Epidemiology and Prevention

Declines in Acute Myocardial Infarction After Smoke-Free Laws and Individual Risk Attributable to Secondhand Smoke

James M. Lightwood, PhD; Stanton A. Glantz, PhD

Background—The estimated effects of recent public and workplace smoking restriction laws suggest that they produce significant declines in community rates of heart attack. The consistency of these declines with existing estimates of the relative risk of heart attack in individuals attributable to passive smoking exposure is poorly understood. The objective is to determine the consistency of estimates of reductions in community rates of heart attacks resulting from smoking restriction laws with estimates of the relative risk of heart disease in individuals exposed to passive smoking.

Methods and Results—Meta-analyses of existing estimates of declines in community rates were compared with a mathematical model of the relationship between individual risk and community rates. The outcome measure is the ratio of community rates of acute myocardial infarction (after divided by before implementation of a smoking restriction law). There is a significant drop in the rate of acute myocardial infarction hospital admissions associated with the implementation of strong smoke-free legislation. The primary reason for heterogeneity in results of different studies is the duration of follow-up after adoption of the law. The pooled random-effects estimate of the rate of acute myocardial infarction hospitalization 12 months after implementation of the law is 0.83 (95% confidence interval, 0.80 to 0.87), and this benefit grows with time. This drop in admissions is consistent with a range of plausible individual risk and exposure scenarios.

Conclusion—Passage of strong smoke-free legislation produces rapid and substantial benefits in terms of reduced acute myocardial infarctions, and these benefits grow with time. (Circulation. 2009;120:1373-1379.)

Key Words: epidemiology ■ meta-analysis ■ myocardial infarction ■ prevention ■ smoking ■ tobacco smoke pollution

Passive smoking increases the risk of coronary heart disease and acute myocardial infarction (AMI) in non-smokers,1–3 which has led to the adoption of many laws making public areas and workplaces 100% smoke free. The risk of AMI falls rapidly after smoking cessation,4 and the effects of secondhand smoke (SHS) on many biological mediators that lead to heart disease occur rapidly and are nearly as large as those of smoking.5–10 It is reasonable to expect that implementing strong smoke-free laws would lead to a reduction in AMI. Several such laws in North America and Europe have been studied as natural experiments to estimate the reduction in community AMI risk,11–24 with reductions ranging from 11% (Italy12 and Ireland13) to 40% (Montana22). These reductions seemed large compared with early estimates of an ∼30% increase in individual risk of heart disease associated with chronic SHS exposure.5,25 These estimates may underestimate the actual individual risks associated with passive smoking because of a downward bias resulting from exposure misclassification; Whincup et al26 reported substantially higher risks of major coronary heart disease when stratifying risk based on levels of cotinine (a biomarker for SHS) than self-report, which should give a more accurate risk estimate because of more accurate SHS exposure assessment. Here, we update previously published meta-analyses of community risk27,28 (that considered only the first 4 and 8 studies, respectively) by adding more studies and accounting for the length of follow-up. The present analysis demonstrates that the community risk reduction associated with smoke-free laws grows with time and is consistent with a wide range of actual observed individual risk and exposure scenarios.

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Methods

This study uses a simple random-effects meta-analysis and a meta-regression to estimate the reduction in the community rate of heart...
attack as a function of time since smoke-free laws went into effect. Simulation estimates of the reduction in community rates of heart attack calculated as a function of individual AMI risk resulting from exposure to passive smoking produce estimates consistent with the observed reductions in community risk.

Study Identification
The first article reporting a drop in AMI after implementation of a comprehensive smoke-free law (in Helena, Mont) was published in 2004. We used PubMed, Science Citation Index, and Social Science Citation Index to locate 9 articles published since then. In addition, we identified 3 studies (Ireland, Massachusetts, and France) reported at meetings. One city (Pueblo, Colo) was used to publish 2 reports: 1 on results through the first 18 months after the law went into effect and 2 after 36 months. Data from Piedmont, Italy, were used as part of 1 study 2 months after the law went into effect (24) and another of Piedmont alone after 6 months. Although there was some overlap in the data used in Pueblo (reuse of the same baseline data) and Italy (Piedmont represented 25% of the total cases in the 2-month study), we treated these 4 studies as independent observations. Table I in the online-only Data Supplement provides detailed descriptions of all the studies and available data on changes in SHS exposure.

Meta-Analysis
The studies were combined using a random-effects meta-analysis. The expected relative reduction in community rates of AMI as a function of follow-up period was estimated from

$$\ln RR_{AMI,j} = \beta_0 + \beta_1 T_j + \varepsilon_j,$$

where $RR_{AMI,j}$ is the relative reduction in community rate of AMI in study $j$, $T_j$ is the follow-up period after adoption of the smoking law in study $j$, $\varepsilon_j$ is the error term, and $\beta_0$ and $\beta_1$ are regression parameters. See Table 1 of the online-only Data Supplement for detailed descriptions of all the studies found. Calculations were done with Stata 9.0 procedures metan, metareg, and metabias. We considered 4 distributions of individual relative risks. The low risk of AMI associated with passive smoking, $p_{s,c}$ and current active smoking, $p_{a,c}$, are population prevalences. The population prevalence of passive smoking exposure, $p_{a,p}$, is lower than the corresponding prevalence in the nonsmoking population, which is $p_{a,p}(1-p_{c})$.

The parameter estimates are average values for 1 year before and after the law. The proportion of current active smokers quitting as a result of a smoking law, $p_{q}$, and relative risk of current active smokers who quit as a result of the law are assumed to be the average values over 6 to 18 months after the law.

Model Parameters and Distributions
Risk of AMI Associated With Passive Smoking
We consider 4 distributions of individual relative risks. The low estimate of 1.31 (95% confidence interval [CI], 1.21 to 1.41), from the Barnoya and Glantz meta-analysis of 29 long-term studies of the risk of AMI and other cardiovascular disease outcomes, is based with 1 exception on self-reported SHS, which tends to underestimate total exposure. These studies likely underestimate individual risk associated with SHS because people considered in the unexposed reference (the denominator) in the risk estimate should have been in the exposed group (the numerator).

Whincup et al published the only longitudinal study in which subjects were classified on the basis of cotinine levels at baseline, with the reference group consisting of subjects with serum cotinine $\leq 0.7$ ng/mL. This cutoff is high enough to include both unexposed nonsmokers and light passive smokers. (This decision may have been necessary because, in England during the 1978 to 1980 period, at baseline almost everyone had some detectable exposure to SHS.) Not surprisingly, Whincup et al found higher risks than the studies based on self-report.

Substituting Equation 3 into Equation 2 and dividing Equation 1 by that result yields the ratio of community rates of AMI after compared with before the smoking law, $RR_{AMI}$:

$$RR_{AMI} = \frac{r_s}{r_b} = \frac{\left[1 - (p_{a,p} - p_{a,c} - p_q) + p_{a,c}R_c + p_{a,c}(D(R_p - 1) + 1) + p_{q}R_q\right]}{\left[1 - (p_{a,p} - p_{a,c}) + p_{a,c}R_c + p_{a,c}R_q\right]}.$$

The base rate of AMI resulting from other causes, $i$, is assumed to be constant and thus cancels out in Equation 4.

Three Exposure Scenarios
The community effects of reduced SHS exposure were estimated for 3 cases to describe the range of prevalence of passive smoking exposure before and after a smoking restriction law, each based on cotinine-validated population data. We considered a person unexposed if his or her level of serum cotinine was below the limit of detection (0.05 to 0.10 ng/mL). We estimated $D$, the reduction in SHS exposure among people who continued to be exposed as the ratio of geometric mean cotinine after the law to geometric mean cotinine before the law.

The prevalence of passive, $p_{s,c}$, and current active smoking, $p_{a,c}$, are population prevalences. The population prevalence of passive smoking exposure, $p_{a,p}$, is lower than the corresponding prevalence in the nonsmoking population, which is $p_{a,p}(1-p_{c})$.
The third case (large drop in exposure) is based on cotinine measured in 1999 to 2002 in the large US National Heath and Nutrition Examination Study (NHANES) among nonsmokers ≥20 years of age,\(^3\) which represents the average experience of US communities with and without smoking laws. Forty-six percent of nonsmokers living in jurisdictions without a law had detectable cotinine (≥0.05 ng/mL) compared with 13% living in jurisdictions covered by strong laws, a 33% difference in prevalence. The ratio of the geometric mean cotinine with laws compared with those without the law was 0.19.

The second case (moderate drop in exposure from moderate base) is based on the New York State population with detectable cotinine (≥0.05 ng/mL) before and after the strong smoking restriction law went into effect.\(^8\) The survey sample excluded New York City and Nassau County, which had already passed strong smoking restriction legislation. The sample, however, did include 3 counties (Dutchess, Suffolk, and Westchester) that had strong local smoke-free laws in place, representing 27% of the sample (personal communication, American Nonsmokers’ Rights Foundation, January 27, 2009). Including these counties in the sample probably reduced the variance of RR\(_{AMI}\) and produce conservative results.) The geometric mean cotinine after the law was 0.61 of that before the law.

The first case (large drop in exposure) is based on cotinine measured in 1999 to 2002 in the large US National Heath and Nutrition Examination Study (NHANES) among nonsmokers ≥20 years of age,\(^3\) which represents the average experience of US communities with and without smoking laws. Forty-six percent of nonsmokers living in jurisdictions without a law had detectable cotinine (≥0.05 ng/mL) compared with 13% living in jurisdictions covered by strong laws, a 33% difference in prevalence. The ratio of the geometric mean cotinine with laws compared with those without the law was 0.19.

The simulation estimates of the ratio of community rates of AMI before divided by after the law, RR\(_{AMI}\), were calculated for 48 combinations of parameters: the passive smoking exposure scenarios (3 cases), individual risk for AMI associated with passive smoking (4 cases), presence or absence of a dose-response effect among people still exposed to SHS after the law took effect (2 cases), and presence

### Table. Input Parameters for Simulation

<table>
<thead>
<tr>
<th>Parameters Varying With Scenarios</th>
<th>Mean (95% Cl)</th>
<th>Distribution</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR resulting from passive smoking, R(_p)</td>
<td>Low estimate</td>
<td>1.31 (1.21, 1.41)</td>
<td>lognormal</td>
</tr>
<tr>
<td>Median level of exposure from Whicup et al</td>
<td>1.49 (1.03, 2.15)</td>
<td>lognormal</td>
<td>Whicup et al(^36)</td>
</tr>
<tr>
<td>5–9 y of follow-up</td>
<td>1.95 (1.09, 3.48)</td>
<td>lognormal</td>
<td>Whicup et al(^36)</td>
</tr>
<tr>
<td>0–4 y of follow-up</td>
<td>3.73 (1.32, 10.56)</td>
<td>lognormal</td>
<td>Whicup et al(^36)</td>
</tr>
<tr>
<td>Exposure scenarios: prevalence of passive smoking among nonsmokers, US average case</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large drop in exposure</td>
<td>Before smoking, p(_{p0})</td>
<td>0.46 (0.42–0.49)</td>
<td>Normal</td>
</tr>
<tr>
<td>After smoking, p(_{pA})</td>
<td>0.13 (0.08–0.17)</td>
<td>Normal</td>
<td>Pickett et al(^31)</td>
</tr>
<tr>
<td>Ratio of geometric mean in serum cotinine after the law to before the law, D</td>
<td>0.19 (0.10–0.32)</td>
<td>Lognormal</td>
<td>Pickett et al(^31)</td>
</tr>
<tr>
<td>Moderate drop in exposure from moderate base</td>
<td>Before smoking, p(_{p0})</td>
<td>0.68 (0.57–0.78)</td>
<td>Normal</td>
</tr>
<tr>
<td>After smoking, p(_{pA})</td>
<td>0.52 (0.47–0.58)</td>
<td>Normal</td>
<td>Bauer et al(^30)</td>
</tr>
<tr>
<td>Ratio of geometric mean in serum cotinine after the law to before the law, D</td>
<td>0.54 (0.36–0.78)</td>
<td>Lognormal</td>
<td>Bauer et al(^30)</td>
</tr>
<tr>
<td>Moderate drop in exposure from high base</td>
<td>Before smoking, p(_{p0})</td>
<td>0.89 (….)</td>
<td>Constant</td>
</tr>
<tr>
<td>After smoking, p(_{pA})</td>
<td>0.72 (….)</td>
<td>Constant</td>
<td>Haw and Gruer(^32)</td>
</tr>
<tr>
<td>Ratio of geometric mean in serum cotinine after the law to before the law, D</td>
<td>0.61 (0.52–0.70)</td>
<td>Lognormal</td>
<td>Haw and Gruer(^32)</td>
</tr>
</tbody>
</table>

**Parameters constant over all scenarios:** reduction in proportion of current direct smokers quitting as a result of the law, p\(_q\)

| Absent | 0 | Constant |
| Present | 0.038 (0.029–0.047) | Normal | Fichtenberg and Glantz\(^27\) |

### Parameters Common to All Scenarios

- RR resulting from current active smoking, R\(_c\)
- RR resulting from quitting active smoking, R\(_q\)
- Prevalence of current smoking before law, p\(_c\)

**RR indicates relative risk. The Appendix can be found in the online-only Data Supplement.**
or absence of smoking cessation resulting from the law (2 cases).
The combinations of individual relative risk, dose-response, and
smoking cessation (16 cases) were called scenarios and were
grouped by the 3 groups of passive smoking exposure parameters.
Crystal Ball Version 5.238 was used for the estimates using a
Monte Carlo simulations with 20 000 trials. Calculations and details
on sources of the parameters are given in the online-only Data
Supplement.

Sensitivity Analysis
Several sensitivity analyses were used to determine the robustness of
the results to changes in the sample and statistical method. These
analyses include dropping the initial (18-month) estimate from
Pueblo, Colo, and the estimate for Piedmont, Italy, from the Vaselli
et al24 study so that all remaining observations were independent;
adjustment for an insignificant trend in AMI baseline incidence, i, for
estimates in Scotland21 and Piedmont,12 which had not accounted
for possible secular trends in AMI; and use of nonparametric tests for
statistical significance of the slope parameter and alternative
random-effects estimators. Sensitivity analyses for the simulation
included alternative formulas for dose-response in nonsmokers who
remained exposed to passive smoking before and after the smoke-
free law, alternative methods of pooling current smoking prevalence,
and different age adjustments for average relative risk resulting from
current smoking.

Results
Meta-Analysis
We used a random-effects model for the meta-analyses
because of significant heterogeneity in the estimates, which
yielded a pooled RR of 0.81 (95% CI, 0.78 to 0.85; Figure 1). A
funnel plot and Begg test did not suggest publication bias
(Figure 2). Some of the heterogeneity could be due to
differences in end points, the confounding variables consid-
ered, the analytical methods, changes in level of SHS expo-
sure after the law, and duration of follow-up.

There was a significant relationship between the duration
of study follow-up and RRAMI (Figure 3), with ln(RRAMI)
falling by 0.0113/month (SE, 0.002; P = 0.0005). The inter-
cept was not significantly different from 0 (−0.046; SE,
0.037; P = 0.242). Duration of follow-up accounted for 76%
of the between-study variance. The meta-regression between
ln RRAMI and duration of study (Figure 3) provided a good
estimate of changes in risk over time for the observed period.
We used the results of the meta-regression in Figure 3 to
standardize all relative risks and associated upper and lower
bounds of the 95% CIs to the values that would be observed
at 12 months after implementation of each law with

\[
\ln(\text{RR}_{\text{AMI}}) = \ln(\text{RR}_{\text{AMI}}) + 0.0113/\text{month}(T - 12\text{ months})
\]

where T is the duration of study follow-up in months. This
adjustment substantially reduced the variability in the esti-
mated values of RRAMI (Figure 4). The random-effects
meta-analysis of the resulting values adjusted to 12 months
yielded a pooled risk estimate of 0.83 (95% CI, 0.80 to 0.87).
Although lower than before, there was still significant het-
erogeneity, probably reflecting the differences noted above
(other than study duration). Geographic heterogeneity may be
an important factor in remaining heterogeneity; after adjust-
ment, the reductions in AMI risk appear slightly larger in the
United States than in Europe and Canada.

Simulation Results
Eight of the 48 scenarios produced point estimates of RR that
were within the 95% CI for the pooled risk estimate

\[
\text{RR}_{\text{AMI}}\text{(12)} = \text{RR}_{\text{AMI}}\text{(T)} + 0.0113/\text{month}(T - 12\text{ months})
\]
produced by the meta-analysis of the individual studies at 12 months, RRAMI(12) (95% CI, 0.80 to 0.87), and 28 of the 48 scenarios produced interval estimates (defined as the 95% CI) of RRAMI that overlapped the 95% CI for the pooled estimate of individual risks (Figure 5). The scenarios that are most consistent with the pooled community risk ratio at 1 year, RRAMI(12), are those with individual SHS relative risks of 1.95. The presence of a dose-response and effect on quitting were not required to obtain reasonable agreement with the observed population risk. The sensitivity analyses did not produce noticeable changes in the meta-analysis or simulation results.

Discussion

After adjustment for variable length of follow-up, the individual community studies yield remarkably consistent elevations of the community benefits of reduced AMIs after implementation of strong smoke-free legislation (Figure 3), with an \( \approx 15\% \) drop during the first year and continuing exponential declines, reaching \( \approx 36\% \) in 3 years (the limit of currently available data). The fact that the intercept of the risk reduction with time has an intercept of 0 is consistent with the results of the very large study of the effects of a strong smoke-free law in New York State,17 which tested for an immediate offset (change in intercept) and interaction between the presence of a law and time after the law went into effect. The New York study found no immediate shift in the AMI rate immediately after the law went into effect but discovered that the rate of AMI declined continuously with time after the law. Results from Bowling Green, Ohio,18 and Pueblo, Colo,11 also showed that AMI rates fell with time.

Eight simulation scenarios have point estimates closest to the point estimates of the meta-analysis at 1 year (Figure 3) and are within their 95% CIs. All 8 scenarios use an individual relative risk of 1.94 (from Whincup et al26 for 5 to 9 years of follow-up). Six of these scenarios assumed a dose-response relationship (lower SHS exposures among people still exposed after the law) except 1, which used the large drop scenario. Four of the 8 scenarios included smokers quitting as a result of the law.

Richiardi et al39 published a mathematical model based on the assumption that the effect of tobacco smoke exposure was a large immediate and short-term increase in individual AMI risk (relative risk of 4.5 in the first hour of passive smoking) combined with a 50% reduction in SHS exposure that was consistent with a reduction in community-level risk of 5% to 15%, less than has actually been observed. In contrast, we assume the smaller individual risks that have been observed in long-term epidemiological studies. By accounting for the fact that the community risk reductions fall with time after the law (something not in the Richiardi et al analysis) and adjusting the observed risks to those corresponding to 12 months after implementation of 100% smoke-free workplace laws. ES indicates the effect size for the adjusted (to 12 months) relative reduction in community AMI risk.
months of follow-up, our model provides results consistent with the full range of observed risks (Figure 4).

Limitations
The largest shortcoming in the simulation is the lack of biomarker-based levels of general population exposure to SHS before and after these laws went into effect. Most of the available data relate to self-reported levels of SHS exposure, which underestimate exposure.\textsuperscript{40,41} with biomarker data (cotinine) generally limited to restaurant and bar workers. The restaurant and bar worker studies are useful for documenting high compliance with the laws, but biomarker data for population samples would be more useful for evaluating health effects of the law in the entire population. It is important to publish not only the changes in arithmetic and geometric mean cotinine levels but also results that can be used to estimate the fraction of the population with very low levels (<0.05 ng/mL in serum) to estimate changes in prevalence of exposure. Changes in current smoking as a result of smoking laws can noticeably affect the decline in prevalence of exposure. Changes in current smoking as a result of smoking laws can noticeably affect the decline in community rates and should also be measured.

All studies with adequate data were included in the meta-regression to avoid potential bias resulting from selection and adjustment of individual estimates. With 1 exception\textsuperscript{23} (omitted from the meta-analysis because of small sample size), there are no reliable data on smoking status at the time of admission for subjects in the studies. It would be desirable to have cotinine measures on admitted patients to assess both smoking status and level of SHS exposure. Such data would allow estimates of any differential effects of smoke-free laws on nonsmokers and smokers and estimates of the effects of the laws on cessation among people at risk of AMI. Collection and reporting such data would be useful in further refining the linkages level of SHS and risk of AMI.

We did not account for the effect of lower SHS exposure among people who quit smoking after implementation of the law or the fact that continuing smokers reduce consumption when smoke-free policies go into effect;\textsuperscript{37} these omissions mean that our model probably underestimates the effect of the law.

The parameters in our model were treated as independently distributed, even though some may covary such as relative risks of current and all former smokers and correlation of prevalence of current smoking and exposure to passive smoking. The parameter distributions were taken from observational studies and may be subject to associated biases.

Conclusions
Analysis of reductions in community rates of AMI shows that the measured effects of smoking laws are consistent after accounting for the follow-up periods of different studies. Simulation estimates of expected reductions in community rates, based on existing estimates of individual relative risk of AMI resulting from exposure to passive smoking, are consistent with each other. This analysis shows, using data from 5 countries, that passage of strong smoke-free legislation produces rapid and substantial benefits in terms of reduced AMIs and that these benefits grow with time.

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Disclosures
None.

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**CLINICAL PERSPECTIVE**

Smoking restriction laws for public areas and workplaces have become increasingly common in the last decade. A number of studies of these laws have found that they have a significant effect in reducing the community rate of heart attack. The estimated size of the reductions attributable to the smoking restriction laws has varied widely, and their consistency with existing estimates of the individual relative risks attributable to exposure to passive smoke exposure is unknown. This study shows that the results of these studies are consistent if the follow-up periods of the individual studies of community rates of heart attack are taken into account and that the estimated effects of smoking restriction laws on community rates are consistent with individual risks of heart attack attributable to exposure to passive smoking. These results strengthen the case that passive smoking is a serious risk factor for acute coronary heart disease and that its elimination reduces the community rate of heart attack quickly. Physicians should counsel their patients to avoid exposure to passive smoking whenever possible, and all healthcare professionals should strongly advocate more, and stronger, public area and workplace smoking restriction laws.

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Declines in Acute Myocardial Infarction After Smoke-Free Laws and Individual Risk Attributable to Secondhand Smoke
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SUPPLEMENTAL MATERIAL

Relative Risk of Coronary Heart Disease Implied by Smoking Ban Natural Experiments: Online Data Supplement

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Table S-3. Parameter distributions used in subsidiary calculations

Table S-4. Regression parameters and variance-covariance matrix for relative risks of current smoking and quitting.

Table S-5. Calculation of proportion of current smokers who are elderly.

Detailed Documentation of Community Risks Estimates Located.

Table S-1 provides detailed summary descriptions of the all the studies containing estimates of the effect of smoking restriction laws that were located for this study.

Average Smoking Prevalence in Study Sites

The most specific regional estimate available was used; ranging from the national prevalence (Scotland and Ireland) to a specific municipality (Saskatoon, Canada). The overall prevalence was used for Saskatoon, since gender-specific estimates were unavailable. Standard errors were not available for some European countries, and when absent, were calculated by multiplying the country estimate by the relative standard error for the pooled estimate from areas with sufficient data. We used the pooled random effects average for the data in Table S-2.
Risk of AMI in Recent Quitters

Appropriate values for the relative risk of recent quitters, $R_q$, and current smokers, $R_c$, are not available in the literature. The appropriate value of $R_q$ for recent quitters is calculated from existing estimates of the decline in relative risk due to smoking as a function of time since quitting. The average relative risk of current smokers for the community, $R_c$, is calculated from sex and age specific rates, then adjusted for all adult smokers in the community. Both relative risks must also be adjusted for change in referent groups, from never smokers to all non-smokers (Table S-3). These adjustments are explained below. The parameter distributions used and subsidiary calculations are shown in Tables S-2 to S-5.

The relative risks of quitters of current smokers and recent quitters, with never-smokers as the referent group, are modeled using estimates for 18 to 64 year old smokers from Lightwood and Glantz:\(^1\)

$$\ln R^*_{c,s}(n) = \ln\left[\left(\left(R^*_{c,m} + R^*_{c,f}F\right) - \left(R^*_{s,m} + R^*_{s,f}F\right)\right)\exp(-n/\tau) + \left(R^*_{s,m} + R^*_{s,f}F\right)\right]$$  \[S-1\]

where

$R^*_{c,s}(n)$ = relative risk for ex-smokers $n$ months following cessation for sex $x$, using never smokers as the referent group

$R^*_{c,s}$ = relative risk of current smokers of sex $s$, using never smokers as the referent group,
\( R_{s,s}^* \) = asymptotic limit of the relative risk of smokers who of sex \( s \) (\( m = \text{men}, f = \text{women} \)) who have quit for \( n = \) infinity,

\( F \) = indicator variable for \( s = \text{women} \),

\( \tau \) = exponential rate of decay for relative risk expressed in months since cessation,

\( n \) = number of months since smoking cessation (\( n = 0 \) indicates current smoking),

and the asterisk (*) indicates that never-smokers are the reference group.

Note that \( R_{c,s}^*(0) \) is defined as the relative risk of current smokers (that is, “ex-smokers” at zero months following cessation and is equal to the parameter \( R_{c,s}^* \). The notation \( R_{c,s}^*(12) \) is the relative risk for an ex-smoker at after twelve months of cessation.

The distributions of \( R_{c,s}^*(n) \) from equation [S-1] is simulated using the regression parameter coefficients and the Cholesky decomposition of the variance-covariance matrix (Table S-4) because of significant correlation between the parameter estimates.

A constant flow of recent quitters are assumed to quit smoking at the beginning of period \( a \), after the ban, and continues for one year, \( n = 12 \). The effect of recent quitting is modeled by multiplying the cumulative percentage of recent quitters, \( p_q \), by the average relative risk of quitters by the average relative risk of the flow of quitters. A midpoint correction is used to calculate the average relative risk of recent quitters:

\[
R_{q,s}^* = \left[ R_{c,s}^*(0) + R_{c,s}^*(12) \right] / 2 ,
\]

where
\( R_{c,s}^*(0) \) = the relative risk of current smokers (that is “ex-smokers” with zero months cessation).

Equation [S-1] uses never smokers as a reference group for the individual relative risk for current and recent quitters, and the relative risks applies to adults age 18 to 64. The conversion is made to the referent group of all non-smokers and all adult age groups in three steps: combine the sex specific relative risks to overall relative risks for all adults 18 to 64, convert the referent group from never smokers to all non-smokers (never and ex-smokers), and finally, adjust the overall relative risk for 18 to 64 year olds to apply to all adults over age 18.

The overall relative risks for current smoking were calculated from smoking population weighted sex-specific relative risks equal to the population weighted sex specific proportion of current smoking:

\[
\begin{align*}
R_c^* &= \left[ p_{b,c,m} R_{c,m}^* + p_{b,c,f} R_{c,f}^* \right] / \left( p_{b,c,m} + p_{b,c,f} \right) \quad [S-3a] \\
R_q^* &= \left[ p_{b,q,m} R_{q,m}^* + p_{b,q,f} R_{q,f}^* \right] / \left( p_{b,q,m} + p_{b,q,f} \right) \quad [S-3b]
\end{align*}
\]

where

\[ p_{b,c,s} = \text{proportion of current smoking in sex } s \text{ before the smoking law}, \]

\[ R_{q,s}^* = \text{average relative risk of recent quitters in sex } s \text{ from adoption of law to 12 months after the law took effect (that is, } n = 12 \text{ months)}. \]

The overall proportion of current and ex-smoking before the ban is equal to the population weighted sex specific proportion of current smoking:

\[
p_{b,z} = p_{b,z,m} p_m + p_{b,z,f} (1 - p_m) , \quad [S-4]
\]

where
\( p_m \) is the proportion of men age 18 to 64 years old,

\( z \) is \( c \) for current smoking, and \( f \) for ex-smokers.

Data limitations prevent treatment of never and former smokers as distinct categories for analysis of passive smoking, so all non-smokers are used as the reference group. First the referent group is changed from never-smokers to all non-smokers who are not recent quitters (that is, anyone who has not quit due to the smoking ban). Therefore the relative risk for current smokers age 18 to 64 is adjusted so that the reference group is all non-smokers. This conversion is done by dividing the average RR for both sexes (the weight average of sex specific relative risks for current and former smokers in equations [S-3a] and [S-3b], respectively) to the average RR of never-smokers for both sexes as the referent group, by dividing through by the smoking population weighted average relative risk of never and former smokers:

\[
\begin{align*}
R_{c,\text{adults}} &= R_c^* / R_{-c}, \\
R_q &= R_q^* / R_{-c},
\end{align*}
\]  
[S-5a]
[S-5b]

where,

\( R_{c,\text{adults}} \) = the relative risk for all adults age 18 to 64, using never-smokers as the reference group,

\( R_{-c} \) = the average relative risk of all non-smokers of both sexes.

The relative risk of all non-smokers (never-smokers and ex-smokers), \( R_{-c} \), is calculated using the relative risk of ex-smokers, population prevalence of current and ex-smokers, and male proportion of the population age 18-64:

\[
R_{-c} = \left[ \left( \frac{p_{b,x,m}}{1 - p_{b,c,m}} \right) p_m + \left( \frac{p_{b,x,f}}{1 - p_{b,c,f}} \right) \left( 1 - p_m \right) \right] (1 - R_x) + R_x,
\]  
[S-6]
where

\[ R_x = \text{the relative risk of ex-smokers.} \]

Conversion of relative risks of current smokers to all adults over 18. The relative risks for AMI from current smoking apply to adults age 18 to 64 is adjusted to apply to all adults over age 18. This adjustment is done by smoking population weighted average adjustment using the ratio of overall relative risk for adults age 18 to 64 and overall relative risk for those age 65 and over:

\[
R_e = R_{c, \text{adults}} \left[ p_{c,e} \left( \frac{R_{c,e}}{R_{c,y}} \right) + (1 - p_{c,e}) \right], \tag{S-7}
\]

where

\[ R_{c,y} = \text{overall relative risk of AMI from active smoking, adults age 18-64,} \]

\[ R_{c,e} = \text{overall relative risk of AMI from active smoking, adults age 65 and over,} \]

\[ p_{c,e} = \text{proportion of adult smokers age 65 and over.} \]

The prevalence of smokers who are over age 65 were calculated from age-specific prevalence of smoking\(^2\) and age distribution of the resident population of the U.S.\(^3\) The data and resultant proportion of current smokers of age 65 and over are shown in Table S-5.
<table>
<thead>
<tr>
<th>Location</th>
<th>Date of study*</th>
<th>End point</th>
<th>Ages</th>
<th>Measure</th>
<th>Statistical Method</th>
<th>Confounders</th>
<th>N (events)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Italy (4 regions)^2</td>
<td>10 Jan 2005</td>
<td>AMI (ICD-9 410)</td>
<td>40-64</td>
<td>Age-standardized rates (European)</td>
<td>Comparison of observed rate after law with expected value based on linear secular trend for same months during the 4 years before the law went into effect.</td>
<td>Age, gender, region</td>
<td>.77 (.74, .82)</td>
<td>Small decreases in smoking prevalence (30.0 to 29.3% in men and 22.5% to 22.1% in women) and consumption (16.7 to 16.3 cig/day for men and 13.7 to 13.4 cig/day for women) led to a 7.6% decline in cigarette consumption^1. Fewer than 100 violations in 6000 checks by police^6. 90-95% reduction in air nicotine in pubs and discos^7. 8.9% decline in cigarette sales in 2005^6. Effect largest among young men and people 45-54. Some regional variation.</td>
</tr>
<tr>
<td>Helena, MT^2</td>
<td>Law in effect 6 months, from Jun 5 – Dec 3, 2002</td>
<td>Primary and some secondary (validated with troponin or CPK) diagnosis of AMI (ICD-9 410)</td>
<td>All</td>
<td>Number of admissions during 6 month period the law was in effect compared to the average for the same 6 months in other years by Poisson test</td>
<td>Comparison with number of admissions from surrounding area (not covered by law)</td>
<td>.56 (.20, .93)</td>
<td>No significant change in admission patterns from patients from surrounding area. Analysis did not consider fact that admissions were increasing with time, which biases comparison toward null.</td>
<td>304</td>
</tr>
</tbody>
</table>

---

^1 (4 regions)^2


^4 AMI (ICD-9 410)

^5 Comparison of observed rate after law with expected value based on linear secular trend for same months during the 4 years before the law went into effect. Age, gender, region.

^6 Fewer than 100 violations in 6000 checks by police.

^7 90-95% reduction in air nicotine in pubs and discos.

^8 8.9% decline in cigarette sales in 2005.
Table S-1. Summary of Studies of the Effects of Individual Studies on Changes in AMIs following Implementation of Smokefree Laws (cont.)

<table>
<thead>
<tr>
<th>Location</th>
<th>Effective Date / Study period</th>
<th>End point</th>
<th>Ages</th>
<th>Measure / Statistical Method</th>
<th>Confounders</th>
<th>Risk Observed</th>
<th>Risk At 12 months</th>
<th>Exposure Change</th>
<th>N (events)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piedmont, Italy</td>
<td>10 Jan 2005</td>
<td>AMI (ICD-9 410)</td>
<td>All</td>
<td>Age-standardized rates (European)</td>
<td>Age</td>
<td>&lt;60: 0.89 (0.81, 0.98)</td>
<td>m: 0.91 (0.82, 1.01)</td>
<td>f: 0.75 (0.56, 0.96)</td>
<td></td>
<td>.83 (76, 92)</td>
</tr>
<tr>
<td></td>
<td>Compared Oct-Dec 2004 (before law) and Feb-June 2005 (after law) with same periods 1 year earlier Post: 6 Pre: 3 (but see above)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>60: 1.05 (1.00-1.11)</td>
<td>m: 1.03 (0.96-1.11)</td>
<td>f: 1.05 (0.91-1.11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scotland</td>
<td>10 Apr 2006</td>
<td>Acute coronary syndrome (detectable troponin after emergency admission for chest pain) (ICD-10 I21)</td>
<td>All</td>
<td>Chi-square and test for trend</td>
<td>Stratified on gender and age (men&lt;55; women&lt;65)</td>
<td>.83 (82, 84)</td>
<td>.81 (80, 84)</td>
<td>.83 (82, 84)</td>
<td>.81 (80, 84)</td>
<td>5919</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>5 July 2004</td>
<td>Acute myocardial infarction</td>
<td>All</td>
<td>Regression</td>
<td>.82 (76, 89)</td>
<td>.82 (76, 89)</td>
<td>.82 (76, 89)</td>
<td>.82 (76, 89)</td>
<td>NA</td>
<td>Much of state was already covered by strong local laws. No effect of state law when already strong local law.</td>
</tr>
</tbody>
</table>
### Table S-1. Summary of Studies of the Effects of Individual Studies on Changes in AMIs following Implementation of Smokefree Laws (cont.)

<table>
<thead>
<tr>
<th>Location</th>
<th>Effective Date / Study period Post/Pre duration (months)</th>
<th>End point Description</th>
<th>Ages</th>
<th>Measure / Statistical Method</th>
<th>Confounder s</th>
<th>Risk</th>
<th>Exposure Change</th>
<th>N (events)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saskatoon, Canada</td>
<td>July 1, 2004 1 Jul 2000 to 30 June 2005 Post: 12 Pre: 48</td>
<td>AMI (ICD-10)</td>
<td>All</td>
<td>Incidence ratio and confidence interval post law compared to pre.</td>
<td>Age</td>
<td>.87 (.85, .93)</td>
<td>.87 (.85, .93)</td>
<td>914 of 924 eligible businesses establishments were inspected by a public health inspector within the first 6 months of the law; only 13 required an initial warning for non-compliance. Re-inspection only required 1 citation being issued during the first year of the law. Smoking prevalence in Saskatoon fell from 24.1% in 2003 (95% CI 20.4-27.7) to 18.2% in 2005 (15.7-20.9); smoking in the rest of Saskatchewan Province (which includes Saskatoon) remained stable from 2003 to 2005 at 23.8% (22.6-25.3). One year after implementation (July 2005), 79% responded that the “smoking ban was a good idea.”</td>
<td>1689</td>
</tr>
<tr>
<td>Rome, Italy</td>
<td>10 Jan 2005 Jan 2000 to Dec 2005 Post: 12 Pre: 48</td>
<td>Acute coronary events, including AMI (ICD-9 410) and “other acute and subacute forms of ischemic heart disease” (ICD-9 411). Cases were included with principal diagnosis of AMI or secondary diagnosis of AMI when principal diagnosis indicated AMI complications. Out of hospital deaths from ischemic heart diseases (ICD-9 410-414) if no evidence of hospitalization for coronary causes in the previous 28 days or any cause in the last 2 days.</td>
<td>35-84</td>
<td>Age standardized rates (European) Poisson regression on number of daily events after 10 Jan 2005 compared to before Separate analyses done for out-of-hospital deaths and hospitalizations and an analysis of incident cases only.</td>
<td>Age, gender, PM_{10} air pollution, flu epidemics, holidays, temperature, secular trend, all-cause hospitalizations, socio-economic status</td>
<td>3.5-64: .89 (.83, .93) 65-74: .92 (.88-.97)</td>
<td>.89 (.85, .93)</td>
<td>Prevalence of smoking decreased from 34.9% to 30.5% in men and from 20.6% to 20.4% in women. Cigarette sales decreased in Rome by 5.5% in 2005 compared to 2004. See also entry for Italy.</td>
<td>2136</td>
</tr>
</tbody>
</table>
| Ireland           | 29 March 2004 Post: 12 Pre: 12                           | Acute coronary syndrome | All  | Poisson regression          |              | .89 (.81, .97) | .89 (.81, .97) | Among bar workers, cotinine concentration fell by 69% and 74% reported reduced secondhand smoke exposure. | ~3300      | Reduction maintained for additional 12 mos.
### Table S-1. Summary of Studies of the Effects of Individual Studies on Changes in AMIs following Implementation of Smokefree Laws (cont.)

<table>
<thead>
<tr>
<th>Location</th>
<th>Effective Date / Study period</th>
<th>End point</th>
<th>Ages</th>
<th>Measure / Statistical Method</th>
<th>Confounders</th>
<th>Risk</th>
<th>Exposure Change</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pueblo, CO</strong></td>
<td>1 Jul 2003, Jan 2002 to Dec 2004</td>
<td>Primary diagnosis of AMI (ICD-9 410) (Primary diagnosis only)</td>
<td>All</td>
<td>Poisson regression</td>
<td>Seasonality, population size</td>
<td>73 (.64, .82) m: .75 (.61, .90) f: .70 (.53, .87)</td>
<td>.78 (.68, .88)</td>
<td>.78 (.68, .88)</td>
</tr>
<tr>
<td>New York State</td>
<td>24 Jul 2003, Jan 1995 to Dec 2004</td>
<td>AMI (ICD-9 410), primary diagnosis only</td>
<td>35+</td>
<td>Multiple regression time series</td>
<td>Age-adjusted (NY population in 2000)</td>
<td>8004 (.7985, .8023) (Juster, personal communication for Confidence Interval)</td>
<td>.886 (.894, .888)</td>
<td>.886 (.894, .888)</td>
</tr>
<tr>
<td>Bowling Green, OH</td>
<td>Mar 2002, Jan 1999 to Jun 2005</td>
<td>Coronary heart disease, including angina, heart failure, arteriosclerosis, and AMI (ICD-9 410-414, 428)</td>
<td>18+</td>
<td>Age-standardized rates</td>
<td>ARIMA</td>
<td>Ordinance effect assumed to start in Oct 2002</td>
<td>.61 (.55, .67) in 2003 (1 year later)</td>
<td>.53 (.45, .59) in first half of 2005, (2.5 years later)</td>
</tr>
<tr>
<td><strong>Pueblo, CO</strong></td>
<td>Extend to Jun 2006</td>
<td>Primary diagnosis of AMI (ICD-9 410) (Primary diagnosis only)</td>
<td>All</td>
<td>Comparison of rate ratios</td>
<td>Comparison with people living in surrounding Pueblo County (not covered by ordinance) and with nearby El Paso County (which did not have an ordinance)</td>
<td>.59 (.49, .70) m: .67 (.52, .82) f: .48 (.36, .60)</td>
<td>.77 (.64, .92)</td>
<td>.77 (.64, .92)</td>
</tr>
</tbody>
</table>

- **Notes**: New York State: By 2002, 73% of New Yorkers were subject to strong local laws, as well as limited restrictions at the state level implemented in 1989.
- New York State: No sudden change with law; rate of decline in AMI admissions increased significantly over moderate or no local laws. Also considered primary diagnosis of stroke (ICD-9 430-438); no association of law with stroke.
<table>
<thead>
<tr>
<th>Location</th>
<th>Effective Date / Study period</th>
<th>End point</th>
<th>Ages</th>
<th>Measure / Statistical Method</th>
<th>Confounders</th>
<th>Risk</th>
<th>Exposure Change</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monroe County, IN</td>
<td>1 Aug 2003, bars added 1 Jan 2005</td>
<td>Acute myocardial infarction (ICD-9 410), confirmed with troponin or CPK, excluding past cardiac procedures, no cardiac risk factors (e.g., hypertension or hypercholesterolemia)</td>
<td>All</td>
<td>Poisson test</td>
<td>Compared with Delaware County (no law)</td>
<td>Significant drop in number of nonsmokers admitted in Monroe County, but not Delaware County (control). No change in number of smokers admitted.</td>
<td>56</td>
<td>Bar provisions only in effect for last 5 months of post period. There was a 48% reduction in AMIs between pre and post period (nonsmokers and smokers combined). No RR or CI available. Unrealistically stringent exclusionary criteria</td>
</tr>
<tr>
<td>France</td>
<td>1 Feb 2007, restaurants, bars, casinos added 1 Jan 2008</td>
<td>Acute myocardial infarction</td>
<td>All</td>
<td>Rate per 100,000 admissions</td>
<td>Age &lt; 65: .85 Age &gt; 66: .88</td>
<td>Between Jan 2007 (before law) and Jan 2008 (after law) SHS exposure dropped from 57% to 14%. PM2.5 levels dropped.</td>
<td>NA</td>
<td>Also report substantial drops in respiratory symptoms &lt;&lt;slide 47 ff&gt;&gt;</td>
</tr>
</tbody>
</table>

---

Notes:  
1 Includes outdoor areas of restaurants and bars  
2 Excludes bars  
3 Pool of estimate of Weak local law (.82; .73, .87) and No local law (.83; .69, .99) (fixed effects meta-analysis; risk estimates homogeneous)  
4 Updated results  
5 RR and confidence intervals adjusted to 12 months using ln(RR) = ln(RR) + 0.0113 (Post – 12)  
6 Random effects meta-analysis  
7 In some cases, there are more studies of changes in exposure. Those listed here are typical. If no citation is included in this column, the results are from the AMI study.  
8 Smoking allowed in separately ventilated public rooms with doors maintained under negative pressure. Few businesses are willing to incur this expense to maintain smoking.  
9 ICD-9 427.1, 427.41, 427.42, 427.5, 428.1, 429.5, 429.6, 429.71, 429.79, 429.81, 518.4, 780.2, 785.5, 414.10, 423.0.
<table>
<thead>
<tr>
<th>Area of smoking law study</th>
<th>men</th>
<th>women</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>prevalence</td>
<td>SE</td>
<td>prevalence</td>
</tr>
<tr>
<td>Helena Montana</td>
<td>0.204</td>
<td>0.015306</td>
<td>0.222</td>
</tr>
<tr>
<td>Pueblo Colorado</td>
<td>0.205</td>
<td>0.011735</td>
<td>0.194</td>
</tr>
<tr>
<td>Piedmont Italy</td>
<td>0.314</td>
<td>0.0163600</td>
<td>0.176</td>
</tr>
<tr>
<td>Bowling Green Ohio</td>
<td>0.204</td>
<td>0.014796</td>
<td>0.265</td>
</tr>
<tr>
<td>New York State</td>
<td>0.19</td>
<td>0.011224</td>
<td>0.188</td>
</tr>
<tr>
<td>Ireland</td>
<td>0.28</td>
<td>0.018312</td>
<td>0.26</td>
</tr>
<tr>
<td>Saskatoon Canada</td>
<td>0.234</td>
<td>0.015306</td>
<td>0.234</td>
</tr>
<tr>
<td>Rome Italy</td>
<td>0.314</td>
<td>0.0163600</td>
<td>0.176</td>
</tr>
<tr>
<td>Glasgow</td>
<td>0.25</td>
<td>0.01635</td>
<td>0.23</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>0.20</td>
<td>0.01204</td>
<td>0.174</td>
</tr>
</tbody>
</table>
### Table S-3. Parameter distributions used in subsidiary calculations

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean (95% CI)</th>
<th>Distribution</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative risk, current smokers, all adults age 18-64, $R_{c,y}$</td>
<td>3.53 (3.21, 3.85)</td>
<td>normal</td>
<td>Teo KK 2006[27]</td>
</tr>
<tr>
<td>Relative risk, current smokers, all elderly, $R_{c,e}$</td>
<td>2.55 (2.34, 2.76)</td>
<td>normal</td>
<td>Teo KK 2006[27]</td>
</tr>
<tr>
<td>Relative risk, ex-smokers, all adults, $R_x$</td>
<td>1.49 (1.39, 1.59)</td>
<td>normal</td>
<td>Teo KK 2006[27]</td>
</tr>
<tr>
<td>current smokers, adult men, before ban, $p_{b,c,m}$</td>
<td>0.239 (0.211,0.267)</td>
<td>normal</td>
<td>CDC State Tobacco Activities Tracking and Evaluation (STATE) System, OECD Health Data 2007[24]</td>
</tr>
<tr>
<td>current smokers, adult women, before ban, $p_{b,c,f}$</td>
<td>0.211 (0.190,0.231)</td>
<td>normal</td>
<td>CDC State Tobacco Activities Tracking and Evaluation (STATE) System, OCD Health Data 2007[24]</td>
</tr>
<tr>
<td>ex-smokers, adult men, before ban, $p_{b,x,m}$</td>
<td>0.259 (0.251,0.267)</td>
<td>normal</td>
<td>CDC State Tobacco Activities Tracking and Evaluation (STATE) System[28]</td>
</tr>
<tr>
<td>ex-smokers, adult women, before ban, $p_{b,x,f}$</td>
<td>0.184 (0.177,0.191)</td>
<td>normal</td>
<td>CDC State Tobacco Activities Tracking and Evaluation (STATE) System[28]</td>
</tr>
<tr>
<td>Proportion of adult population, men, $p_m$</td>
<td>0.498</td>
<td>constant</td>
<td>resident population, Census Bureau[3]</td>
</tr>
<tr>
<td>Proportion of smokers who are elderly, $p_{c,e}$</td>
<td>0.07</td>
<td>constant</td>
<td>resident population, Census Bureau[28]; MMWR 2005[2] (See Table S-4)</td>
</tr>
</tbody>
</table>
### Table S-4. Regression parameters and variance-covariance matrix for relative risks of current smoking and quitting

<table>
<thead>
<tr>
<th>Active Smoking Relative Risk Variables</th>
<th>Variable</th>
<th>Mean</th>
<th>Covariance</th>
<th>Matrix</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>$R_{c,m}$</td>
<td>$R_{c,f}$</td>
</tr>
<tr>
<td>$R_{c,m}$</td>
<td>2.881</td>
<td>0.09927</td>
<td>-0.07451</td>
<td>0.0006320</td>
</tr>
<tr>
<td>$R_{c,f}$</td>
<td>0.9719</td>
<td>-0.07451</td>
<td>0.1139</td>
<td>0.002037</td>
</tr>
<tr>
<td>$R_{\infty,m}$</td>
<td>1.166</td>
<td>0.0006320</td>
<td>0.002037</td>
<td>0.01783</td>
</tr>
<tr>
<td>$R_{\infty,f}$</td>
<td>0.2304</td>
<td>0.01129</td>
<td>-0.007080</td>
<td>0.01129</td>
</tr>
<tr>
<td>$\tau$</td>
<td>19.10</td>
<td>-1.010</td>
<td>0.2175</td>
<td>-0.2391</td>
</tr>
</tbody>
</table>

Source: Lightwood and Glantz

### Table S-5.—Calculation of proportion of current smokers who are elderly

<table>
<thead>
<tr>
<th>age group</th>
<th>resident population</th>
<th>prevalence of current smoking</th>
<th>current smoking smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-24 years old</td>
<td>28,889,168</td>
<td>0.260</td>
<td>7511184</td>
</tr>
<tr>
<td>25-44 years old</td>
<td>84,216,990</td>
<td>0.284</td>
<td>23917625</td>
</tr>
<tr>
<td>45-64 years old</td>
<td>68,646,935</td>
<td>0.234</td>
<td>16063383</td>
</tr>
<tr>
<td>&gt; 64 years old</td>
<td>35,957,792</td>
<td>0.101</td>
<td>3631737</td>
</tr>
<tr>
<td>total</td>
<td></td>
<td></td>
<td>51123929</td>
</tr>
<tr>
<td>proportion elderly among smokers</td>
<td></td>
<td></td>
<td>0.071</td>
</tr>
</tbody>
</table>

REFERENCES


