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IMPACT OF TOBACCO CONTROL ON LUNG CANCER MORTALITY IN THE U.S. OVER THE PERIOD 1975–2000 SUMMARY AND LIMITATIONS

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Abstract

Background—A consortium of six research groups estimated the impact on lung cancer mortality of changes in smoking behavior that began around the publication of the surgeon general's report. This chapter presents the results of that effort. We quantified the cumulative impact of changes in smoking behaviors on lung cancer mortality in the U.S. over the period 1975–2000.

Methods—The six groups used common inputs and independent models to estimate the number of U.S. lung cancer deaths averted over the period 1975–2000 as a result of changes in smoking behavior beginning in the mid-fifties, and the number of deaths that could have been averted if tobacco control had completely eliminated all smoking following issuance of the first Surgeon General's report (SGR) on Smoking and Health in 1964.

Results—Approximately 795,000 deaths (550,000 men and 245,000 women) were averted over the period 1975–2000 as a result of changes in smoking behavior since in 1950s. In the year 2000 alone approximately 70,000 lung cancer deaths were averted (44,000 among men and 26,000 among women). However, these represent approximately 30% of lung cancer deaths that could have potentially been averted over the period 1975–2000 if smoking was eliminated completely. In the ten-year period 1991–2000, this fraction increased to about 37%.

Conclusions—Our results show the substantial impact of changes in smoking behavior since the 1950s. Despite a major impact of changing smoking behaviors, tobacco control effort are still needed to further reduce the burden of this disease.

Keywords

lung cancer; tobacco control; simulation modeling

1. INTRODUCTION

An important question in tobacco control is the impact of the seminal surgeon general's 1964 report and its sequelae on lung cancer mortality in the U.S. This monograph attempts to quantitatively address that question. A consortium of six research groups set out to measure the impact of changes in smoking behavior that began around the time that the surgeon general's report appeared. Specifically, the research groups set out to investigate the relationship between smoking behavior and lung cancer mortality in the United States over

the period 1975–2000, focusing on the impact of changes in smoking behavior associated with tobacco control on death from lung cancer.

While the basic relationships between smoking and lung cancer risk are well understood from epidemiological studies, lung cancer trends in the population over time are too complex to be captured in standard epidemiological models. The study of populations-level trends in lung cancer mortality as they relate to changes in smoking behavior requires the use of a comprehensive modeling approach consisting of two distinct elements. First, the U.S. population in the period 1975–2000 must be partitioned into sub-groups of never-smokers, ex-smokers, and smokers. For ex-smokers and smokers, information is also needed on the detailed smoking histories, including age of initiation, age at quit and the intensity of smoking. To complicate matters further, information is needed about competing causes of mortality by smoking status. These problems were addressed by developing a detailed smoking history generator (SHG) for individuals born in the birth cohorts of 1900–1960. The development of the SHG is described briefly below and in detail in chapter 5 of this monograph.

The second element in the comprehensive model is the development of exposure-response (dose-response) models for cigarette smoking and lung cancer. The exposure-response model uses the population of smokers and ex-smokers generated by the SHG to estimate lung cancer risks associated with cigarette smoking in the population. These models also provided estimates of lung cancer rates among the never-smokers. Each of the research groups involved in this effort developed its own dose-response model for this purpose. Thereby, the six groups involved in this effort began with common inputs (i.e., specific smoking scenarios and mortality from other causes as simulated by the SHG as described below) and used their own dose-response models to estimate the number of lung cancer deaths in each smoking group for the various smoking scenarios.

Starting with common inputs by cohort and year on smoking behaviors and mortality from causes other than lung cancer, each group modeled the lung cancer mortality rates among males and females in the U.S. over the period 1975–2000. Each group then estimated the total number of lung cancer deaths avoided in males and females over the same period as a direct result of tobacco control measures as well as the number of lung cancer deaths that could have been avoided had smoking stopped entirely in 1965.

2. METHODS

2.1. Model Overview

Each model includes a dose response module with a quantitative description of the age-specific mortality of lung cancer by detailed history of smoking. The dose-response modules did not incorporate other risk factors, such as environmental tobacco smoke, diet, radon exposure, and air pollution. Some groups addressed this limitation of the dose-response module by calibrating their models to describe actual deaths in the U.S. population over the period 1975–2000 (see Table I. Other groups estimated lung cancer deaths in hypothetical populations with the smoking habits and the age structure of the U.S. population over the period 1975–2000. The overall model structure is shown in Fig. 1.

Three smoking scenarios were considered. Each of these involved a detailed description of smoking behavior by gender, age, and birth cohort for births from 1890 to 1970. The first scenario, called actual tobacco control (ATC), describes the observed smoking histories in the U.S. The second scenario, called no tobacco control (NTC), describes smoking behavior predicted under the assumption of no effect from tobacco control efforts, i.e., prior trends continue. The third scenario, called complete tobacco control (CTC), assumes that all

smoking ceased in 1965, i.e., all smokers quit and no individuals initiated smoking after 1964. Calibration allows the ATC runs to match observed mortality. These same calibration factors are then applied to the NTC and CTC runs.

2.2. The Smoking History Generator

The smoking history generator (SHG) is a shared precursor model that produces cohort-specific smoking histories and related rates of mortality from other cause than lung cancer. The SHG was used to generate inputs for the larger dose-response, survival generation models (Figure 1). For each smoking scenario (ATC, NTC, CTC), the SHG provides as its output detailed individual level smoking histories for individuals born between 1890 and 1970 for the period 1975 up to the calendar year 2000. Causes of death other than lung cancer are also outputs of the SHG. Each individual history is terminated at age 85 or at the age of death from a cause other than lung cancer if the death occurred prior to age 85. More detail on the SHG is contained in Chapter 2. Figure 2 shows one result of the SHG, the proportion of current smokers generated by the SHG among the simulated individual histories under the three smoking scenarios. Figure 2 also shows that smoking prevalence started to decline from the 1950s, which is after the first evidence of lung cancer risk due to smoking became available, but before the Surgeon General's report. This early decline is also considered to be due to tobacco control.

The outputs of the smoking history generator are used as inputs by each model's dose response module to determine the timing of mortality from lung cancer. The individual level cohort models of the consortium use individual smoking histories as inputs for a biologic model to determine when a lung cancer death occurs, while the aggregated, cohort models use cross sectional proportions of the population defined by calendar-year, gender, and age, stratified by smoking status. Regardless of model scale, the event of lung cancer is super-imposed over the SHG-generated events to produce a complete life history (see Figure 1).

2.3. Specific Models

The specific models of the consortium are described in greater detail in chapters 7–12 of this monograph and on the CISNET website developed to facilitate cross-model comparisons (<http://cisnet.cancer.gov/profiles/>). The models are also summarized in chapter 13. The models represent a diverse group with respect to both their prior and future areas of application. Although this monograph focuses on the effect of tobacco control on lung cancer mortality, another important aim of the collaboration was to facilitate comparison of model structure, assumptions, and the relative strengths and weaknesses of data sets utilized for model calibration and validation. Future models will be used to address other questions, which can then serve as a further basis for comparing key components of the models.

With the exception of the MGHMS group, which used a logistic regression model, the other groups used the two-stage clonal expansion (TSCE) model^(1–3) for the dose-response module. The TSCE model is a mathematical formulation of the widely accepted biological theory of initiation, promotion, and progression. It recognizes that carcinogenesis is a process of mutation accumulation and clonal expansion of partially altered cells on the pathway to malignancy. This type of model has generally shown that clonal expansion (promotion) of partially altered (initiated) cells by cigarette smoke is the dominant model mechanism to explain patterns of lung cancer risk, and confirmed that smoking duration has a stronger association with lung cancer risk than cigarettes smoked per day^(2–5). The TSCE model is capable of accommodating detailed individual-level smoking histories. That model includes at maximum one smoking period, characterized by age(s) at start and quit, and changes in the intensity of smoking.

Three of the groups (FHCRC, MGH-HMS, and Yale) calibrated their models against the U.S. lung cancer mortality data by embedding their dose-response module in age-period-cohort (APC) models^(6–8). The outputs from these models were direct counts of the numbers of lung cancer deaths in the U.S. population by year of observation and age over the period 1975–2000. One group (PIRE) used a partial calibration (considering overall time trends) instead of a complete APC calibration. Two (Erasmus MC and Rice-MDA) groups did not use any calibration against the U.S. mortality data and used the smoking generator to estimate the distribution of smoking histories representative of the U.S. population under the three smoking scenarios (ATC, NTC, and CTC) described above. Each group then used its dose-response module to estimate the age-specific lung cancer rates in these populations.

The decision whether to calibrate to observed trends in mortality in the context of the smoking base case is subject to a variety of different approaches. Ideally, the model would incorporate all factors that influence population trends in lung cancer risk, including changes in the composition and manufacture of cigarettes, uncertainties in the estimated smoking histories distribution, the effect of smoking on mortality from other causes than lung cancer, radon remediation efforts, changes in diet, the changing impact of second hand smoke, increasing disparities in income/education and other relevant factors. In the absence of reliable information on the various factors influencing population trends, some modelers chose to add non-specific factors to calibrate to population level trends. These factors are not attributed to specific causal influences on lung cancer but can help reveal if the missing components are associated with calendar time or year of birth. While the relationship between the calibration factors and tobacco control in the U.S. population is not known, calibration of a model to the observed U.S. trends in lung cancer mortality does not necessarily improve the accuracy of the estimated effects of tobacco control.

3. RESULTS

For presentation purposes of this chapter, we chose to show results for only one exemplar model (the Yale model). The results from the other models are different, but the predicted trends and predicted mortality differences between scenarios are similar. Figure 3 shows the actual rates and numbers of lung cancer deaths among males and females in the U.S. over the period 1975–2000, and also the numbers that would have been expected under the NTC and CTC scenarios. Over the period 1975–2000, there were 2,067,775 lung cancer deaths among males and 1,051,978 lung cancer deaths among females in the U.S. Under the NTC scenario, the Yale model estimates 2,670,897 lung cancer deaths among males and 1,273,151 lung cancer deaths among females, while under the CTC scenario, deaths number 958,862 and 438,858, respectively, among males and females. The observed number of lung cancer deaths among men reached a plateau by the year 1990, while under the NTC scenario this number would have continued to increase.

Among females, the observed number of lung cancer deaths was still increasing by 2000 but not as rapidly as under NTC. The difference between the NTC scenario and the observed numbers provides an estimate of the number of lung cancer deaths avoided (A), which for the Yale model is just over 603,000 and 221,000 for males and females respectively. The difference between NTC and CTC scenarios provides an estimate of the total number of lung cancer deaths that could have been avoided if tobacco control efforts had been immediately and completely successful (B), approximately 1,712,035 males and 834,293 females for the Yale model.

The other models calibrated to US mortality (FHCRC, MGH-HMS) yielded similar estimates of the number of lung cancer deaths under the three scenarios. Counts of the differences in the number of deaths between scenarios are shown for all models in Table II

and Figure 4. The fraction of realized to potential (i.e., those potentially realized) lung cancer deaths is also presented in Table II. Means are presented in order to give an impression of around which value the model results vary but are not representing a best point estimate. The models estimate that of all avoidable deaths from smoking-related lung cancer, between 24 and 32 percent of female deaths and between 30 and 37 percent of male deaths were actually avoided as a result of the tobacco control efforts beginning in the mid-fifties. For both genders combined, approximately 30% (28% to 35% across models) of all avoidable deaths were averted. Table II also shows the impact of tobacco control efforts on lung cancer mortality for the decade 1991–2000 and for the year 2000. In the decade 1991–2000, the fraction of lung cancer deaths averted in males and females combined increased to about 37% (34% to 43% across models). In the year 2000, this fraction increased to roughly 45% (39% to 50% across models). The increasing trend in the fraction of lung cancer deaths averted reflects both changes in smoking habits and a continuing decrease in risk among ex-smokers.

The results can be compared to those of Thun and Jemal ⁽⁹⁾. Using a straightforward projection, they estimated that reductions in tobacco smoking averted approximately 146,000 lung cancer deaths among U.S. males over the period 1991–2003. The CISNET models predict that under NTC, the increasing trend of mortality rates bend off toward a plateau instead of continuing to increase at a constant slope as assumed by Thun and Jemal ⁽⁹⁾. This method tends to lead to a lower estimated effect of tobacco control. However, the CISNET models also predict that tobacco control started affecting lung cancer mortality long before 1975, yielding reductions in lung cancer mortality much larger than those estimated by Thun and Jemal. Over the period 1991–2000, approximately 345,000 lung cancer deaths among U.S. males and 175,000 deaths among U.S. females were averted due to changes in smoking habits starting in the mid-fifties. In the year 2000 alone, approximately 44,000 deaths among U.S. males and 26,000 deaths among U.S. females were averted. Over the period 1975–2000, the models estimate that approximately additional 1,500,000 lung cancer deaths among males and females combined could have been averted had tobacco control efforts been completely effective in eliminating smoking as of 1965.

Also of interest is the specific role of smoking in lung cancer deaths. The population-attributable fraction (PAF) is a measure of the fraction of disease in a population attributable to a specific risk factor. The vast majority of lung cancer deaths in the U.S. over the period 1975–2000 are attributable to cigarette smoking. Figure 5 shows the observed lung cancer deaths among U.S. males and the Yale model results for the hypothetical situation in which the U.S. male population only includes never smokers. The difference between the number of observed deaths and the number expected in a population of never smokers is 1,740,284. Under the ATC scenario, the PAF among males declines modestly from approximately 86% in 1975 to about 80% in 2000. Among females, the PAF actually increases from about 73% to about 80% reflecting the increase in cigarette smoking by females over this period. Under the NTC scenario, the increase among females would have been even larger. Thus, while changes in smoking habits have been responsible for a substantial slowing in the rate of increase of lung cancer mortality over the period 1975–2000, the vast majority of lung cancer deaths in 2000 are still attributable to cigarette smoking.

4. DISCUSSION

4.1. Summary

The results of the base case analyses show the dramatic impact of the reduction in smoking associated with tobacco control efforts on lung cancer mortality from 1975–2000. Nevertheless, the results indicate that only approximately 30% of the total lung cancer deaths that could have been averted had tobacco control been complete were actually saved.

This result reflects that the gradual decline in smoking rates from around the time of the first Surgeon's General Report on Smoking, while a sizable fraction of the population continues to smoke and the risk of lung cancer remains elevated for many years after smoking cessation. With The CISNET group used a comparative modeling approach to address this complex problem. However, multiple, independent efforts to model the same outcomes often yield disparate results that are difficult to reconcile. When coordinated using common inputs, as in this effort, there may still be differences, but they are easier to isolate. The various models yield a range of estimates of the fraction of lung cancer deaths avoided by the tobacco control efforts in the U.S.

Differences between the model results are due to several reasons. First, even though five of the six groups used the TSCE model as the dose-response module, the estimated parameters were different because they were estimated by fits to data corresponding to different cohorts. It is well known that the risks of tobacco smoking have changed over time and are modified, moreover, by other factors, such as diet, not incorporated in any of the models. Therefore, different study cohorts may yield different estimated dose-response relationships. Three of the models were calibrated against U.S. mortality data. As a consequence, these models weight the relative effects of tobacco control over time differently than the models that did not calibrate, which in turn may have resulted in differences in the estimated fractions of lung cancer deaths avoided.

Despite these differences, the estimated number of deaths avoided and deaths that could have been avoided with perfect tobacco control are quite similar across all models (Table II). The main message of these analyses is clear: Tobacco control starting mid-20th century has averted hundreds of thousands of lung cancer deaths in the U.S. over the period 1975–2000, but these are only approximately 30% of the lung cancer deaths that could have been averted had tobacco control been complete.

4.2. Limitations

The FHCRC, MGH-HMS, and Yale groups calibrated their models to U.S. mortality over the period 1975–2000 using birth cohort and period effects. These calibrations are necessary to describe lung cancer mortality rates and trends in the U.S. and indicate that the lung cancer mortality experience of the entire population cannot be adequately described by extrapolating from the SEER registry in one decade, or from different cohort studies of smoking and lung cancer. While the calibrated ATC models match observed mortality well, the resulting differences between the runs (the primary outcome of this work), are not necessarily improved for models that are calibrated over different scenarios (i.e., the CTC and NTC scenarios). Lung cancer mortality, particularly among males, is considerably higher than would be expected from cohort studies. In addition, models based on cohort studies and available population smoking histories cannot adequately describe the age, period and cohort components of trends.

One reason for the discrepancy between the observed population rates and those predicted by the fit of models to specific cohorts is that the analytical cohorts may not be representative of the general population. For example, a healthier cohort than the rest of the population, such as those in the CPS-I and II, would yield lower mortality rates. Second, smoking histories for birth cohorts in the general population are inferred by reconstructing through simulation using cross-sectional histories. Those histories rely on the recall of events that will have occurred in previous years. By contrast, prospective cohort studies directly determine exposures for each individual. Third, potentially important covariates, such as diet, air pollution, radon exposure and occupational exposures including asbestos and ionizing radiation are not available for the overall population, and different exposure distributions may contribute to rate discrepancies. Fourth, the models discussed above

assume a consistent effect of exposure on lung cancer mortality, but temporal changes in the manufacture of cigarettes and smoking behavior could explain some of the discrepancies in trend. Unfortunately, data on changes in cigarette composition are not readily available.

In each of the analyses by the group members, models of carcinogenesis were used to predict lung cancer deaths by smoking history in the first stage. The *total* number of lung cancer deaths was then aggregated over smoking groups. A limitation of this approach is that it does not specifically distinguish how risks may have varied over time by cohort and among current, former and never smokers. Without imposing structure through assumptions, different cohort and period effects cannot be estimated in smoking sub-groups because of problems of statistical identification. The FHCRC group fit period and cohort effects to smokers alone and to smokers and ex-smokers separately. Judged by the Akaike information Criterion ⁽¹⁰⁾, the separate fit of period and cohort effects to smokers and ex-smokers separately produced only a small amount of fit improvement, which did not justify the added complexity of the model.

Finally, uncertainty remains with respect to the models themselves, as indicated by the different models of the different groups. In a close collaboration, it might be expected that a consensus would be reached on a best model design, but such a consensus was not reached. The models of this collaboration include numerous design differences. Several of the differences are associated with a general design plan of the model that was determined before starting to answer the question of this monograph. Other differences could be implemented in different types of model, many of which were described above. In some instances, discussion between the different groups led to relatively broad agreement. For example, five out of six groups eventually used a TSCE type model for the dose response relationships. However, often consensus was not reached. An important reason for this lack of consensus on model implementation is the high degree of uncertainty behind the disease processes. It is our opinion that a lack of consensus in this collaborative group did not reflect doctrinaire attitudes towards each other's approach but, instead, reflected uncertainties that probably would not have been revealed without this collaborative effort. Undoubtedly, this comparative modeling covers aspects of uncertainty that cannot be revealed by a sensitivity analysis involving parameter values of any one of the models. Nevertheless, there was good agreement among the results of the different models, which suggests that the resulting conclusions are robust.

Finally, the modeling described here only investigated the relationship between smoking and lung cancer. Lung cancer comprises about one-third of the deaths attributable to smoking, with substantial contributions also from heart disease, stroke and chronic obstructive pulmonary disease. While the role of competing risk factors makes it more difficult to validate the role of smoking for these other diseases (especially heart disease and stroke) in the manner used by CISNET modelers, the findings here suggest that the role of smoking as a risk factor for these non-lung cancer diseases may be underestimated.

4.3. Potential Extensions

Several extension of the analysis may be of interest. While the base case analysis does not distinguish types of lung cancer, four major histopathologic types of lung cancer, all related to smoking, are recognized and the risks associated with smoking may vary by histologic sub-type ^(11–12). The epidemiologic evidence indicates that the incidence of adenocarcinoma of the lung, in both men and women, increased over the last half of the twentieth century and that this increase was real and not attributable to changes in diagnostic criteria ^(13–15). However, based on analyses of the SEER data, Chen et al. ⁽¹⁵⁾ reported a decline in adenocarcinoma rates between 1999 and 2003 in both men and women. Second, the base case analysis does not distinguish racial-ethnic differences. Some previous studies indicate

important differences in lung cancer rates and the role of smoking by race⁽¹⁶⁾. Third, the Smoking Base Case analysis examined the effect on lung cancer deaths of tobacco control policies implemented at the time of the Surgeon General's Report in 1964. Smoking rates in the absence of tobacco control were obtained by extrapolating the smoking rates from before the Report to obtain the smoking rates that would have occurred in the absence of the tobacco control. They did not consider the role of specific tobacco control policies. The models can be extended to include histology-specific dose effect relationships, race/ethnicity-specific smoking histories, and/or specific tobacco control measures in order to study their effects on lung cancer risk in the population.

Another extension would be to consider other models relating lung cancer rates to smoking intensity and duration. All, but one of the models used the TSCSE model. As reported in chapter 13, the PIRE and Yale groups jointly estimated lung cancer rates using models by Knoke et al.⁽¹⁷⁾ and Flanders et al.⁽¹⁸⁾. Before calibration, the results were quite similar to those using the TSCE carcinogenesis model. However, while the TSCE and Knoke models exhibit very similar estimates of overall rates, the separate contributions by smoking category are quite different. The Knoke model implies that former smokers' risk returns much more quickly to never smokers than TSCE I.

Another potential extension would be to extend the models past the year 2000. Smoking data from the National Health Interview Survey and data from the National Center for Health Statistics on lung cancer deaths are now available through 2010, making it possible to extend the analysis of the effect of past policies through the year 2010. In addition, this additional data may be used to validate the models.

4.4. Summary

In sum, the model based evaluation of this study combined data on smoking histories in the US population, obtained from epidemiological studies and from surveillance of trends in lung cancer risk, in order to estimate the effects of tobacco control on lung cancer risk. The availability of a unique set of data on smoking prevalence has enabled the CISNET lung group to consider the effects of smoking over a longer time period and a larger population than previous studies. However, the longitudinal data employed to estimate risks is limited select years and a non-representative population, indicating that previous estimates of relative risk may be understated. Our study shows that changes in smoking habits led to a substantial reduction in the lung cancer mortality that would not have been expected had the smoking trends in the 1950s continued into the future. As consistently established by the different models, the proportion of potentially avoidable deaths that were actually avoided is large; less than half of the potential reduction in lung cancer deaths by the year 2000 have yet been realized. Increased tobacco control efforts will be needed to further slow lung cancer death rates.

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Figure 1. Shared process flow used by all models. Population and smoking inputs were used to develop the smoking history generator, which, in turn, simulates detailed individual-level smoking and other-cause mortality histories. These individual histories are used by each of the modeling groups to generate lung cancer mortality rates in the population.

**U.S. Men Current-Smokers
by 10-Year Birth Cohort (1900-1960)**

**U.S. Women Current-Smokers
by 10-Year Birth Cohort (1900-1960)**

No Tobacco Control

No Tobacco Control

Complete Tobacco Control

Complete Tobacco Control

Figure 2.

US Population percentage of current smokers by gender and birth cohort for three different tobacco control scenarios. This is one of the outputs that can be generated from the smoking history generator. The output from the actual tobacco control scenario describes the observed data well (not shown).

Figure 3. Lung cancer mortality rates, standardized to the 2000 US standard population, and crude counts for tobacco control scenarios as predicted by the Yale model.

Figure 4.

Comparison of model results for actual and potential cumulative lung cancer deaths avoided during the period 1975–2000, shown on equal scales.

E = Erasmus MC; F = Fred Hutchinson Cancer Research Center; M = Massachusetts General Hospital - Harvard Medical School; P = Pacific Institute for Research and Evaluation; R = Rice University-MD Anderson Cancer Center; Y = Yale University

Figure 5. Observed numbers of lung cancer deaths and hypothetical numbers of lung cancer deaths in a population with only never smokers from the Yale model.

Table I

Key model attributes.

	Erasmus MC (Miscan)	FHCRC	MGH-HMS	PIRE	Rice-MDA	Yale
Original motivation for model development	Screening evaluation with risk factors	Analysis of Epidemiological data	Screening evaluation	Policy evaluation	Mortality risk due to smoking and suboptimal DNA repair	Population trend effects on lung cancer rates
Unit of analysis	Individual	Individual	Individual	Group	Individual	Group
Central Dose Response Model	TSCE	TSCE	Probabilistic/Logistic Regressions	TSCE	TSCE	TSCE
Central Module parameter estimation source	HPFS (male incidence) NHS (female incidence) SEER (survival)	HPFS (male mortality), NHS (female mortality)	SEER (incidence, survival)	CPS II (mortality)	MDA CCS (smoking histories for fitting TSCE) CPS II (male mortality rates) NHS (female mortality rates)	HPFS (male mortality), NHS (female mortality)
Other Intermediate Outputs	Onset, Clinical Diagnosis Survival	Onset, Clinical Diagnosis, Treatment Specific Survival				
Lung Cancer Outcome Calibration Target		US Mortality	US Mortality			US Mortality

Abbreviations: TSCE=Two-stage clonal expansion model; SEER=Surveillance, Epidemiology and End Results; HPFS=Health Professionals' Follow-up Study; NHS=Nurses' Health Study; CPS II=Cancer Prevention Study II; FHCRC=Fred Hutchinson Cancer Research Center; MGH-HMS=Massachusetts General Hospital - Harvard Medical School; PIRE= Pacific Institute for Research and Evaluation; MDA=MD Anderson Cancer Center

Table II

Realized and potential reductions in lung cancer mortality from changes in smoking behavior.

Realized fraction of potential benefit from tobacco control, by year(s)	Women			Men			Overall		
	Realized (NTC-ATC)	Potential (NTC-CTC)	Fraction Realized	Realized (NTC-ATC)	Potential (NTC-CTC)	Fraction Realized	Realized (NTC-ATC)	Potential (NTC-CTC)	Fraction Realized
1975–2000									
ERASMUS MC	201,788	806,320	0.25	658,529	1,757,857	0.37	860,317	2,564,177	0.34
FHCRC	202,817	862,610	0.24	508,777	1,680,867	0.30	711,594	2,543,477	0.28
MGH-HMS	214,830	854,112	0.25	487,263	1,597,733	0.30	702,092	2,451,845	0.29
PIRE	333,976	1,064,443	0.31	454,517	1,329,972	0.34	788,493	2,394,415	0.33
RICE-MDA	285,079	878,359	0.32	603,236	1,645,651	0.37	888,316	2,524,010	0.35
YALE	221,173	834,293	0.27	603,122	1,712,035	0.35	824,294	2,546,328	0.32
mean	243,277	883,356	0.27	552,574	1,620,686	0.34	795,851	2,504,042	0.32
1991–2000									
ERASMUS MC	143,273	462,528	0.31	384,882	834,310	0.46	528,155	1,296,837	0.41
FHCRC	152,574	521,040	0.29	318,279	842,602	0.38	470,853	1,363,642	0.35
MGH- HMS	153,549	511,509	0.30	310,210	846,300	0.37	463,759	1,357,809	0.34
PIRE	253,711	687,156	0.37	342,558	865,306	0.40	596,269	1,552,462	0.38
RICE-MDA	185,782	461,559	0.40	346,266	785,168	0.44	532,048	1,246,727	0.43
YALE	157,388	507,085	0.31	366,815	871,273	0.42	524,203	1,378,358	0.38
mean	174,380	525,146	0.33	344,835	840,826	0.41	519,214	1,365,973	0.38
The year 2000									
ERASMUS MC	20,277	55,337	0.37	48,897	94,979	0.51	69,173	150,316	0.46
FHCRC	22,271	63,373	0.35	39,076	92,434	0.42	61,347	155,807	0.39
MGH-HMS	21,532	60,774	0.35	38,375	92,187	0.42	59,907	152,961	0.39
PIRE	40,496	90,001	0.45	50,943	110,800	0.46	91,439	200,802	0.46
RICE-MDA	28,365	55,988	0.51	42,351	86,863	0.49	70,716	142,851	0.50
YALE	23,559	62,628	0.38	45,165	96,794	0.47	68,723	159,422	0.43
mean	26,083	64,684	0.40	44,135	95,676	0.46	70,218	160,360	0.44