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Coronary Heart Disease Attributable to Passive Smoking:

CHD Policy Model

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Abstract

Background—Passive smoking is a major risk factor for coronary heart disease (CHD), and existing estimates are out of date due to recent and substantial changes in the level of exposure.

Objective—To estimate the annual clinical burden and cost of CHD treatment attributable to passive smoking.

Outcome measures—Annual attributable CHD deaths, myocardial infarctions (MI), total CHD events, and the direct cost of CHD treatment.

Methods—A Monte Carlo simulation estimated the CHD events and costs as a function of the prevalence of CHD risk factors, including passive-smoking prevalence and a low (1.26) and high (1.65) relative risk of CHD due to passive smoking. Estimates were calculated using the CHD Policy Model, calibrated to reproduce key CHD outcomes in the baseline Year 2000 in the U.S.

Results—At 1999–2004 levels, passive smoking caused 21,800 (SE=2400) to 75,100 (SE=8000) CHD deaths and 38,100 (SE=4300) to 128,900 (SE=14,000) MIs annually, with a yearly CHD treatment cost of \$1.8 (SE=\$0.2) to \$6.0 (SE=\$0.7) billion. If recent trends in the reduction in the prevalence of passive smoking continue from 2000 to 2008, the burden would be reduced by approximately 25%–30%.

Conclusions—Passive smoking remains a substantial clinical and economic burden in the U.S.

Introduction

Evidence that passive cigarette smoking is a risk factor for coronary heart disease (CHD) has existed for more than 20 years.^{1,2} Current estimates of the clinical and economic burden of passive smoking for the entire U.S. population^{3,4} are based mostly on estimates that are from 10 to 15 years ago.⁵ The prevalence of active and passive smoking has changed over the last decade, as has the understanding of the clinical significance of low exposure levels.^{2,6,7} The objectives of this study were to estimate the current population prevalence of

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passive smoking, the annual clinical and cost burden of CHD in the U.S. attributable to passive smoking, and the main sources of uncertainty in these estimates.

Methods

The Coronary Heart Disease Policy Model

The CHD Policy Model is a computer simulation of CHD incidence, prevalence, mortality, and costs in the U.S. population aged >35 years.^{8–10} The demographic–epidemiologic submodel uses a multivariate logistic function to predict CHD incidence and non-CHD mortality among subjects without CHD, stratified by age, gender, and up to six additional categorized risk factors: diastolic blood pressure (<85, 85–90, >90 mmHg); smoking status (number of cigarettes per day among active smokers, nonsmoker with exposure to passive smoking, nonsmoker without exposure); high-density lipoprotein (HDL) cholesterol (<35, 35–49, >49 milligrams per deciliter [mg/dL]), low-density lipoprotein (LDL) cholesterol (<100, 100–130, >130 mg/dL); BMI (<25, 25–29, >29 kilograms per meter squared [kg/m²]); and diabetes mellitus (yes or no). After CHD develops, the bridge submodel characterizes the initial CHD event (cardiac arrest, myocardial infarction [MI], or angina) and its sequelae for 30 days. Then, the disease-history submodel, a state-transition (Markov cohort) model, predicts subsequent CHD events, revascularization procedures, CHD mortality, and non-CHD mortality among patients with CHD, stratified by age, gender, and history of events. Each state and event has an annual cost and quality-of-life adjustment. All population distributions, risk-factor levels, coefficients, event rates, case-fatality rates, costs, and quality-of-life adjustments can be modified.

Data Sources

The new version of the model includes data from prior versions^{8–10} and updates. The 2000 U.S. Census¹¹ provides the baseline population and number of people aged 35 years for the years 2000–2050.¹² CHD deaths were estimated using the ICD-10 codes I20–I25 (ischemic heart disease)¹³ as well as two thirds of ICD codes I49 (other cardiac arrhythmias); I50 (heart failure); and I51 (complications and ill-defined descriptions of heart disease).^{14,15} Other deaths were considered non-CHD.

Incident-CHD cases (cardiac arrest, MI, or angina) and non-CHD deaths in each risk-factor cell for the at-risk U.S. population were determined by risk functions (r) incorporating age- and gender-specific parameters (α) and risk factor–specific β s $\{\beta_k, k = 1, 2, 3, \dots, 6\}$, which are constant over the time span of a simulation, and cell-specific risk-factor means $\{m_k, k = 1, 2, 3, \dots, 6\}$, which are altered by user-defined intervention:

$$r = \frac{\exp\left(\alpha + \sum_{k=1}^k \beta_k m_k\right)}{1 + \exp\left(\alpha + \sum_{k=1}^k \beta_k m_k\right)} \quad [1]$$

β coefficients for CHD risk were determined for a person aged 60 years, the average age of the first onset of CHD in individuals in Examinations 9–13, 24, and 25 from the original Framingham Heart Study cohort, and Examinations 1–6 from the Framingham Offspring Study cohort, for which adequate data were available on LDL and HDL cholesterol for a time-dependent logistic regression analysis.¹⁶ For LDL cholesterol, the slope of the β coefficient with age was determined from the interaction between LDL cholesterol and age, after adjusting for age and LDL cholesterol. For the other risk factors, the gender-specific slopes with age were calculated from a large pooled analysis of multiple epidemiologic studies that reported β coefficients for these risk factors, but not for LDL cholesterol (S Coady, National Heart Lung and Blood Institute [NHLBI], personal communication, 2006).

For diastolic blood pressure, the slope was adjusted for the age- and gender-specific average ratio of diastolic to systolic blood pressure. Transitions from one risk-factor level to another were included to preserve the National Health and Nutrition Evaluation Survey (NHANES) percentages of the population with each risk-factor level.

The MI and cardiac arrest components of incidence were constrained to match estimates from Olmsted County MN¹⁷ (VL Roger, Mayo Clinic, personal communication, 2002). U.S. risk-factor values were based on conditional distributions from the 2002 NHANES.¹⁸

Cardiovascular Disease: Events and Prevalence

Risk factors were assumed to affect the incidence of cardiac arrest, MI, and angina in proportion to overall incidence. Passive smoking was assumed to have a higher relative risk (RR) for MI and cardiac arrest because of the more-intense acute vascular effects of exposure to passive smoke.^{6,19} To retain the overall impact of smoking from the Framingham estimates, a proportionately lower coefficient was assumed for angina. Passive smoking was assumed to carry an RR of 1.26 for MI and cardiac arrest compared with non-exposed nonsmokers²⁰ but not to influence angina. RRs for active smoking were adjusted to be consistent with the referent group of nonsmokers not exposed to passive smoking by dividing by the average RR of all nonsmokers. The model calibrates the background risk of CHD to be consistent with estimated prevalences of the risk factors and their respective RRs. The Framingham risk coefficients have been useful across many populations,^{21–24} and this study's model closely replicates the reduction in coronary events with statins.²⁵

The number of MIs was obtained from discharges coded as ICD-9 code 410 in the National Hospital Discharge Survey (NHDS),²⁶ adjusted for likely miscoding²⁷ such as patients who were discharged alive after 2 days or fewer without a percutaneous coronary intervention and transfer patients. Case-fatality rates and rates of MI in subgroups were estimated from national data²⁶ and other studies.^{28–30} Prehospital cardiac arrest deaths were estimated from the National Vital Statistics System,³¹ and out-of-hospital cardiac arrests surviving to hospital discharge were estimated from national²⁶ and observational studies.³² Subsequent survival was estimated using data on the ratio of in-hospital survival to 30-day survival³³ and data from Medicare and Seattle WA.^{34,35} The levels of coronary revascularization were estimated from the NHDS,³⁶ with mortalities estimated from historical data.^{36,37}

The background prevalence of CHD in 2000 was estimated from the Year 2000 National Health Interview Survey (NHIS),³⁸ assuming that the imperfect positive predictive value of survey data is offset by its imperfect sensitivity.^{39,40} The background prevalence of prior revascularization was estimated from revascularizations before 2000 and estimated survival after revascularization.^{36,41,42}

Healthcare Costs

Total healthcare costs were estimated in Year-2000 dollars by the method of Hodgson and Cohen,^{43,44} using the Medical Expenditure Panel Survey to estimate the proportion of expenditures due to CHD and applying that estimate to aggregate national data.⁴⁵ The inpatient-CHD-cost component was estimated using California data⁴⁶ and deflated using cost-to-charge ratios⁴⁷ and the ratio of the U.S. national average costs to the California average.⁴⁸

Passive Smoking

Four parameters that determine the burden of passive smoking were modeled under several assumptions. First, two criteria were used to determine exposure to passive smoking: serum cotinine 0.05 nanograms per milliliter (ng/mL) and 0.10 ng/mL as measured in

NHANES.⁴⁹ Sensitivity analyses using common serum cotinine cut-off points for current smoking revealed little difference in the estimated population prevalence of passive smoking in nonsmokers, a result that is consistent with previous research.⁷ Active cigarette smokers are also passive smokers, but current expert opinion suggests that active smokers do not suffer any measurable excess risk due to exposure to passive smoking.⁵⁰ Therefore, only nonsmokers were assumed to be liable to exposure to passive smoking.

Second, two different risk-factor levels were used to estimate the burden of passive smoking. The lower risk level used an average RR of exposure of 1.26, and the higher level used an RR of 1.65. These two levels approximate the lower and upper bounds for RRs in observational studies.^{2,51–54} Comparison of the distribution of serum cotinine levels in the exposed population in the U.S.^{18,49} to the study population in Whincup et al.⁵⁴ and Jarvis et al.⁵³ suggests, but does not conclude, that the lower RR is more appropriate for the U.S.

Third, different scenarios were used for the prevalence of active and passive smoking. The status-quo scenario assumed that active and passive smoking remains at levels existing in 1999 to 2004. The trend scenario assumed that trends (by age, gender, and the two serum cotinine exposure cut-off criteria) in the reduction in passive smoking observed from 1988 to 2006 will continue to 2008. For the trend scenario, annual estimates of prevalences and variances by age group and gender for active smoking from NHIS data for the Years 2000–2006³⁸ were used to estimate future prevalence, using a log linear regression model, weighted by the inverse of the variance of estimated smoking prevalence.

Fourth, three alternatives were considered for the temporal decline in excess risk for CHD among passive smokers following the elimination of passive smoking: an instantaneous reduction, a gradual reduction with residual risk, and a gradual reduction without residual excess risk. The first alternative estimated the total burden of passive smoking. Gradual reduction with residual risk assumed that excess risk followed the same time path as in active smokers following cessation—an exponential (53% per year) decline that eliminates 88% of the excess risk after 7 years, with 12% excess risk remaining permanently.⁵⁵ Gradual reduction to no residual risk assumed the same exponential rate of decline, ultimately to no permanent excess risk.

Source of exposure, which was estimated using pooled NHANES survey data for 1999 to 2004, was categorized using self-reported home and work exposure. Because small sample sizes did not provide stable estimates for each age group, gender, and source of exposure,⁵⁶ the demographic groups were combined with all out-of-home exposure.

The effects over 30 years, from 2008 to 2037, were analyzed to estimate the impact of eliminating all passive smoking, holding active smoking constant. Annual events were estimated by age- and gender-specific exposures and aggregated by year. The use of average annual differences over 30 years smoothed the annual differences to provide a medium-term estimate of burden. Changes in the annual estimates of burden over 30 years are relatively small compared to the estimated mean over that period.

In the base-case analyses, active smoking status was determined by self-report and a serum cotinine cut-off of >0.14 ng/mL to account for false reports of nonsmoking status. Sensitivity analyses used self-report or a serum cotinine cut-off of >0.10 ng/mL, although previous analyses suggest that results should be insensitive to this alternative.^{7,54} Using serum cotinine-validated active-smoking status limited the survey sample used in the analysis to those who participated in the examination component of NHANES; therefore, the examination weights were used for estimates of the national prevalence.

Monte Carlo Simulation

Monte Carlo simulations estimated the effect of alternative estimates of the prevalence of passive smoking and the risk-function coefficients (Equation [1]). All the variables were considered independently because the correlations among the risk coefficients were all 0.14 in absolute value. Each simulation consisted of 300 trials. Variations in means and SEs among trials were <0.2% and 2%, respectively, and the distributions of simulation results were identical to those using 1000 trials. Reported results are the mean and SEs from Monte Carlo simulations.

Results

Prevalence of Active and Passive Smoking

Overall, the population prevalence of current active smoking for the status-quo scenario was estimated to be 0.253 for men and 0.213 for women (Table 1 and Figure 1). The population prevalence of passive smoking was estimated to be 0.353 for men and 0.322 for women for the 0.05 ng/mL cut-off, and 0.242 for men and 0.210 for women for the 0.10 ng/mL cut-off. The best-fitting model for estimating the trend scenario for active smoking predicted a linear decline of 0.19 percentage points per year through the end of 2008 with no age or gender interaction. Unweighted or weighted (using inverse of variance of estimated prevalence) passive-smoking prevalence regression estimates produced essentially the same results: an estimated annual reduction of 0.008 percentage points per year for both the 0.05 ng/mL and 0.10 ng/mL criteria. The forecasted prevalence of current active smoking for 2008 in the trend scenario was 0.218 for men and 0.179 for women (Figure 1). The forecast prevalence of passive smoking for the end of 2007 was 0.263 for men and 0.229 for women for the 0.05 ng/mL cut-off, and 0.160 for men and 0.126 for women for the 0.10 ng/mL cut-off.

Source of Exposure

At the 0.05 and 0.10 ng/mL cut-off levels for exposure, 13% of exposed nonsmokers reported exposure only at work and 18% at home or at both work and home. These self-reported prevalences of exposure in 2000 to 2004 were close to the corresponding prevalences for 1988 to 1994.

Burden of Passive Smoking

In the status-quo scenario using an RR of 1.26 for both the 0.05 ng/mL and 0.10 ng/mL criteria, passive smoking caused an estimated 21,800 (SE=2400) to 34,100 (SE=3700) CHD deaths; 21,300 (SE=2400) to 32,700 (SE=3600) first MIs; 38,100 (SE=4300) to 58,400 (SE=6400) total MIs; 45,800 (SE=5100) to 70,400 (SE=7700) CHD events; and \$1.8 (SE=0.2) to \$2.7 (SE=0.3) billion in CHD treatment costs annually (Table 2). In the trend scenario, passive smoking caused an estimated 15,200 (SE=800) to 25,900 (SE=1000) CHD deaths; 14,700 (SE=800) to 24,900 (SE=900) first MIs; 26,300 (SE=1300) to 44,400 (SE=1600) total MIs; 31,600 (SE=1600) to 53,600 (SE=1900) total CHD events; and \$1.2 (SE=0.1) to \$2.1 (SE=0.1) billion in CHD treatment costs annually (Table 2). If the trends in the decline of passive smoking from 1988 to 2004 continued through the end of 2007, then approximately 25% of the estimated status-quo-scenario burden has been eliminated for the 0.05 ng/mL criterion and 30% for the 0.10 ng/mL criterion. The estimated burden of all clinical outcomes and costs increases by between 210% and 230%, using the RR level of 1.65: The burden is 75,100 CHD deaths and \$6.0 billion in CHD-treatment costs per year in the status-quo case, using the 0.05 ng/mL cutoff (Table 2).

The actual realized reduction in the burden of passive smoking will be smaller if the reduction in excess risk following the elimination of exposure is gradual rather than

instantaneous, and smaller still if long-term, chronic exposure to passive smoking causes irreversible damage and, consequently, residual excess risk. In the status-quo scenario, assuming gradual reduction with residual excess risk, eliminating passive smoking immediately would prevent an estimated 17,700 (SE=700) to 27,700 (SE=800) CHD deaths; 18,000 (SE=700) to 27,600 (SE=800) first MIs; 31,400 (SE=1200) to 48,400 (SE=1400) total MIs; 37,800 (SE=1500) to 58,400 (SE=1700) total CHD events; and \$1.5 (SE=0.1) to \$2.3 (SE=0.1) billion in CHD treatment costs annually over the next 30 years (Table 3). In the trend scenario, eliminating passive smoking would prevent an estimated 11,900 (SE=600) to 20,900 (SE=800) CHD deaths; 12,300 (SE=700) to 20,900 (SE=800) first MIs; 21,500 (SE=1100) to 36,500 (SE=1300) total MIs; 25,900 (SE=1000) to 44,100 (SE=1600) CHD events; and \$1.0 (SE=0.1) to \$1.7 (SE=0.1) billion in CHD-treatment costs annually over the next 30 years (Table 3). The proportional differences between the status-quo and trend scenarios and between the 1.26 and 1.65 RR levels is approximately the same as for the instantaneous excess-risk assumption.

The estimates of the avoidable burden under the alternative of a gradual reduction with no residual risk were only 8%–10% higher than if there is a gradual reduction with a residual 12% excess risk due to passive smoking that is irreversible. This small difference indicates that a permanent irreversible increase in excess risk due to passive smoking is a relatively minor public health factor compared to the rate of decline in excess risk following the elimination of exposure.

Discussion

This study's estimates of the current prevalence of active and passive smoking indicate that 25% or more of the population aged 35–84 years is exposed to cigarette smoke, even using the higher 0.10 ng/mL cut-off level for serum cotinine. They also indicate that the *Healthy People 2010*⁵⁷ goal of <45% of the population with a serum cotinine level of <0.10 ng/mL has been met, consistent with previous findings.^{7,57} However, almost 40% of men and 30% of women aged 35–84 years remain exposed to some tobacco smoke at the lower threshold for exposure of 0.05 ng/mL.

Based on serum cotinine levels, substantial passive smoking and resulting CHD occur in people with no self-report of exposure. However, regardless of the specific self-reported sources of exposure, the impact of work and public-area smoking restrictions depends crucially on the nature of these unknown sources of exposure. If a considerable percentage of the unknown sources of exposure exists at work or in public areas where work restrictions would apply, then the potential impact of such restrictions on public health would be quite large.

Passive smoking produces substantial clinical and economic burden in the U.S., accounting for an estimated 15,200–75,100 CHD deaths per year and for \$1.2–\$6.0 billion in CHD treatment costs per year. To put these public health consequences in context, the number of deaths attributable to exposure to passive smoking is between 17% and 86% of the approximately 87,000 annual deaths from ischemic heart disease attributable to active smoking.^{3,4} If the reduction in excess risk due to passive smoking is rapid, then the avoidable burden over the next 30 years is equal to the total burden. If the excess risk due to passive smoking declines at the same rate following the elimination of exposure as for active smoking, then the avoidable burden over this time frame is approximately 70%–90% of the total burden. Recent evidence from smoking bans indicates that a relatively rapid reduction in excess risk is the more likely case, and that the avoidable burden approximates the total burden.^{58–60}

Limitations

This analysis has several limitations. Former smokers were not specifically considered because the use of the Framingham data prevented the consistent measurement of former smoking status. These results are conditional on the historical level of former smoking, which has not changed significantly since the early 1970s⁶¹; statistical adjustments used to adjust for differences between the original and offspring cohort in the Framingham data did not reveal any significant differences in the smoking coefficient. If former smoking increases, mainly because of increased quitting, and if never smoking remains constant, then the RRs for current and passive smoking used in the current simulation will be too large. If the population prevalence of former smoking decreases because never smoking increases, then these two RRs will be too small. Such changes in the RRs for current smokers would have a small effect on the results of these analyses, mainly through changes in the equilibrium base incidence rate of CHD that is consistent with the RRs and the prevalence of exposure to the risk factors. Overestimates of the RR of exposure to passive smoking would result in overestimates of the burden, and vice versa.

The main analysis used serum cotinine-validated smoking with an upper-bound criterion for current and active smoking status of 0.14 ng/mL,⁵⁴ but the current study's results were virtually identical when a 0.10 cut-off ng/mL was used.⁷ The use of self-report and the 10 ng/mL serum cotinine cut-off criterion for active current smoking would produce estimates from 10% lower to 5% higher, due to differences in the estimated prevalence of exposure to passive smoking.

Two serum cotinine levels were used to determine passive-smoking exposure. There is strong evidence that passive smoking has clinical effects within this range of exposure,^{6,62} although the higher <0.10 ng/mL cut-off has been chosen as a target for *Healthy People 2010*.⁵⁷ The precise lower bound of clinically significant exposure, if one exists, is unknown.^{54,63} Another limitation is the lack of reliable estimates for each source of exposure to passive smoking for the demographic groupings used in this analysis, due to small sample sizes for most groups in the NHANES.

Estimating the uncertainty in future passive-smoking exposure required the assumption of a fixed prevalence after 2008. Data on the percentage of the population covered by workplace and public-area smoking restrictions provide only crude information on the prevalence of passive smoking. Forecasting a reliable long-run trend of future coverage or its effectiveness is difficult. Recent substantial increases in protection against passive-smoking exposure have occurred because of legislative and regulatory action in large jurisdictions. However, these kinds of actions are inherently difficult to predict, and the resulting degree of protection likely will vary substantially.⁶⁴ More-frequent estimates of exposure to passive smoking from the new NHANES survey design may remedy this problem in the future.

Summary

Exposure to passive smoking has been reduced by 25% to 40%, and its burden has been reduced by between 25% and 30% over the last 8–10 years, but the burden remains substantial. Most of the uncertainty in estimates of the burden is due to uncertainty about the RR associated with the average intensity of exposure in the U.S. population, rather than uncertainty about the appropriate cut-off for measuring the prevalence of exposure, uncertainty about prevalence, or uncertainty about other risk factors for CHD and other chronic diseases. The true prevalence of passive smoking as measured by serum cotinine measurements is substantially greater than would be estimated by self-report. The future burden of passive smoking may be driven mainly by political and legal processes to ban

smoking in public areas and the workplace as well as campaigns to encourage smoke-free homes.

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Figure 1.

Prevalence of active and passive smoking, U.S.

Note: Status-quo scenario: average 2000–2004; trend scenario: projected 2008; in percent

Source: status-quo scenario, National Health and Nutrition Examination Survey; trend scenario, see text.

Table 1

Population prevalences of active and passive smoking, by age and gender

Demographic group	Status-quo scenario (1999-2004)			Trend scenario ^a (forecast for 2008)		
	Active smoking	Passive smoking (0.05 ng/mL)	Passive smoking (0.10 ng/mL)	Active smoking	Passive smoking (0.05 ng/mL)	Passive smoking (0.10 ng/mL)
Total aged 25-84 years	0.248	0.337	0.229	0.214	0.253	0.150
Total aged 35-84 years	0.232	0.337	0.225	0.199	0.247	0.144
Men	0.295	0.350	0.247	0.237	0.270	0.167
25-34	0.337	0.341	0.263	0.306	0.292	0.189
35-44	0.322	0.357	0.250	0.292	0.253	0.150
45-54	0.247	0.342	0.222	0.224	0.265	0.162
55-64	0.217	0.362	0.275	0.197	0.273	0.170
65-74	0.153	0.374	0.228	0.139	0.277	0.174
75-84	0.114	0.303	0.203	0.103	0.245	0.141
Women	0.238	0.324	0.212	0.189	0.235	0.132
25-34	0.253	0.332	0.221	0.230	0.258	0.154
35-44	0.255	0.312	0.228	0.231	0.219	0.115
45-54	0.245	0.327	0.186	0.222	0.231	0.136
55-64	0.176	0.312	0.226	0.160	0.239	0.127
65-74	0.125	0.367	0.233	0.113	0.243	0.139
75-84	0.050	0.290	0.142	0.046	0.210	0.107
Men aged 35-84 years	0.253	0.353	0.242	0.218	0.263	0.160
Women aged 35-84 years	0.213	0.322	0.210	0.179	0.229	0.126

Note: Pooled National Health and Nutrition Evaluation Survey estimates for 1999-2004 used for passive smoking
ng/mL, nanograms/milliliter

^a Combined age and gender category prevalences calculated using projected U.S. resident population for Year 2008

Table 2

Annual burden of CHD due to passive smoking, 2008-2037

Exposure criterion: serum cotinine (ng/mL)	Status quo 2001		Trend case 2008	
	0.05	0.10	0.05	0.10
RR of CHD due to passive smoking: 1.26				
Deaths				
M	34,100	21,800	25,900	15,200
SE	3,700	2,400	1,000	800
Total MIs				
M	58,400	38,100	44,400	26,300
SE	6,400	4,300	1,600	1,300
First MIs				
M	32,700	21,300	24,900	14,700
SE	3,600	2,400	900	800
Total CHD events				
M	70,400	45,800	53,600	31,600
SE	7,700	5,100	1,900	1,600
CHD treatment cost (million \$₂₀₀₀)				
M	2,700	1,800	2,100	1,200
SE	300	200	100	100
RR of CHD due to passive smoking: 1.65				
Deaths				
M	75,100	48,900	56,900	33,500
SE	8,000	5,400	6,200	3,900
Total MIs				
M	128,900	85,500	97,700	57,800
SE	14,000	9,500	10,800	6,700
First MIs				
M	73,400	48,500	55,700	32,700
SE	8,100	5,500	6,300	3,900
Total CHD events				
M	154,300	101,500	116,900	68,700
SE	16,800	11,300	12,900	8,000
CHD treatment cost (million \$₂₀₀₀)				
M	6,000	4,000	4,600	2,700
SE	700	400	500	300

CHD, coronary heart disease; MI, myocardial infarction; ng/mL, nanograms/milliliter; RR, relative risk

Table 3

Annual avoidable burden of CHD due to passive smoking, 2008-2037

Exposure criterion: serum cotinine (ng/mL)	Status quo 2001		Trend case 2008	
	0.05	0.10	0.05	0.10
RR of CHD due to passive smoking: 1.26				
Deaths				
M	27,700	17,700	20,900	11,900
SE	800	700	800	600
Total MIs				
M	48,400	31,400	36,500	21,500
SE	1,400	1,200	1,300	1,100
First MIs				
M	27,600	18,000	20,900	12,300
SE	800	700	800	700
Total CHD events				
M	58,400	37,800	44,100	25,900
SE	1,700	1,500	1,600	1,000
CHD treatment cost (million \$₂₀₀₀)				
Mean	2,300	1,500	1,700	1,000
SE	100	100	100	100
RR of CHD due to passive smoking: 1.65				
Deaths				
M	59,300	38,100	44,600	25,800
SE	1,800	1,500	1,700	1,400
Total MIs				
M	105,100	69,100	79,300	46,300
SE	3,400	2,800	3,000	2,500
First MIs				
M	61,800	40,700	46,700	27,300
SE	2,000	1,600	1,800	1,500
Total CHD events				
M	126,000	82,100	95,000	55,100
SE	4,000	3,300	3,600	2,900
CHD treatment cost (million \$2000)				
M	5,000	3,300	3,700	2,200
SE	200	100	100	100

CHD, coronary heart disease; MI, myocardial infarction; ng/mL, nanograms/milliliter; RR, relative risk