported home or work exposure, but fully 42.4% had detectable serum cotinine.⁸ Several recent studies have examined the association between cotinine levels and cardiovascular disease and reported a greater risk of cardiovascular disease among SHS– exposed adults than among those not exposed.^{10,11}

The purpose of this study was to estimate the number of SHS-attributable deaths, years of potential life lost (YPLL), and the value of lost productivity for different US racial/ ethnic groups in 2006. We estimated the number of SHS-attributable deaths for adults using cotinine-measured SHS exposure for the first time. *Objectives.* We estimated the number of deaths attributable to secondhand smoke (SHS), years of potential life lost (YPLL), and value of lost productivity for different US racial/ethnic groups in 2006.

Methods. We determined the number of SHS-related deaths among nonsmokers from 2 adult and 4 infant conditions using an epidemiological approach. We estimated adult SHS exposure using detectable serum cotinine. For each death, we determined the YPLL and the value of lost productivity.

Results. SHS exposure resulted in more than 42 000 deaths: more than 41 000 adults and nearly 900 infants. Blacks accounted for 13% of all deaths but 24% to 36% of infant deaths. SHS–attributable deaths resulted in a loss of nearly 600 000 YPLL and \$6.6 billion of lost productivity, or \$158 000 per death. The value of lost productivity per death was highest among Blacks (\$238 000) and Hispanics (\$193 000).

Conclusions. The economic toll of SHS exposure is substantial, with communities of color having the greatest losses. Interventions need to be designed to reduce the health and economic burden of smoking on smokers and nonsmokers alike and on particularly vulnerable groups. (*Am J Public Health.* 2012; 102:2173–2180. doi:10.2105/AJPH.2012.300805)

METHODS

We estimated 3 SHS-attributable mortality outcome measures: deaths, YPLL, and productivity losses. Because separating the health impacts of active and passive smoking is difficult, we focused on nonsmokers, as have most previous studies of the health effects of SHS exposure.² We calculated mortality measures for 2 conditions (lung cancer and IHD) found in adults (aged 20 years and older) and 4 conditions (sudden infant death syndrome, low birth weight, respiratory distress syndrome, and other respiratory conditions of newborns) found in infants younger than 1 year. We selected these conditions because strong statistical evidence has indicated a causal link between SHS exposure and death from the condition.^{1,12} For each condition, we determined the number of SHS-attributable deaths and the number of YPLL and the productivity losses associated with these deaths. We describe each of these 3 SHS-attributable mortality measures in detail in the Deaths Attributable to Seconhand Smoke section.

Data Sources

We used multiple data sources for the current study. The National Health and Nutrition Examination Survey is a household survey conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention. It contains a nationally representative sample of noninstitutionalized civilians of all ages selected on the basis of a complex sampling design.¹³ Participants complete a faceto-face interview survey, which includes questions about demographic and socioeconomic characteristics, health-related conditions, smoking and tobacco use, and exposure to SHS. They then receive a physical examination, which includes drawing blood samples for serum cotinine analysis for all individuals aged 3 years and older. Beginning in 1999, Blacks, Mexican Americans, adolescents aged 12 to 19 years, older adults aged 60 years or older, and low-income people have been oversampled to improve the stability of the statistical estimates for these subgroups. We analyzed data from the 2003 to 2006 National Health and Nutrition Examination Survey cycles, which were

pooled to increase the sample size. The combined dataset included 12 704 adults: 6562 non-Hispanic Whites, 2436 non-Hispanic Blacks, 3230 Hispanics, and 476 others.

The National Health Interview Survey is a cross-sectional household interview survey. The sampling plan permits the representative sampling of US dwelling units containing members of the civilian noninstitutionalized population (households and noninstitutional group quarters such as college dormitories).¹⁴ Since 2006, Asians, Blacks, and Hispanics have been oversampled.¹⁴ The 2006 National Health Interview Survey contains information on 23 322 adults: 14 041 non-Hispanic Whites, 3820 non-Hispanic Blacks, 4032 Hispanics, and 1429 others.

The Multiple Cause of Death Data contains records for all deaths that occur in the 50 states and the District of Columbia each year and is compiled by the National Center for Health Statistics.¹⁵ The 2006 Multiple Cause of Death Data file¹ contains information on 2 426 264 deaths.

Measures

Race/ethnicity. We considered 4 different race/ethnicity groups: non-Hispanic Whites (referred to as Whites), non-Hispanic Blacks (referred to as Blacks), Hispanics, and other race/ ethnicity, including Asians, Pacific Islanders, American Indian/Alaska natives, and multiple races.

Secondhand smoke exposure. Data on infant exposure to maternal smoking in utero are available from birth certificates. In 2006, 17 states included a question asking about tobacco use during each trimester of pregnancy as well as in the 3 months before becoming pregnant.¹⁶ Exposure estimates are reported for Whites, Blacks, and Hispanics. We used the exposure reported for all races and origins for the other race/ethnicity category.

We determined adult SHS exposure from the 2003–2006 National Health and Nutrition Examination Survey data, which includes both self-reported SHS exposure and serum cotinine-measured SHS exposure. The primary estimates we report are based on cotininemeasured SHS exposure. We conducted a sensitivity analysis using self-reported SHS exposure. Cotinine-measured SHS exposure was defined as having a detectable serum cotinine level of 0.05 nanograms per milliliter or higher.⁷ Selfreported exposure to SHS at home was defined as living in a household in which any household member smokes inside the home. Self-reported workplace SHS exposure was defined as smelling the smoke from other people's cigarettes, cigars, or pipes for 1 or more hour during the previous week. Anyone exposed to SHS at home or at work was considered to be exposed according to self-report.

Smoking prevalence. To determine the number of deaths among nonsmokers, we needed to determine the number of deaths from active smoking. Thus, for the analyses we needed the prevalence of current, former, and never smoking. All infants younger than 1 year were assumed to be never smokers. Among adults, a current smoker was someone who had smoked at least 100 cigarettes in his or her lifetime and who currently smoked. A former smoker was someone who had smoked 100 cigarettes but did not now smoke. A never smoker was someone who had not smoked 100 cigarettes. We obtained adult smoking prevalence from the 2006 National Health Interview Survey data.

Deaths from causes related to secondhand smoke. We obtained the total number of deaths from the 2006 Multiple Cause of Death Data for each condition by gender, 5-year age group, and race/ethnicity. We identified deaths from the 6 SHS–associated conditions from the International Classification of Disease, 10th Revision, codes:

Ischemic heart disease: I20–I25 Lung cancer: C33–C34 Low birth weight: P07 Sudden infant death syndrome: P22 Respiratory distress syndrome: P23–P28 Respiratory conditions of the newborn: R95

Relative risk of death. The relative risk (RR) of death represents the rate of death among those exposed compared with the rate of death among those who were not exposed. We obtained adult RRs of death for current and former smoking from the 2004 adult module of the Smoking-Attributable Mortality, Morbidity, and Economic Costs computer application maintained by the Centers for Disease Control and Prevention.¹⁷ We obtained the RR for IHD from SHS exposure from Whincup et al.¹⁰ This study prospectively measured the

risk of coronary heart disease associated with SHS exposure using serum cotinine concentration. They reported a RR of 1.32. We obtained the RR for lung cancer from the 2005 California Environmental Protection Agency (EPA) report,¹ which recommended using a RR of 1.29, the lower bound of the range it reported. This estimate was reported in "the best US study which quantified the exposure on the basis of cotinine levels."^{1(p7)} We obtained the RRs of deaths from infant exposure to maternal smoking in utero from the 2004 Maternal and Child Health module of Smoking-Attributable Mortality, Morbidity, and Economic Costs;¹⁷ these RRs, which are for infants younger than 1 year, reflect risks from maternal smoking during pregnancy: low birth weight, 1.83; sudden infant death syndrome, 2.29; respiratory distress syndrome, 1.30; and respiratory conditions of newborns, 1.41.

Deaths Attributable to Secondhand Smoke

We determined the number of SHS– attributable deaths for each condition by multiplying the SHS–attributable fraction by the total number of deaths for that condition among nonsmokers.

Secondhand smoke–attributable fraction among nonsmokers. The SHS–attributable fraction, SAF_{shs} , is calculated by the standard epidemiological formula as¹⁸

where P_{shs} is the prevalence of SHS exposure among nonsmokers, and RR_{shs} is the RR of death of SHS–exposed nonsmokers compared with that of unexposed nonsmokers.

Number of deaths among nonsmokers. Because separate mortality statistics for smokers and nonsmokers in the United States are not available, we estimated the number of deaths among nonsmokers. All infants were regarded as nonsmokers. For the 2 adult conditions (lung cancer and IHD), we determined the number of deaths among nonsmokers following the method used by Gan et al.¹⁹ and Oberg et al.² First, we determined the number of excess deaths attributable to current smoking for each condition. Second, we subtracted these excess deaths from the total deaths among

all adults for each condition to derive the total number of deaths that were not attributable to current smoking but that resulted from other (i.e. nonsmoking) risk factors that affect both smokers and nonsmokers.²⁰ Third, we apportioned the total non–smoking-attributable deaths for each condition to smokers (those who die from the disease but whose death is not attributable to smoking) and nonsmokers according to the proportion of smokers and nonsmokers in the adult population. These steps are expressed by the following formula:

$$(2) \ \ D_{nons} = [D - (D \times SAF_{cs})] \times (1 - P_{cs}), \label{eq:Dnons}$$

where $D_{\rm nons}$ is the total number of deaths among nonsmokers, D is the total number of deaths among all adults, SAF_{\rm cs} is the smoking-attributable fraction from current smoking, and $P_{\rm cs}$ is the prevalence of adult current smoking in United States.

We derived SAF_{cs} from the prevalence of smoking and the relative risk of death from smoking according to the standard epidemiological formula¹⁸

(3)
$$SAF_{cs} = [P_{cs} * (RR_{cs} - 1)]/[P_{ns} + P_{fs} * RR_{fs} + P_{cs} * RR_{cs}]$$

where P_{cs} is the prevalence of adult current smoking in the United States, P_{ns} is the

prevalence of never-smoking adults in the United States, P_{fs} is the prevalence of formersmoking adults in the United States, RR_{cs} is the RR of death from current smoking, and RR_{fs} is the RR of death from former smoking.

Years of Potential Life Lost Attributable to Secondhand Smoke

We estimated SHS-attributable YPLL as the product of SHS-attributable deaths and the average number of years of life expectancy remaining at the age of death, which were obtained from the 2006 United States Life Tables by Hispanic Origin.²¹ Years of life expectancy remaining were determined separately for Whites, Blacks, Hispanics, and others.

Productivity Losses Attributable to Secondhand Smoke

We estimated the value of lost productivity from SHS–attributable deaths as the product of SHS–attributable deaths and the present value of lifetime earnings for each person who died. We calculated 2006 age- and gender-specific present value of lifetime earnings using a computer program maintained at the University of California, San Francisco. The program takes into account life expectancy, the probability that a person of a given age and gender will be in the labor market or keeping house, labor market earnings, and an imputed value for household production. Future earnings were discounted at 3% to convert all dollars into their 2006 present value. Further details about this program are available elsewhere.²² These productivity losses represent an indirect social cost rather than a forgone dollar expenditure.

RESULTS

Infant rates of exposure to maternal smoking in utero and adult SHS exposure measured by detectable serum cotinine are provided in Table 1. Infant exposure in utero ranged from 2.8% for Hispanics to 18.1% for Whites. Adult SHS exposure ranged from 34.7% for Hispanics to 58.4% for Blacks. On the basis of bivariate logistic regression analysis, Black adults had significantly greater exposure rates than did Whites in all age groups and for men and women. For the other race/ethnicity group, we estimated SHS exposure by combining all aged 20 years and older because of small sample size. The highest SHS exposure was for Black men aged 45 to 64 years (63.6%) followed by Black men aged 20 to 44 years (62.7%). Black women aged 20 to 44 years had a higher exposure rate (62.3%) than did any other women.

TABLE 1—Infant Exposure to Maternal Smoking In Utero (2006) and Adult Cotinine-Measured Secondhand Smoke Exposure (2003–2006) by Gender, Age, and Race/Ethnicity: United States

Age, Y	White, Mean (95% CI)	Black, Mean (95% Cl)	Hispanic, Mean (95% Cl)	Other, ^a Mean (95% CI)	All, Mean (95% Cl)
All infants ^b < 1	18.1	10.6	2.8	13.2	13.2
All adults ≥ 20	36.9 (32.1, 41.8)	58.4* (52.6, 64.2)	34.7 (30.3, 39.0)	40.0 (29.9, 50.1)	39.1 (35.2, 42.9)
Men					
≥20	43.2 (38.3, 48.1)	61.4* (54.1, 68.6)	40.9 (35.5, 46.4)	41.6 (25.1, 58.1)	44.5 (40.4, 48.6)
20-44	50.5 (43.4, 57.5)	62.7* (52.6, 72.7)	41.9 (34.9, 49.0)	41.6 (25.1, 58.1)	49.7 (44.2, 55.1)
45-64	40.0 (33.6, 46.4)	63.6* (55.1, 72.1)	40.6 (26.7, 54.5)	41.6 (25.1, 58.1)	42.2 (36.8, 47.5)
≥65	36.7 (31.5, 41.9)	50.7* (39.3, 62.0)	33.8 (18.8, 48.8)	41.6 (25.1, 58.1)	37.5 (32.8, 42.2)
Women					
≥20	32.1 (26.6, 37.7)	56.6* (50.8, 62.4)	29.6 (24.8, 34.4)	39.0 (29.8, 48.2)	35.0 (30.8, 39.2)
20-44	32.6 (26.2, 38.9)	62.3* (55.1, 69.6)	29.9 (23.1, 36.7)	39.0 (29.8, 48.2)	36.9 (32.7, 41.2)
45-64	34.9 (26.9, 42.9)	50.7* (42.4, 59.1)	30.4 (21.0, 39.8)	39.0 (29.8, 48.2)	36.3 (29.7, 42.8)
≥65	27.6 (22.4, 32.8)	48.9* (36.3, 61.6)	24.9 (14.1, 35.8)	39.0 (29.8, 48.2)	29.2 (24.3, 34.2)

Note. Cl = confidence interval.

^aCell sizes < 25 were combined for stability of estimates.

^bInfant exposure rates derived from Martin, Hamilton, Sutton, et al.¹⁶ Confidence intervals were not available.

*Statistically significant difference from Whites (Ref) at P < .05, 2-tailed test, based on bivariate logistic regression.

TABLE 2—Deaths Attributable to Secondhand Smoke (SHS) and Years of Potential Life Lost (YPLL) by Cause of Death, Gender, and Race/Ethnicity: United States, 2006

	White		Black	I	Hispan	ic	Other		Total	
	SHS-Attributable		SHS-Attributable		SHS-Attributable		SHS-Attributable		SHS-Attributable	
Cause of Death	Deaths, No.	YPLL, No.	Deaths, No.	YPLL, No.	Deaths, No.	YPLL, No.	Deaths, No.	YPLL, No.	Deaths, No.	YPLL, No
				In	fants (aged < 1 y)					
Low birth weight										
Male	132	9946	83	6261	12	877	12	882	239	17 966
Female	102	8191	71	5655	9	732	11	847	192	15 426
Total	235	18 137	154	11 917	21	1609	22	1730	432	33 393
Sudden infant dea	th syndrome									
Male	140	10 548	46	3442	6	442	10	721	202	15 154
Female	95	7651	38	3029	4	333	4	327	141	11 340
Total	236	18 199	84	6471	10	775	14	1048	343	26 494
Respiratory distres	s syndrome									
Male	11	824	4	322	1	60	1	63	17	1269
Female	7	582	4	326	1	47	0	37	12	992
Total	18	1406	8	648	1	107	1	100	29	2261
Respiratory condit	ions									
Male	24	1769	9	654	2	115	1	81	35	2619
Female	15	1197	7	534	1	95	1	86	24	1912
Total	38	2966	15	1188	3	210	2	167	59	4531
				Adu	ults (aged \ge 20 y)					
Ischemic heart dis	sease									
Male	15 638	204 375	2086	30 336	872	14 476	556	7792	19 152	256 980
Female	11 425	120 386	2293	30 029	648	9211	433	5331	14 799	164 958
Total	27 063	324 761	4379	60 365	1520	23 688	989	13 124	33 951	421 938
Lung cancer										
Male	3720	53 243	410	5874	117	1908	128	1787	4374	62 813
Female	2435	38 886	359	5933	74	1421	91	1588	2959	47 829
Total	6155	92 129	769	11 807	190	3330	219	3375	7333	110 642
					Total					
Male	19 666	280 706	2638	46 889	1008	17 879	707	11 327	24 019	356 801
Female	14 080	176 894	2771	45 508	736	11 839	541	8217	18 128	242 457
Total	33 746	457 599	5410	92 397	1745	29 718	1247	19 544	42 147	599 258

Note. Columns may not sum because of rounding.

Deaths Attributable to Secondhand Smoke

In 2006, more than 42 000 Americans died of SHS-attributable diseases, including more than 41 000 adults and nearly 900 infants (Table 2). Among these deaths, 80% were Whites, 13% were Blacks, and 4% were Hispanics. IHD accounted for 34 000 deaths, and lung cancer caused 7000 deaths. Fully 36% of the infants who died of low birth weight caused by exposure to maternal smoking in utero were Blacks, as were 28% of those dying of respiratory distress syndrome, 25% dying of other respiratory conditions, and 24% dying of sudden infant death syndrome.

Years of Potential Life Lost

These deaths represented a loss of nearly 600 000 YPLL (Table 2), or an average of 14.2 years per death. However, they were not equally distributed across racial/ethnic subgroups. Blacks accounted for fully 15% of YPLL. The average YPLL per death was 17.0 for Hispanics and 17.1 for Blacks compared with 13.6 for Whites because people of color died at younger ages than did Whites.

Value of Lost Productivity

As a result of the deaths from SHS– attributable diseases, \$6.6 billion was lost in productivity (Table 3), which amounts to \$158 000 per death. However, the value of lost productivity per death differed by race/ ethnicity, ranging from \$238 000 for Blacks and \$193 000 for Hispanics to \$181 000 for other race/ethnicity and \$142 000 for Whites.

Sensitivity Analysis

We conducted sensitivity analyses for the estimates of IHD and lung cancer. Conducting

TABLE 3—Value of Lost Productivity From Deaths Attributable to Secondhand Smoke by Cause of Death, Gender, and Race/Ethnicity: United States, 2006

	Wh	nite	Bla	ack	Hisp	anic	Ot	her	То	tal
Cause of Death	Total, \$ Thousands	Per Death, \$								
				Infa	nts (aged < 1 y)					
Low birth weight										
Male	161 788	1 221 623	101 849	1 221 623	14 261	1 221 623	14 354	1 221 623	292 253	1 221 623
Female	98 899	968 308	68 280	968 308	8841	968 308	10 231	968 308	186 251	968 308
Total	260 687	1 111 326	170 129	1 105 548	23 102	1 110 452	24 585	1 101 691	478 504	1 108 726
Sudden infant										
death syndrome										
Male	171 583	1 221 623	55 988	1 221 623	7197	1 221 623	11 732	1 221 623	246 500	1 221 623
Female	92 380	968 308	36 574	968 308	4017	968 308	3945	968 308	136 916	968 308
Total	263 963	1 119 158	92 563	1 107 176	11 214	1 116 955	15 677	1 146 167	383 416	1 117 251
Respiratory distress	3									
syndrome										
Male	13 401	1 221 623	5233	1 221 623	977	1 221 623	1024	1 221 623	20 636	1 221 623
Female	7032	968 308	3939	968 308	565	968 308	443	968 308	11 978	968 308
Total	20 433	1 120 726	9173	1 098 237	1542	1 114 803	1466	1 132 218	32 614	1 114 536
Respiratory condition	ons									
Male	28 778	1 221 623	10 634	1 221 623	1872	1 221 623	1317	1 221 623	42 601	1 221 623
Female	14 449	968 308	6453	968 308	1143	968 308	1044	968 308	23 089	968 308
Total	43 227	1 123 389	17 087	1 111 784	3015	1 111 394	2361	1 094 965	65 690	1 118 754
				Adu	lts (aged ≥20 y)					
lschemic heart										
disease										
Male	2 655 392	169 801	557 357	267 147	198 542	227 686	109 370	196 854	3 520 661	183 820
Female	697 792	61 075	301 604	131 527	67 693	104 529	39 764	91 798	1 106 854	74 792
Total	3 353 184	123 901	858 962	196 136	266 235	175 201	149 134	150 830	4 627 515	136 299
Lung cancer										
Male	609 949	163 962	91 099	222 231	20 516	175 992	19 881	155 473	741 445	169 49
Female	241 003	98 961	47 941	133 557	10 215	138 979	12 535	137 441	311 693	105 33
Total	850 952	138 245	139 040	180 834	30 731	161 679	32 415	147 966	1 053 138	143 608
					Total					
Male	3 640 892	185 139	822 162	311 608	243 366	241 321	157 677	223 108	4 864 096	202 507
Female	1 151 555	81 785	464 791	167 730	92 474	125 619	67 961	125 727	1 776 781	98 013
Total	4 792 446	142 015	1 286 953	237 905	335 839	192 500	225 638	180 905	6 640 877	157 563

Note. Columns may not sum because of rounding.

sensitivity analyses for the infant conditions was not possible because neither confidence intervals of exposure nor alternative estimates of RR were available. For the adult conditions, we estimated SHS–attributable deaths, YPLL, and lost productivity using the upper and lower bounds of the 95% confidence interval of cotinine-measured exposure and self-reported exposure at home or at work, and upper and lower values for RR. We obtained the RR values from the California EPA report:¹ 1.2 to 1.68 for IHD and 1.29 to 1.74 for lung cancer. The results are shown in Table 4.

Estimates for the number of SHS-attributable IHD deaths ranged from 31 000 to 36 000 using cotinine-measured exposure and from 6000 to 11 000 using self-reported exposure. Estimated deaths from lung cancer ranged from 6500 to 8500 using cotinine-measured exposure and from 1400 to 2300 using self-reported exposure. Varying the relative risks results in estimated deaths from IHD ranging from 22 000 to 65 000 and those from lung cancer ranging from 7300 to 16 300. The range of estimates of YPLL and lost productivity as well as the range of each measure by race/ethnicity are also shown in Table 4.

SHS- Cause of Death Attributable Cause of Death Deaths Ischemic heart disease 24 067 Upper bound of 95% Cl 21 317 Midpoint ^b 27 063 Lower bound of 95% Cl 27 363 Lung cancer 27 063 Lung cancer 27 063 Upper bound of 95% Cl 5463 Upper bound of 95% Cl 6981					Black			Hispanic			Other			Total	
art disease und of 95% Cl und of 95% Cl und of 95% Cl und of 95% Cl		\$ JAFLE	Lost Productivity, \$ Thousands	SHS- Attributable Deaths	YPLL	Lost Productivity, \$ Thousands	SHS- Attributable Deaths	YPLL	Lost Productivity, \$ Thousands	SHS- Attributable Deaths	YPLL	Lost Productivity, \$ Thousands	SHS- Attributable Deaths	YPLL	Lost Productivity, \$ Thousands
art disease und of 95% Cl und of 95% Cl und of 95% Cl und of 95% Cl						Cotinine-m	Cotinine-measured exposure ^a	sure ^a							
und of 95% Cl und of 95% Cl und of 95% Cl und of 95% Cl															
und of 95% Cl und of 95% Cl und of 95% Cl		283 907	2 814 921	4256	58 468	829 027	1397	21 678	242 177	875	11523	126 383	30 594	375 575	4 012 508
und of 95% Cl und of 95% Cl		326 344 3	3 570 311	5334	72 539	1 003 223	2381	35 782	368 810	1359	18 028	204 215	36 391	452 693	5 146 559
und of 95% Cl und of 95% Cl		324 761	3 353 184	4379	60 365	858 962	1520	23 688	266 235	989	13 124	149 134	33 951	421 938	4 627 515
nd of 95% Cl nd of 95% Cl															
nd of 95% CI		80 805	718 239	752	11 479	133 636	172	3012	27 496	193	3008	28 286	6279	98 304	907 658
		103 646	916 556	943	14 299	163 341	295	5044	43 209	302	4662	44 691	8521	127 651	1 167 798
		92 129	850 952	769	11 807	139040	190	3330	30 731	219	3375	32 415	7333	110 642	$1\ 053\ 138$
						Self-repor	Self-reported SHS exposure ^c	ure ^c							
lschemic heart disease															
Lower bound of 95% Cl 4809		70 433	1 049 677	872	13 313	231 771	214	4896	94 766	318	4241	49 440	6213	92 882	1 425 654
Upper bound of 95% CI 7788		108 374	1 496 148	1636	24 079	392 976	663	11 812	171 519	725	9743	116 494	10 812	154 008	2 177 138
Midpoint 6333		89 772	1 275 466	1257	18 753	313 670	442	8405	133 766	527	7070	83 970	8559	124 000	1 806 873
Lung cancer															
Lower bound of 95% Cl 1123		19 261	241 603	159	2584	35 028	25	595	9446	70	1070	10 436	1377	23 509	296 513
Upper bound of 95% Cl 1797		29 822	350 219	292	4667	61 432	85	1593	18 664	162	2439	24 181	2335	38 522	454 497
Midpoint 1467		24 641	296 556	226	3634	48 408	55	1101	14 120	117	1772	17 489	1865	31 147	376 572
						ž	Relative risk								
Ischemic heart disease															
Lower bound (1.2) ^d 17 573		211 481	2 199 359	2892	39 929	570 664	983	15355	173 307	646	8571	97 438	22 094	275 336	3 040 767
Upper bound (1.68) ^d 51 724		616242 (6 246 521	8022	$110\ 106$	1550213	2930	45 471	505 446	1861	24 698	280 402	64 537	796 517	8 582 583
Best estimate (1.32) ^{b,e} 27 063		324 761	3 353 184	4379	60 365	858 962	1520	23 688	266 235	989	13 124	149 134	33 951	421 938	4 627 515
Lung cancer															
Lower bound or best 6155		92 129	850 952	769	11 807	139040	190	3330	30 731	219	3375	32 415	7333	110642	$1\ 053\ 138$
estimate (1.29) ^{b,d}															
Upper bound (1.74) ^d 13735		204 655	1855549	1631	24 961	290 309	429	7502	68 481	481	7408	71 097	16 275	244 526	2 285 436
Mote. Cl = confidence interval; SHS = secondhand smoke; YPLL = years of potential life lost. Th e estimated upper and lower bounds of the 95% confidence intervals were reported in Table 1. Th e assumption used in the main analysis with the same SHS-attributable mortality results as reported in Tables 2-3. ^{We} estimated the upper and lower bounds of the 95% confidence intervals of self-reported SHS exposure at home or at work using the 2003-2006 National Health and Nutrition Examination Survey data.	secondhan Ids of the Ialysis with ounds of t	nd smoke; 95% cont 1 the sam the 95% c	; YPLL = years fidence interva ne SHS-attribut confidence inte	years of potential life lost. intervals were reported in Table 1. ttributable mortality results as rep cce intervals of self-reported SHS e	e lost. ed in Table results as r ported SHS	1. eported in Tabl exposure at h	es 2-3. ome or at wor	k using the	2003-2006 Na	tional Health	and Nutritic	un Examination	Survey data.		

DISCUSSION

This article makes several new contributions: We presented estimates of the economic impact of SHS exposure on mortality, including YPLL and the value of productivity losses, and presented the impact of SHS-attributable mortality for different racial/ethnic groups. Finally, we calculated estimates of SHSattributable deaths from IHD and lung cancer for the first time using cotinine-measured exposure. Cotinine-measured exposure reflects SHS exposure in all settings, not just at home or at work, and results in greater SHS exposure estimates than obtained from self-report.

The impact of SHS exposure on mortality outcome measures differs by race/ethnicity, with Blacks accounting for 13% of all SHS– attributable deaths, 15% of YPLL, and 19% of productivity losses, whereas they accounted for 13% of the US population in 2006.²³ Black infants dying as a result of exposure to maternal smoking in utero accounted for a startlingly high 24% to 36% of all SHS– attributable infant deaths. The value of lost productivity per death was highest among Blacks and Hispanics. Deaths caused by SHS exposure have a disproportionate impact on communities of color.

Our estimates of SHS-attributable deaths from IHD are lower than the California EPA estimates.^{1,3} Three factors account for this difference. The EPA estimates are based on selfreported SHS exposure, which underestimates exposure. Our cotinine-based estimates tended to be higher. At the same time, several factors caused our estimates to be lower. Death data by age are now available. Age-specific death is important because the oldest age group (aged 65 years and older) experienced 85% of IHDrelated deaths²⁴ but had the lowest self-reported exposure of any age group. Finally, the number of deaths from IHD has been decreasing over time because of fewer people smoking, lifestyle changes, and improvements in therapies. The same factors accounted for the difference in our estimates of SHS-attributable lung cancer deaths, except that the number of deaths from lung cancer has been increasing over time.²⁵ The net effect is that we estimated the number of SHS-attributable deaths from IHD to be approximately 25% lower than previously

estimated, whereas the number of deaths from lung cancer was about twice as high.

Our findings are subject to several limitations. First, the analysis focused on deaths among nonsmokers because of the difficulty in separating the impact of SHS exposure and active smoking on health among smokers. However, smokers are also negatively affected by exposure to SHS. Thus, we underestimated the impact of SHS exposure.

Second, the RR of IHD from SHS is based on estimates developed from SHS exposure measured in 1978 to 1980 and heart disease developed over the next 20 years. Although these estimates are the best available, SHS exposure has been decreasing over time, with nonsmokers in 1980 exposed to greater levels of SHS than nonsmokers today. One recent study²⁶ suggested a diminished effect of lower level SHS exposure on IHD in older adults. Thus, newer estimates of RR need to be developed.

Third, we assumed productivity losses were the same for a person of a given age and gender, regardless of race/ethnicity. We did not consider earnings differentials by race/ethnicity because many of them result from labor market imperfections or past labor market discrimination that led to lower wages for some population groups. We assumed that anyone could earn what the average person earns today.

Fourth, the analysis was limited to 6 conditions for which death has been shown to be associated with SHS exposure. We selected these conditions because both the EPA¹ and the US Surgeon General¹² reports unequivocally agreed that the evidence was sufficient to establish a causal link. However, many more conditions are thought to be caused or exacerbated by SHS exposure. For example, the recent study by Oberg et al.² included adult deaths from asthma and estimated a substantial number of SHS-attributable asthma deaths. However, because the EPA and US Surgeon General reports both indicated that the evidence for asthma was suggestive but not sufficient to indicate a causal link, we did not include asthma. Similarly, evidence for a link between SHS exposure and breast cancer continues to build. Broader inclusion of SHS-attributable diseases would result in larger estimates.

Fifth, we used the same RR estimates of death from SHS exposure for all racial/ethnic groups because the RR estimates were not

available by race/ethnicity. Sixth, we were unable to calculate the 95% confidence intervals for SHS-attributable deaths, YPLL, or the value of lost productivity. However, we did conduct a sensitivity analysis. Finally, the purpose of this analysis was to estimate the impact of SHS exposure on mortality-related outcomes. We did not include the substantial impact of SHS on health care costs.

Progress has been made in reducing smoking in public places, but much work remains to be done. As of 2009, only 27 states banned smoking in private workplaces, 29 banned it in restaurants, and 22 banned it in bars,²⁷ leaving many people vulnerable to SHS exposure. Reducing SHS exposure at home is even more challenging, but signs are encouraging.²⁸ Among US households with smokers and children, the proportion with a complete smoking ban has tripled since 1992 to 1993 to 50% in 2006 to 2007.29 However, home smoking bans were less likely among households with older children, in Black households, and in households in states with high smoking prevalence.²⁸ Smoke-free laws have been shown to have a positive association with smoke-free home rules, suggesting that banning smoking in public places can have a farreaching impact of reducing SHS exposure in other settings.³⁰ Comprehensive smokefree legislation has also been shown to be associated with significantly fewer hospitalizations and deaths from coronary events and other heart disease.³¹ Thus, strengthening SHS policies will have the effect of reducing deaths from SHS exposure and the associated economic burden.

The economic toll resulting from SHSattributable deaths from just 2 adult and 4 infant conditions is substantial, totaling 42 000 deaths, 600 000 YPLL, and \$6.6 billion in lost productivity. These estimates likely underestimate the true economic impact of SHS on mortality. This burden results in communities of color suffering relatively greater losses. With the high rates of smoking prevalence and the resulting high rates of SHS exposure in the United States and in many parts of the world, interventions need to be designed that target particularly vulnerable groups and that reduce the health and economic burden of smoking on smokers and nonsmokers alike.

About the Authors

Wendy Max, Hai-Yen Sung, and Yanling Shi are with the Institute for Health and Aging, University of California, San Francisco. Wendy Max and Hai-Yen Sung are also with the Department of Social and Behavioral Sciences, University of California, San Francisco.

Correspondence should be sent to Wendy Max, PhD, Institute for Health & Aging, Suite 340, 3333 California Street, San Francisco, CA 94118 (e-mail: wendy.max@ ucsf.edu). Reprints can be ordered at http://www.ajph.org by clicking on the "Reprints" link.

This article was accepted March 14, 2012.

Contributors

W. Max obtained the funding, designed the study, and prepared the first draft of the article. H.-Y. Sung helped design the study and oversaw the data analyses. Y. Shi managed the data and conducted the analyses. All authors participated in reviewing statistical analyses and revising the article.

Acknowledgments

This research was supported by the Flight Attendants Medical Research Institute (FAMRI) and the California Tobacco Related Disease Research Program (grant 16RT-0075).

We thank the members of the University of California, San Francisco, FAMRI Bland Lane Center of Excellence on Second Hand Smoke for many helpful suggestions during the conduct of this research. We particularly appreciate the advice of Neal Benowitz and the helpful suggestions and insights of Stan Glantz.

Human Participation Protection

This study was certified as exempt by the University of California, San Francisco, Committee on Human Research.

References

1. California Environmental Protection Agency. *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant*. Sacramento, CA: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment; 2005.

2. Oberg M, Jaakola M, Woodward A, Peruga S, Pruss-Ustun A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet.* 2011;377(9760):139–146.

3. Centers for Disease Control and Prevention. Smokingattributable mortality, years of potential life lost, and productivity losses—United States, 2000–2004. *MMWR Morb Mortal Wkly Rep.* 2008;57(45):1226–1228.

4. US Department of Health and Human Services. *Tobacco Use Among U.S. Racial/Ethnic Minority Groups– African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics: A Report of the Surgeon General.* Atlanta, GA: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2008.

5. Max W, Sung H-Y, Tucker L-Y, Stark B. The disproportionate cost of smoking for African Americans in California. *Am J Public Health*. 2010;100(1):152–158.

6. Max W, Sung H-Y, Tucker L-Y, Stark B. The cost of smoking for California's Hispanic community. *Nicotine Tob Res.* 2011;13(4):248–254.

7. Pirkle JL, Bernert JT, Caudill SP, Sosnoff CS, Pechacek TF. Trends in the exposure of nonsmokers in the U.S. population to secondhand smoke: 1988-2002. *Environ Health Perspect.* 2006;114(6):853–858.

8. Max W, Sung H-Y, Shi Y. Who is exposed to secondhand smoke? Reported and serum cotinine measured exposure in the U.S., 1999-2006. *Int J Environ Res Public Health.* 2009;6(5):1633–1648.

 Schober SE, Zhang C, Brody DJ. Disparities in secondhand smoke exposure–United States, 1988– 1994 and 1999–2004. *MMWR Morb Mortal Wkly Rep.* 2008; 57(27):744–747.

 Whincup PH, Gilg JA, Emberson JR, et al. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. *BMJ*. 2004;329(7459):200–205.

11. Hamer M, Stamatakis E, Kivimaki M, Lowe GD, Batty GD. Objectively measured secondhand smoke exposure and risk of cardiovascular disease. *J Am Coll Cardiol.* 2010;56(1):18–23.

12. US Department of Health and Human Services. *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General.* Atlanta, GA: Centers for Disease Control and Prevention, Office of Smoking and Health; 2006.

13. National Center for Health Statistics. National Health and Nutrition Examination Survey Analytic Guidelines: June 2004 Version. Centers for Disease Control and Prevention Web site. Available at: http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/analytical_guidelines.htm. Accessed March 8, 2011.

14. National Center for Health Statistics. National Health Interview Survey: Questionnaires, datasets, and related documentation 1997 to the present. Available at: http:// www.cdc.gov/nchs/nhis/quest_data_related_1997_forward. htm. Accessed March 8, 2011.

15. National Center for Health Statistics. Multiple cause of death data file. Centers for Disease Control and Prevention Web site. Available at: http://www.cdc.gov/nchs. Accessed March 8, 2011.

16. Martin JA, Hamilton BE, Sutton PD, et al. Births: final data for 2006. *Natl Vital Stat Rep.* 2009;57(7).

17. Smoking-Attributable Mortality, Morbidity, and Economic Costs (SAMMEC) [computer program]. Available at: http://apps.nccd.cdc.gov/sammec/index.asp. Accessed March 9, 2011.

 Lilienfeld DE, Stolley PD. Foundations of Epidemiology. 3rd ed. New York: Oxford University Press; 1994.

19. Gan Q, Smith K, Hammond S, Hu T- W. Disease burden of adult lung cancer and ischaemic heart disease from passive tobacco smoking in China. *Tob Control.* 2007;16(6):417–422.

20. Thun MJ, Myers DG, Day-Lally C, et al. Age and the exposure-response relationships between cigarette smoking and premature death in Cancer Prevention Study II. In: *Changes in Cigarette Related Disease Risks and Their Implication for Prevention and Control, Smoking and Tobacco Control.* Monograph No. 8. NIH Publication No. 97-4213. Bethesda, MD: National Cancer Institute; 1997: 383–413.

21. Arias E. 2006 United States life tables by Hispanic origin. *Vital and Health Statistics*. 2010;2(152).

22. Max W, Rice DP, Sung H-Y, Michel M. Valuing human life: estimating the present value of lifetime

earnings 2003. eScholarship Web site. Available at: http://repositories.cdlib.org/ctcre/esarm/PVLE2000. Accessed March 8, 2011.

23. US Census Bureau. *Statistical Abstract of the United States: 2011.* 130th ed. Washington, DC: US Government Printing Office; 2010.

24. Heron M, Hoyert DL, Murphy SL, Xu J, Kochanek KD, Tejada-Vera B. Deaths: final data for 2006. *National Vital Statistics Reports.* 2009;57(14):Table 10.

 American Lung Association. Trends in lung cancer morbidity and mortality. 2010. Available at: http:// www.lungusa.org/finding-cures/our-research/trendreports/lc-trend-report.pdf. Accessed January 27, 2012.

 Jefferis BJ, Lawlor DA, Ebrahim S, et al. Cotinineassessed second-hand smoke exposure and risk of cardiovascular disease in older adults. *Heart.* 2010;96(11): 854–859.

27. Giovino GA, Chaloupka FJ, Hartman AM, et al. Cigarette Smoking Prevalence and Policies in the 50 States: An Era of Change–the Robert Wood Johnson Foundation ImpacTeen Tobacco Chart Book. Buffalo, NY: University at Buffalo, State University of New York; 2009.

 Zhang X, Martinez-Donate A, Kuo D, Jones N, Palmersheim K. Trends in home smoking bans in the USA, 1995-2007: prevalence, discrepancies and disparities. *Tob Control.* 2012;21(3):330–336.

29. Mills AL, White MM, Pierce JP, Messer K. Home smoking bans among U.S. households with children and smokers. Opportunities for intervention. *Am J Prev Med.* 2011;41(6):559–\65.

30. Cheng K-W, Glantz SA, Lightwood JM. Association between smokefree laws and voluntary smokefree-home rules. *Am J Prev Med.* 2011;41(6):566–572.

 Tan CE, Glantz SA. Meta-Analysis of the Effects of Smokefree Laws on Cardiac, Cerebrovascular, and Respiratory Hospitalizations. San Francisco, CA: Center for Tobacco Control Research and Education, University of California; 2012.