

Chapter 7

The Impact of Smoking on Disease and the Benefits of Smoking Reduction

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Overview

The preceding chapters have reviewed the extensive scientific evidence regarding the diverse illnesses caused by tobacco use. The causation of multiple diseases by smoking and the related loss of life expectancy have long motivated policy actions to control tobacco use. To support policy actions and decision making based on the health evidence, quantitative estimates of the burden of disease associated with smoking in the population are made. These numbers complement the epidemiologic studies that estimate the risks to individuals associated with various smoking patterns.

This chapter reviews methods used to estimate the burden of disease attributable to smoking and provides updated estimates of this burden. The chapter is

limited to consideration of risks from cigarette smoking and does not include those attributable to smokeless tobacco use, cigar smoking, or other forms of tobacco use. It considers methodologies and data sets used to estimate disease burden, summarizes past reports and critiques of smoking attributable disease estimates, presents current estimates of smoking attributable mortality for the nation and for individual states, and reviews estimates of the economic costs of illness attributable to smoking. Data are also presented on the reduction of mortality achievable nationwide by meeting the *Healthy People 2010* prevalence objectives for reducing smoking (U.S. Department of Health and Human Services [USDHHS] 2000).

Introduction

For diseases attributable to a causal risk factor, such as smoking, the “disease burden” associated with that risk factor can be estimated for a particular population using epidemiologic methods. Different types of estimates can be made, such as mortality, morbidity, disability-adjusted life years (DALYs) lost, changes in disability-adjusted life expectancy (DALE), quality-adjusted life years (QALYs) lost, years of potential life lost (YPLL), economic costs of illness, and population attributable risk (PAR) (Table 7.1). In 1996, the World Health Organization (WHO) published the landmark document *The Global Burden of Disease* (Murray and Lopez 1996), which used mortality and DALYs to describe the burden of disease associated with major risk factors for each country in 1990. Updated estimates were published in 2002 (Ezzati et al. 2002). A key goal of these efforts is to clearly link these burden-of-disease measurements to health policy decision making. The 1996 WHO report included the following rationales for estimating disease burden:

1. Assessing the performance of a health care system with respect to actual health outcomes.

2. Generating a forum for an informed debate of values and priorities.
3. Identifying national disease-control priorities.
4. Allocating training for clinical and public health practitioners according to priority illnesses.
5. Allocating research and development resources to address major disease burdens.
6. Allocating resources across health interventions in order to shift resources to the most cost-effective approaches for prevention.

This chapter focuses on the main measure of disease burden used to assess the impact of smoking in the United States, the PAR. The calculation of the PAR for a particular risk factor represents a form of quantitative risk assessment (National Research Council 1983), a systematic approach that translates research

Table 7.1 Disease burden measures used to evaluate the impact of population risk factors

Measure	Data elements	Use
Mortality	Information provided by death certificates on specific causes of death	Describes disease (death) according to age, gender, race, and other demographic factors for specific diagnoses and certain antecedent conditions
Morbidity	Information on hospitalizations, outpatient treatments, prescription drugs, nursing home admissions, other medical care	Describes the disability, costs, and medical care utilization related to specific diagnoses
Disability-adjusted life years (DALYs)*	Standard life table data, disability-adjusted ages at death, discounted contribution of years of life lost	Estimates a single measure of disease burden for comparisons across populations
Quality-adjusted life years (QALYs)	Arithmetic product of the life expectancy and the quality of the remaining years; quality of additional life was assessed by questionnaires or preference studies	Estimates the extra quantity and quality of life provided by an intervention combined within a single measure
Disability-adjusted life expectancy (DALE) [†]	Standard life table data, survey data on physical and cognitive disabilities and general health status	Determines the maximum level of health expected within the surveyed health care system
Years of potential life lost (YPLL) [‡]	Mortality data and life expectancy at the time (age) of death	Estimates the burden of premature death in a given population
Economic costs of illness	Costs of specific medical services, data on utilization of services by specific population groups, rates of utilization according to risk factors	Estimates the costs of illness attributable to a specific risk factor for a given population group
Population attributable risk (PAR)	Mortality data, life expectancy at death, relative risk of death according to risk factor prevalence	Estimates the proportion of deaths attributable to a specific risk factor in a given population
Smoking attributable fractions (SAFs)	Smoking prevalence data by smoking status, age, and gender; and relative risk of death for smoking-related diseases by age and gender	Estimates the proportion of an outcome that could be avoided if smoking were eliminated

*Includes life years lost to premature mortality and years lived with disability. For a comprehensive discussion of DALYs, see Murray and Lopez 1996, *The Global Burden of Disease*.

[†]Life expectancy was adjusted to account for disability and is simply premature mortality. For a comprehensive discussion of DALE, see Murray and Lopez 1996, *The Global Burden of Disease*.

[‡]YPLL is usually calculated from age at death to age 65 years, 85 years, or life expectancy.

Table 7.1 Continued

Measure	Data elements	Use
Smoking attributable mortality (SAM)	Mortality data for smoking-related diseases by age and gender; smoking prevalence data by smoking status, age, and gender; relative risk of death for smoking-related diseases by age and gender	Estimates the number of deaths that could be avoided if smoking were eliminated

Source: Murray and Lopez 1996.

findings for the purpose of guiding the implementation and evaluation of policies (Samet and Burke 1998). The elements of a risk assessment include hazard identification (e.g., does smoking cause disease[s]?), exposure assessment (e.g., what is the population pattern of smoking?), dose-response assessment (e.g., how does risk vary with duration and amount of smoking?), and risk characterization (e.g., what is the disease burden caused by smoking?). The PAR is estimated for a particular disease based on the conclusion that smoking causes the disease, an assumption equivalent to the hazard identification component of risk assessment. The PAR calculation incorporates the prevalence of smoking, analogous to exposure assessment, and the relative risk (RR) associated with various amounts of smoking, analogous to dose-response assessment. The PAR itself characterizes risk, and uncertainties associated with the PAR estimates can be described.

In applying this approach to smoking, researchers first evaluate epidemiologic and other evidence for causality for a particular disease or effect, as described in Chapter 1 of this report. Large cohort studies, such as the Cancer Prevention Study I (CPS-I) and Cancer Prevention Study II (CPS-II) of the American Cancer Society (ACS) (Stellman and Garfinkel 1986), the U.S. Veterans Study (Kahn 1966), and the British Doctors Study (Doll and Peto 1976; Doll et al. 1994), provide robust RR estimates for current smokers and former smokers, compared with lifetime nonsmokers, for major causes of death. Population exposures to smoking are measured using survey data, biologic markers, or proxy information from relatives of decedents. For the United States, large population-based surveys of tobacco use provide uniform and consistent assessments of the prevalence of current and former smoking. Finally, the RRs and the smoking prevalence data are then combined to estimate the PAR, the proportion of deaths attributable to the exposure.

In addition, public health decision makers consider estimates of the population disease burden in terms of the number of deaths caused by exposure to smoking and the burden of premature deaths, which can be expressed as YPLL. YPLL can be calculated from the age at death up to specific ages or to full life expectancy. By making the calculation to specific ages, YPLL can be estimated at younger, middle, and older ages.

Measuring changes in smoking attributable mortality (SAM) over time provides a periodic ongoing indication of the burden of disease caused by tobacco use. This information can be used to guide national and state comprehensive tobacco control programs, facilitating decisions on resource allocation and needs by comparing the impact of tobacco use with other risk factor disease burdens (McGinnis and Foege 1993).

An appendix to this chapter reviews the methods used to estimate the burden of smoking along with previous SAM estimates in the United States. The appendix also describes the databases used for these calculations. The chapter includes new annual SAM and YPLL estimates for 1995–1999; state-specific, age-adjusted SAM; total SAM for 1964 (the year of the first Surgeon General’s report on the health consequences of smoking and health) through 1999; and estimates of SAM that could be avoided by meeting the *Healthy People 2010* objectives for the nation (USDHHS 2000).

To summarize, the overall approach to estimating SAM includes the following:

- Identifying those diseases caused by (cigarette) smoking.
- Developing RR estimates for these diseases for current and former smokers, compared with lifetime nonsmokers; the currently used estimates are for CPS-II follow-up from 1982–1986.

- Developing estimates of smoking prevalence for the nation and the states using National Health Interview Survey (NHIS) data for the years of interest.
- Estimating the disease- and gender-specific PARs.
- Applying the PARs to the disease-specific mortality counts to estimate the SAM.

This listing makes the critical assumptions clear and acknowledges the cross-sectional nature of the SAM estimates, which are not for particular birth

cohorts but for particular time points. They are representations of the SAM for a population with the smoking prevalence profile of a particular year, on the assumption that the population would experience the selected RR estimates across its full life span. The calculations thus refer to theoretical, nonexistent populations, albeit based in actual data, but the same methodology is applied uniformly over time, yielding estimates that are informative about relative changes in SAM over time. The estimates are useful for indicating the general scope of the public health burden from smoking.

Current Impact of Smoking

Smoking Attributable Mortality and Years of Potential Life Lost

For this report, the annual SAM and YPLL calculations for 1995–1999 have been updated from the most recent Centers for Disease Control and Prevention (CDC) report (CDC 2002a) by using the additional diseases now causally attributed to smoking (stomach cancer and acute myeloid leukemia), using new estimates for perinatal RRs, and excluding hypertension, which was previously included as a cause of smoking-related deaths on the assumption that smoking attributable heart disease deaths were included in this category. These estimates include adult and perinatal deaths for 19 disease categories among adults and 4 adverse infant health outcomes (also listed in the tenth revision of the *International Classification of Diseases [ICD-10]* [CDC 2002b,d]) that are caused by smoking (see Appendix 7-1). Deaths attributable to residential fires caused by smoking (589 males and 377 females [Hall 2001]) and deaths from secondhand smoke exposure for adults are also included (nationally, 3,000 for lung cancer and 35,000 to 62,000 for heart disease [National Cancer Institute (NCI) 1999; CDC 2002d; International Agency for Research on Cancer (IARC) 2002]).

Relative risks for smoking-related diseases and smoking prevalence estimates for current and former smokers 35 years of age and older and for maternal smokers were used to calculate smoking attributable fractions (SAFs) and SAMs as in the previous CDC report (2002a). Age-adjusted RR data were obtained

from CPS-II (1982–1988, see Appendix 7-1), and gender-specific smoking prevalence data for adults aged 35 years and older were obtained from NHIS (Table 7.2). Relative risk estimates of the deaths of infants whose mothers smoked during pregnancy were obtained from McIntosh (1984) and Gavin and colleagues (2001). Maternal smoking prevalence data from most states for 1995–1999 were obtained from birth certificates (see <http://www.cdc.gov/nchs/births.htm>). Age- and gender-specific mortality data were obtained from National Center for Health Statistics (NCHS) reports (Hoyert et al. 2001). YPLL for persons aged 35 years and older were calculated using remaining life expectancy (life expectancy at any given age of death minus age at death and for infants, from birth). SAM and YPLL include nationally reported deaths from cigarette-caused residential fires; SAM includes lung cancer and heart disease deaths from secondhand smoke exposures (15,500 men and 22,500 women [NCI 1999]).

Smoking caused an estimated total of 263,600 deaths in males and 176,500 deaths in females (total 440,100) in the United States each year from 1995–1999 (Table 7.3). For men aged 35 years and older, annual smoking attributable deaths were 105,700 for cancers, 87,600 for cardiovascular diseases (CVDs), and 53,700 for respiratory diseases. For women aged 35 years and older, the annual SAM was 53,900 for cancers, 55,000 for CVDs, and 44,300 for respiratory diseases. Among adults, the most smoking attributable deaths were from lung cancer (124,800), ischemic heart disease (IHD) (82,000), and chronic airways obstruction (64,700).

Table 7.2 Annual prevalence of current smoking and former smoking among adults aged 35 years and older, selected years, National Health Interview Survey, United States, 1965–1999

Year	Men						Women					
	35–44 years		45–64 years		≥65 years		35–44 years		45–64 years		≥65 years	
	CS*	FS†	CS	FS	CS	FS	CS	FS	CS	FS	CS	FS
1965	54.3	22.8	54.3	22.8	36.4	21.5	36.5	9.0	36.5	9.0	9.6	4.5
1970	49.8	27.0	44.7	32.2	23.4	39.2	39.2	14.1	32.5	12.2	10.9	7.3
1974	51.4	26.9	42.7	36.5	24.7	41.6	39.7	14.4	33.4	14.8	12.1	10.8
1977	48.5	25.5	40.5	35.2	23.3	43.5	38.6	15.1	34.4	15.3	13.5	12.3
1980	42.6	27.8	40.6	37.2	17.8	47.8	34.9	18.9	30.6	17.2	17.1	14.4
1983	40.4	28.0	35.4	40.4	21.4	48.4	33.8	17.1	30.6	18.7	13.0	18.6
1985	39.0	30.6	34.4	41.5	19.9	51.8	33.4	19.2	31.4	21.3	14.2	20.3
1987	37.4	27.4	34.8	39.0	18.8	52.0	30.8	18.5	29.8	20.9	13.6	19.3
1988	37.2	26.0	33.4	40.7	18.8	52.9	29.0	18.7	29.0	24.3	13.4	20.7
1990	35.2	26.1	31.2	41.0	14.6	55.2	26.5	19.7	26.1	24.4	11.5	23.2
1992	32.9	26.2	30.6	40.5	16.2	54.0	28.5	18.3	26.8	23.8	12.4	24.0
1994	30.6	34.4	30.6	34.4	13.3	58.3	24.6	23.5	24.6	23.5	11.1	26.9
1995	29.1	31.4	29.1	31.4	14.9	52.9	25.4	21.9	25.4	21.9	11.5	26.8
1996	29.4	30.5	29.4	30.5	13.5	55.1	24.5	22.1	24.5	22.1	11.5	26.1
1997	29.6	30.1	29.6	30.1	12.8	56.2	24.0	22.1	24.0	22.1	11.5	25.8
1998	28.8	29.9	28.8	29.9	10.4	58.5	24.2	21.2	24.2	21.2	11.2	27.0
1999	27.6	29.5	27.6	29.5	10.5	57.9	23.3	21.7	23.3	21.7	10.7	27.8

*CS = Current smokers, defined as having smoked at least 100 cigarettes and currently smoked every day or some days (the some days condition was added in 1992).

†FS = Former smokers, defined as having smoked at least 100 cigarettes but not currently smoking.

Sources: National Center for Health Statistics, public use data tapes, 1965, 1970, 1974, 1977, 1980, 1983, 1985, 1987, 1988, 1990, 1992, 1994, 1995, 1996, 1997, 1998, 1999.

Smoking during pregnancy was estimated to result in 560 deaths in infant boys and 410 deaths in infant girls annually. Excluding adult deaths from second-hand smoke, the estimated SAM was responsible for a total annual YPLL of 3,319,000 for males and 2,152,600 for females.

The annual SAM will likely remain fairly stable if trends in smoking prevalence among adults do not decrease substantially. Adult smoking prevalence rates have decreased over the past few years (Table 7.2) (CDC 1999a, 2001a), but the prevalence of smoking among adolescents increased from 1992 until 1997. However, youth smoking has also decreased more recently (CDC 2002f). Yet, the burden of disease attributable to smoking is driven by those with long-term previous exposures, so unless smoking cessation among current smokers increases quite rapidly, SAM is not expected to decline substantially for many years. Estimates of various SAM projections under several scenarios of prevalence rate reductions are presented later in this chapter.

Total Smoking Attributable Mortality, 1965–1999

The total SAM estimates for 1965–1999 were derived from annual PAR estimates for the time since the publication of the first Surgeon General's report on the health consequences of smoking in 1964 (Table 7.4). The PARs for each of 19 smoking-related disease categories were calculated using smoking prevalence and the RR estimates for mortality for current and former smokers aged 35 years and older. The PARs for each of four adverse health outcomes were calculated using maternal smoking prevalence and RR estimates for smoking-related infant deaths. The mortality RR estimates for adults were obtained from both CPS-I and CPS-II data (see Appendix 7-1). CPS-I data (1959–1965) were used in conjunction with NHIS smoking prevalence data from 1965–1971, CPS-II data (1982–1988) were applied to NHIS prevalence data from 1982–1999, and the midpoint RRs between CPS-I and

Table 7.3 Annual deaths, smoking attributable mortality (SAM), and years of potential life lost (YPLL), stratified by cause of death and gender, United States, 1995–1999

Disease category (ICD-9 code)*	Males			Females		
	Total deaths	SAM	YPLL	Total deaths	SAM	YPLL
Neoplasms†						
Lip, oral cavity, pharynx (140–149)	5,200	3,900	64,000	2,600	1,300	20,600
Esophagus (150)	8,600	6,300	94,400	2,800	1,600	24,300
Stomach (151)	7,600	2,200	30,000	5,300	600	9,200
Pancreas (157)	13,400	3,100	46,100	14,300	3,400	49,800
Larynx (161)	3,000	2,500	37,800	800	600	10,300
Trachea, bronchus, lung (162)	91,300	80,600	1,106,100	61,600	44,200	719,900
Cervix uteri (180)	NA‡	NA	NA	4,100	500	13,400
Urinary bladder (188)	7,800	3,700	40,200	3,800	1,100	12,500
Kidney, other urinary (189)	7,100	2,800	41,900	4,500	200	4,000
Acute myeloid leukemia (205.0)	3,200	800	11,000	2,700	300	4,600
Total	147,200	105,700	1,471,400	102,700	53,900	868,700
Cardiovascular diseases†						
Ischemic heart disease (410–414)						
Aged 35–64 years	53,000	22,100	514,900	19,400	7,100	185,600
Aged 65 years	191,200	29,300	252,400	218,000	23,500	207,200
Other heart disease (390–398, 415–417, 420–429)	98,100	18,800	243,300	117,600	10,500	122,900
Cerebrovascular disease (430–438)						
Aged 35–64 years	9,700	3,900	93,900	8,100	3,600	101,500
Aged 65 years	51,400	4,700	37,800	88,500	5,300	45,000
Atherosclerosis (440)	9,000	1,600	14,900	10,100	900	7,700
Aortic aneurysm (441)	10,000	6,500	76,600	6,200	3,100	37,200
Other arterial disease (442–448)	4,700	700	8,500	6,200	900	11,800
Total	424,000	87,600	1,242,300	474,000	55,000	718,900
Respiratory diseases†						
Pneumonia, influenza (480–487)	38,300	8,800	84,900	47,400	6,800	69,100
Bronchitis, emphysema (490–492)	10,900	9,900	109,000	9,600	7,800	99,800
Chronic airways obstruction (496)	42,800	34,900	353,100	39,700	29,800	353,300
Total	92,000	53,700	547,000	96,700	44,300	522,200
Perinatal conditions†						
Short gestation/low birth weight (765)	2,200	220	15,970	1,770	180	13,870
Respiratory distress syndrome (769)	930	40	2,600	640	20	1,930
Other respiratory conditions in newborns (770)	910	50	3,460	650	30	2,650
Sudden infant death syndrome (798.0)	1,770	260	18,940	1,200	180	13,870
Total	5,810	560	40,960	4,250	410	32,310

Note: All figures are rounded and hence do not add up.

*International Classification of Diseases, 9th Revision.

†Among persons aged ≥ 35 years.

‡NA = Not applicable.

§NR = Data were not reported.

Table 7.3 Continued

Disease category (ICD-9 code)	Males			Females		
	Total deaths	SAM	YPLL	Total deaths	SAM	YPLL
Burn deaths	NA	590	17,300	NA	380	10,500
Secondhand smoke deaths						
Lung cancer	NR ¹	1,100	NR	NR	1,900	NR
Ischemic heart disease	NR	14,400	NR	NR	20,600	NR
Total		15,500			22,500	
Overall total	669,100	263,600	3,319,000	677,600	176,500	2,152,600
Grand total	Males and females					
SAM	440,100					
YPLL	5,466,600					

Sources: McIntosh 1984; U.S. Department of Health and Human Services 1989b; National Center for Health Statistics, public use data tapes, 1995–1999; Thun et al. 1997b; National Cancer Institute 1999; Gavin et al. 2001; Hall 2001; Hoyert et al. 2001; Mathews 2001; Centers for Disease Control and Prevention 2002a,b,d; International Agency for Research on Cancer 2002; American Cancer Society, unpublished data.

CPS-II were used with NHIS prevalence data for 1972–1981, applied to each year's mortality data during that period. Current and former smoking prevalence data, by gender and for ages 35 through 44 years, 45 through 64 years, and 65 years and older, were obtained from NHIS (Table 7.2). Linear extrapolation was used to estimate prevalence in the years that surveys were not conducted. Data on maternal smoking status for earlier years were extrapolated using the ratio of maternal smoking prevalence to current smoking prevalence among women aged 18 through 24 years from 1995–1999. These data produced more conservative prevalence estimates than smoking rates among women of childbearing age (18 through 44 years).

SAM estimates were calculated by multiplying each cause-specific SAF by the total number of annual deaths for each smoking-related disease. To compare mortality data across differing ICD code systems, data for 1965–1967 (ICD-7), 1968–1978 (ICD-8), and 1999 (ICD-10) were translated into ICD-9 codes using comparability ratios¹ obtained from NCHS (Klebbba 1975; Anderson et al. 2001) (also see Appendix 7-1).

From 1965–1999, smoking has caused an estimated 4.1 million cancer deaths, 5.5 million CVD deaths, 2.1 million respiratory disease deaths, 94,000 infant deaths, and 11.9 million deaths total (Table 7.4). Excluding deaths from fires and exposures to secondhand smoke, approximately 350,000 persons in the United States have died each year from 1965–1999 because of smoking. Since 1995, annual deaths in the United States that were caused by smoking increased to more than 440,000 (Table 7.3).

Despite the methodologic variability in estimation techniques over the years, cigarette smoking remains the leading cause of preventable mortality in the United States, resulting in nearly 16 million deaths since the first Surgeon General's report on smoking and health in 1964. These calculations do not reflect all determinants of the disease impact of smoking. First, as previously discussed, the reported SAM rates were derived from smoking rates in the current year, whereas actual smoking attributable deaths in the current year were the result of higher smoking rates in previous decades. The lower RRs for former

¹Comparability ratios measure the effect of changes in classification and coding rules between versions of the ICD. These ratios are derived by coding the same deaths by both ICD-10 and ICD-9 (for example) criteria separately, and then dividing the number of classified ICD-10 deaths by classified ICD-9 deaths.

Table 7.4 Smoking attributable mortality in the United States, 1965–1999, stratified by gender*

Disease category (ICD-9 code) [†]	Males	Females	Total
Neoplasms[‡]			
Lip, oral cavity, pharynx (140–149)	145,100	36,200	181,300
Esophagus (150)	151,000	38,500	189,500
Stomach (151)	97,000	14,400	111,300
Pancreas (157)	116,500	77,100	193,500
Larynx (161)	85,000	14,600	99,600
Trachea, bronchus, lung (162)	2,286,800	812,200	3,099,000
Cervix uteri (180)	NA [§]	18,000	18,000
Urinary bladder (188)	113,900	29,700	143,600
Kidney, other urinary (189)	74,700	8,200	82,900
Acute myeloid leukemia (205.0)	21,800	4,800	26,600
Total	3,091,600	1,053,700	4,145,400
Cardiovascular diseases[‡]			
Ischemic heart disease (410–414)			
Aged 35–64 years	1,302,400	335,700	1,638,100
Aged 65 years	1,214,800	646,100	1,860,900
Other heart disease (390–398, 415–417, 420–429)	608,300	253,800	862,100
Cerebrovascular disease (430–438)			
Aged 35–64 years	170,400	156,100	327,200
Aged 65 years	175,200	134,200	309,400
Atherosclerosis (440)	145,800	61,800	207,500
Aortic aneurysm (441)	203,300	75,100	278,500
Other arterial disease (442–448)	33,000	22,300	55,300
Total	3,853,200	1,685,800	5,539,000
Respiratory diseases[‡]			
Pneumonia, influenza (480–487)	287,300	127,100	414,400
Bronchitis, emphysema (490–492)	459,000	169,800	628,800
Chronic airways obstruction (496)	694,400	419,000	1,113,400
Total	1,440,700	715,800	2,156,500
Perinatal conditions			
Short gestation/low birth weight (765)	16,700	13,300	29,900
Respiratory distress syndrome (769)	10,800	6,700	17,500
Other respiratory conditions in newborns (770)	20,600	15,400	36,000
Sudden infant death syndrome (798.0)	6,140	4,800	10,900
Total	54,200	40,200	94,400
All conditions	8,439,700	3,495,500	11,935,200

Note: All figures are rounded and hence do not add up.

*Estimates exclude deaths from residential fires caused by smoking and deaths from secondhand smoke exposure.

[†]International Classification of Diseases, 9th Revision.

[‡]Among persons aged ≥ 35 years.

[§]NA = Not applicable.

Sources: National Center for Health Statistics, public use data tapes, 1965–1999; Klebba 1975; Klebba and Scott 1980; McIntosh 1984; U.S. Department of Health and Human Services 1989b; Thun et al. 1997b; Gavin et al. 2001; American Cancer Society, unpublished data.

smokers may not fully capture their risks from past smoking behaviors because they may have quit very recently and thus have RRs similar to long-term current smokers (CDC 1993). Second, the RR estimates were restricted to adults aged 35 years and older based on available CPS-I and CPS-II data, and thus may exclude risks for death in earlier ages. Third, the RRs were adjusted for the effects of age but not for other potential confounders. As described in Appendix 7-1, there was little additional impact on the SAM estimates for lung cancer, chronic airways obstruction, IHD, and cerebrovascular disease when the effects of education, alcohol, and other confounders were included (Malarcher et al. 2000; Thun et al. 2000). Fourth, deaths from cigar smoking, pipe smoking, and smokeless tobacco use were not included, nor were deaths from fires and secondhand smoke.

1999 State Smoking Attributable Mortality Estimates

Four sets of data are necessary to calculate SAM and SAM rates per 100,000 population for each state (Nelson et al. 1994): (1) state-specific smoking prevalence, (2) mortality (number of deaths), (3) demographic data that are available for all states and for some large municipalities, and (4) national RR estimates—those from CPS-II (CDC 2002d). State-specific smoking prevalence data are available for states that conducted the telephone-based Behavioral Risk Factor Surveillance System (BRFSS) survey supported by CDC. By 1995, all 50 states conducted the BRFSS (CDC 1996b). Mortality data were obtained from vital statistics registries (Hoyert et al. 2001).

Total SAM was approximately 398,000 (ranging from 460 in Alaska to 38,050 in California) (Table 7.5). The 50-state SAM total (397,640) differs somewhat from the average annual national total reported in the previous section (440,200) for several reasons. First, state-specific prevalence estimates from BRFSS data that were used in the PAR calculation are somewhat lower than those from the NHIS data used in national estimates (CDC 2001c, 2002c). Second, cigarette-caused fire deaths, secondhand smoke deaths, and deaths attributable to stomach cancer and myeloid leukemia are not included in each state SAM estimate. Third, California, with the largest state population, has the second-to-lowest smoking prevalence and associated lower mortality rates for many smoking-related diseases of those found in most other states; thus, California weighs down the national SAM total.

The average age-adjusted SAM rate per 100,000 persons was 289.5 (ranging from 156.6 per 100,000 in Utah to 398.8 per 100,000 in Nevada) (Table 7.6). These rates reflect, in part, differences in smoking prevalence and in population and mortality distributions among states. In general, lower SAM rates are found in states with lower rates of smoking.

Smoking Attributable Economic Costs

Economic Cost-of-Illness Measures

Measuring the economic costs of smoking gives policymakers and the public an additional dimension for understanding the burden of disease caused by smoking. Until the early 1990s, only a few estimates of the cost of smoking had been made in the United States (Warner et al. 1999). Estimates of the costs of smoking received increased attention in the 1990s when the states were estimating damages for purposes of lawsuits. For instance, states then engaged in negotiations that led to the 1998 Master Settlement Agreement among 46 states, the District of Columbia, and five commonwealths and territories with the tobacco industry. Published studies on the medical costs of smoking have used a number of approaches to estimate costs, including PAR calculations (Shultz et al. 1991), model-based approaches (CDC 1994; Miller et al. 1998, 1999; Adams et al. 2002), incidence-based measures of present and future costs attributable to smoking (Hodgson 1992), indirect costs of human capital lost from disability and premature deaths, and net social costs (Manning et al. 1989; Herdman et al. 1993; Barendregt et al. 1997; Warner et al. 1999). These studies have produced a wide range of estimates, depending on methodologies, assumptions incorporated into models, data sets used, and other methodologic issues. One key issue is the comparison of the net versus the gross costs of smoking to society. Net costs would include consideration of the economic benefits of taxes, agricultural revenue, ancillary economic activity, and the “costs” of longer lives among nonsmokers that might offset the medical care costs of smokers or their lost productivity while they are alive (Warner 1987; Viscusi 1994; Barendregt et al. 1997; U.S. Department of the Treasury 1998). A thorough discussion of the various methodologies and results is beyond the scope of this chapter, but Warner and colleagues (1999), Chaloupka and Warner (2000), Lightwood and colleagues (2000), and Max (2001) have provided extensive reviews of these issues. The discussion that

Table 7.5 State annual smoking attributable mortality (SAM) estimates, selected causes of death, United States, 1999

State	Lung cancer*	Ischemic heart disease*	Cerebro-vascular diseases*	Chronic obstructive pulmonary disease*	Total SAM
Alabama	2,360	1,410	390	1,680	7,540
Alaska	150	90	20	110	460
Arizona	2,010	1,390	300	1,880	6,870
Arkansas	1,620	990	260	1,040	4,900
California	10,900	8,830	1,620	9,920	38,050
Colorado	1,090	750	170	1,410	4,300
Connecticut	1,440	1,030	190	1,080	4,810
Delaware	440	250	40	250	1,210
District of Columbia	230	150	40	110	690
Florida	9,260	6,340	1,020	7,000	28,610
Georgia	3,260	2,050	570	2,350	10,650
Hawaii	340	220	70	190	1,100
Idaho	400	300	70	430	1,510
Illinois	5,500	4,260	870	3,890	18,360
Indiana	3,230	2,140	470	2,350	10,260
Iowa	1,330	1,010	170	1,220	4,620
Kansas	1,160	690	160	1,010	3,920
Kentucky	2,480	1,590	330	1,830	7,780
Louisiana	2,170	1,360	310	1,200	6,350
Maine	660	400	80	580	2,140
Maryland	2,280	1,440	270	1,450	6,750
Massachusetts	2,870	1,620	300	2,150	9,020
Michigan	4,390	3,510	620	3,280	14,700
Minnesota	1,740	930	240	1,450	5,620
Mississippi	1,560	1,080	260	960	4,900
Missouri	2,990	2,370	450	2,370	10,220
Montana	420	220	50	440	1,440
Nebraska	720	400	100	690	2,450
Nevada	980	670	160	830	3,290
New Hampshire	530	340	60	460	1,690
New Jersey	3,560	2,350	380	2,270	10,760
New Mexico	510	440	90	650	2,120
New York	7,450	6,520	760	5,050	24,450
North Carolina	3,760	2,380	560	2,640	11,500
North Dakota	230	200	40	200	860
Ohio	5,840	4,160	750	4,470	18,860
Oklahoma	1,780	1,360	260	1,290	5,780
Oregon	1,520	850	250	1,330	4,970
Pennsylvania	6,200	4,240	730	4,540	19,770
Rhode Island	570	410	60	380	1,720

Note: All figures are rounded and hence do not add up.

*International Classification of Diseases, 9th Revision (ICD-9), codes 162, 410–414, 430–438, 490–492, and 496.

Table 7.5 Continued

State	Lung cancer	Ischemic heart disease	Cerebrovascular diseases	Chronic obstructive pulmonary disease	Total SAM
South Carolina	1,880	1,220	360	1,290	5,950
South Dakota	320	230	50	250	1,080
Tennessee	3,120	2,150	460	2,110	9,570
Texas	7,390	5,440	1,070	5,650	24,080
Utah	300	210	50	380	1,230
Vermont	270	150	30	220	820
Virginia	3,060	1,710	420	2,010	9,120
Washington	2,450	1,450	340	2,060	7,770
West Virginia	1,260	830	130	950	4,230
Wisconsin	2,190	1,670	400	1,760	7,830
Wyoming	190	120	30	260	740
Total					397,640

Sources: Thun et al. 1997b; Behavioral Risk Factor Surveillance System: Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion, Division of Adult and Community Health, public use data tape, 1999; Gavin et al. 2001; Hoyert et al. 2001; CDC 2002a,d,e; American Cancer Society, unpublished data.

follows includes a brief review of recently published findings.

In the United States, direct medical costs for the detection, treatment, and rehabilitation of persons with smoking attributable clinical diseases have been the primary outcome variable in the cost models. These smoking attributable costs have been consistently estimated at 6 to 8 percent of the total annual expenditures for health care, with an estimated upper bound as high as 14 percent (Warner et al. 1999). Indirect morbidity and mortality costs are defined as the costs for excess sickness and disability days for smoking-linked illnesses, as well as lost productivity due to premature death from the effect of smoking on longevity (Rice et al. 1985).

The earliest attempts to estimate national health care expenses date from around 1950, and the cost-of-illness methodology was formalized and upgraded by Rice and colleagues through multiple iterations during the last three decades (Cooper and Rice 1976; Hodgson and Kopstein 1984; Rice et al. 1985). In 1986, Rice and colleagues (1986) estimated costs for direct health care, including physician care, hospital care, pharmaceuticals, home health care, and nursing home care for broad disease categories including CVD, respiratory diseases, and cancers. Using ratios of hospital days and physician visits for ever smokers

compared with lifetime nonsmokers, these investigators estimated \$14.4 billion in 1984 direct medical care costs attributable to smoking from neoplastic, circulatory, and respiratory diseases only.

Rice and colleagues (1986) applied NHIS data for work-loss days, disability days, and the percentage of the population unable to work due to disabling illnesses or premature death in a similar fashion to the direct-cost method used to estimate smoking attributable indirect morbidity and mortality costs. Relative rates of disability and work-loss for ever smokers and lifetime nonsmokers were used to estimate the SAF of morbidity costs at \$7.4 billion in 1984. Indirect mortality costs, defined as the economic value of forfeited future earnings for persons who die prematurely from smoking-related causes (Herdman et al. 1993), were valued at \$16.8 billion in 1984. Thus, the total estimate of smoking attributable costs for 1984 was \$38.6 billion in 1980 dollars. Indirect costs are substantial and account for one-half to three-quarters of total costs, with mortality alone accounting for 40 to 66 percent of total costs (Max 2001).

The Office of Technology Assessment (OTA 1985) calculated smoking attributable costs using the same method as Doll and Peto (1981), applying attributable mortality to CPS-I data from the 1960s and 1970s. OTA staff consulted with an expert committee of health

Table 7.6 State age-adjusted smoking attributable mortality (SAM) rates per 100,000 persons, selected causes of death, United States, 1999

State	Lung cancer*	Ischemic heart disease*	Cerebrovascular diseases*	Chronic obstructive pulmonary disease*	Total SAM
Alabama	104.3	63.1	17.3	75.5	336.5
Alaska	84.7	46.0	16.1	83.9	288.2
Arizona	81.3	61.4	12.4	74.7	286.1
Arkansas	113.2	70.3	18.4	72.3	342.1
California	73.3	60.0	10.9	67.6	257.0
Colorado	61.5	41.1	9.1	84.6	247.0
Connecticut	78.6	54.8	9.8	55.5	255.3
Delaware	113.0	67.8	10.9	66.6	317.1
District of Columbia	82.4	52.1	12.7	39.2	245.5
Florida	91.9	64.2	10.8	65.6	278.4
Georgia	101.4	63.5	17.5	77.6	335.0
Hawaii	51.9	33.7	10.4	28.7	167.8
Idaho	66.4	48.5	11.5	71.6	247.7
Illinois	91.5	69.9	14.2	63.9	302.1
Indiana	107.9	71.4	15.6	78.6	342.6
Iowa	79.8	57.8	9.8	68.9	266.0
Kansas	83.4	48.0	11.2	69.6	271.6
Kentucky	122.4	79.1	16.7	92.4	388.8
Louisiana	105.6	66.4	15.1	60.3	312.2
Maine	95.5	56.6	10.7	82.4	305.5
Maryland	93.6	60.0	11.3	61.4	280.3
Massachusetts	86.0	47.6	8.5	61.3	263.8
Michigan	88.8	71.3	12.6	66.7	297.4
Minnesota	73.5	37.6	9.7	58.8	229.6
Mississippi	117.5	81.7	19.3	72.7	368.9
Missouri	102.0	80.1	15.1	79.3	344.6
Montana	84.6	43.6	10.9	88.9	290.5
Nebraska	80.6	43.0	10.2	72.3	263.0
Nevada	110.8	81.3	19.9	106.4	398.8
New Hampshire	92.0	58.1	9.6	78.9	290.6
New Jersey	81.1	53.7	8.7	51.0	244.3
New Mexico	61.3	54.4	11.0	80.5	259.4
New York	77.0	67.0	7.8	51.6	251.5
North Carolina	96.3	63.4	14.9	70.9	305.0
North Dakota	62.6	51.8	10.1	49.6	225.0
Ohio	98.2	70.8	12.7	74.7	317.2
Oklahoma	98.6	75.5	14.6	71.4	319.9
Oregon	84.7	46.2	13.8	73.4	273.6
Pennsylvania	86.0	59.6	10.3	60.1	272.2
Rhode Island	98.7	69.9	9.7	61.0	288.6

*International Classification of Diseases, 9th Revision (ICD-9), codes 162, 410–414, 430–438, 490–492, and 496.

Table 7.6 Continued

State	Lung cancer	Ischemic heart disease	Cerebrovascular diseases	Chronic obstructive pulmonary disease	Total SAM
South Carolina	97.7	65.0	19.0	70.3	316.6
South Dakota	78.3	53.5	11.5	55.1	250.6
Tennessee	112.0	78.2	16.5	77.9	347.6
Texas	87.1	64.0	12.5	69.8	287.3
Utah	37.6	26.2	6.9	48.8	156.6
Vermont	90.2	49.4	7.8	75.6	272.3
Virginia	95.5	54.3	13.3	66.0	291.2
Washington	89.1	51.4	10.0	75.4	279.4
West Virginia	116.3	77.4	11.7	87.4	392.8
Wisconsin	79.2	58.5	13.9	61.4	275.9
Wyoming	80.5	48.8	11.0	113.0	315.1
Average age-adjusted SAM rate					289.5

Sources: Thun et al. 1997b; Behavioral Risk Factor Surveillance System: Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion, Division of Adult and Community Health, public use data tape, 1999; Gavin et al. 2001; Hoyert et al. 2001; CDC 2002a,d,e; American Cancer Society, unpublished data.

economists and epidemiologists to develop a consensus methodology for performing these computations. In 1985 dollars, the median estimate for direct health care costs was \$22 billion, indirect lost productivity costs were \$43 billion, and total costs were \$65 billion. The confidence interval (CI) around this estimate was large, ranging from \$38 billion to \$95 billion. National direct costs were equivalent to \$0.72 per pack sold in 1985 dollars, and indirect costs were equal to \$1.45 per pack, for a total of \$2.17 per pack (OTA 1985).

An incidence-based method reported by Hodgson (1992) estimates costs of illness over the lifetimes of smokers and former smokers, separating the survivors and decedents. This approach models expected expenditures during different age intervals given survival, death, the probability of survival, and the probability of dying during these age intervals.

Expected per person expenditures during age interval t are

$$E(st) = E(st)P(st) + E(dt)P(dt),$$

where $E(st)$ = expenditures during age interval t for survivors s ,

$$E(dt) = \text{expenditures during age interval } t \text{ if the individual dies in } t,$$

$P(st)$ = probability of surviving through age interval t , and

$P(dt)$ = probability of dying during age interval t .

Expenditures are discounted to obtain the present value of the stream of dollars that occurs over time. This method accounts for uneven medical care expenditures for different age groups, especially at the end of life. Higher medical care use among smokers may be partially offset by the higher mortality of smokers, which reduces lifetime expenditures. Hodgson (1992) estimated that the current population of smokers would increase the cost of health care by about \$500 billion over their remaining lifetimes.

CDC (1994) used a two-stage econometric model from Duan and colleagues (1983) and estimated that smoking attributable costs were \$50 billion annually in 1993 dollars. Researchers developed a model for smoking attributable risks using data from the 1987 National Medical Expenditures Survey (NMES-2) and from the Health Care Financing Administration (now called the Centers for Medicare & Medicaid Services) to provide estimates for direct medical care expenditures for adults resulting from smoking attributable illnesses for five cost categories (Table 7.7) (CDC 1994;

Table 7.7 National medical expenditures and percentage of total health care expenditures attributable to cigarette smoking for adults, United States, 1993

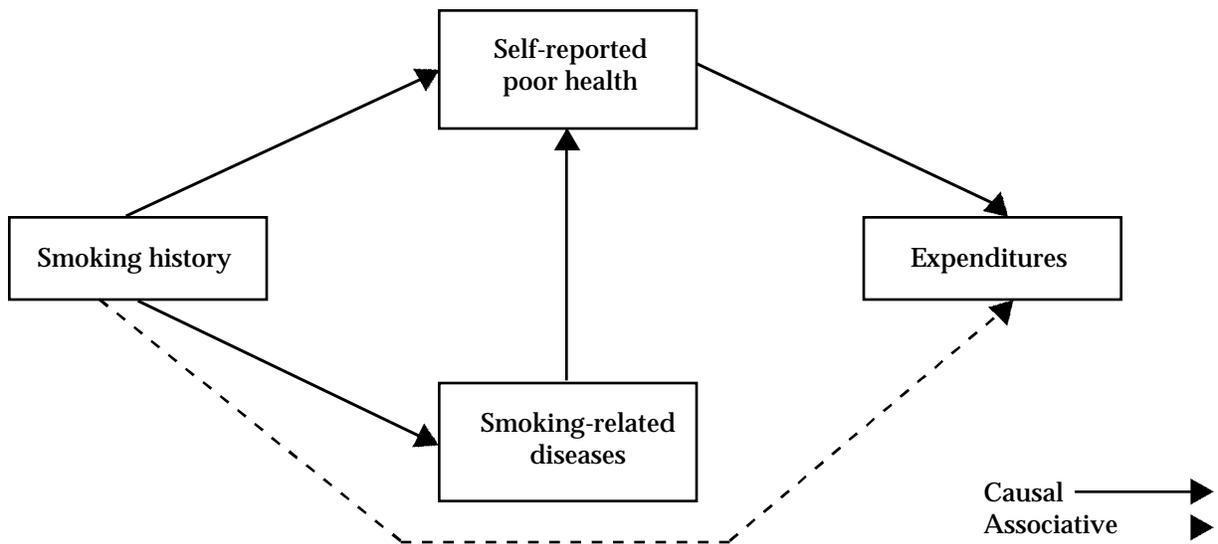
Expense category	Smoking attributable fraction (%)	Expense (\$ in billions)
Hospitals	7.5	26.9
Ambulatory care	7.7	15.5
Nursing home care	6.6	4.9
Prescription drugs	2.6	1.8
Home health care	7.0	0.9
Total	7.1	\$50.0

Source: Centers for Disease Control and Prevention 1994.

Miller et al. 1998). NMES-2 data were first used to estimate the effect of smoking history on the presence of smoking-related medical conditions (i.e., heart disease, emphysema, arteriosclerosis, stroke, and cancer). They were also used to estimate the probability of having any expenditures, and the level of expenditures, for those with positive expenditures related to prescription drugs, hospitalizations, ambulatory care, home health care, and nursing home care as a function of smoking, medical conditions, and health status. This method controlled for age, race, ethnicity, poverty status, marital status, education level, medical insurance status, region of residence, and other variables associated with health status. The model estimated smoking-related expenditures for the U.S. population during the 1988 NMES-2 study period (Figure 7.1).

Using the national model described above with data on populations likely to be receiving publicly funded medical care and data from various state-specific behavioral risk factor surveys, Miller and colleagues (1998) calculated the SAFs for Medicaid costs for each state (national average, 14.4 percent; range, 8.6 percent in Washington, D.C., to 19.2 percent in Nevada). The total Medicaid cost to the states attributable to smoking in 1993 was \$12.9 billion. This

Figure 7.1 Schematic representation of the national model to estimate smoking-related expenditures for 1988



Note: Data elements shown in each box were collected on the National Medical Expenditure Survey in 1988–1989. Source: Miller et al. 1998.

estimate (as well as the national estimate of \$50 billion noted earlier) may be low because it does not include neonatal costs or costs for illnesses among children exposed to smoking in the home (estimated at \$1.97 billion in 1993 [Aligne and Stoddard 1997]), costs of burn injuries from cigarette-caused fires, costs of medical care for persons terminally ill or institutionalized (including military and veterans hospitals), and costs of secondhand smoke-caused illnesses among adults (Novotny 1998). The estimates are also limited by not having direct information on the risk of nursing home utilization for smokers compared with nonsmokers. The calculations for direct nursing home care costs used the SAF for hospitalization costs for persons aged 65 years and older because data from institutionalized persons were not collected in NMES-2. A later study (Miller et al. 1999) attempted to model the SAF for nursing home expenditures using a separate NMES survey on nursing home admissions. This model estimated the probability of admission to a nursing home, given a smoking history. Large potential costs were indicated by the model. However, multiple admissions and length of stay were not considered, and these elements may increase the SAF for nursing home costs substantially.

CDC (2002a) used the methodology of Miller and colleagues (1999) to estimate annual total and per smoker indirect morbidity costs and smoking attributable medical expenditures for 1995–1999 (Table 7.8). Total annual costs (including all sources of payment) were approximately \$75.5 billion using this methodology. Approximate losses of \$82 billion are attributed to lost productivity resulting from smoking attributable diseases. Costs for neonatal health care attributable to smoking were estimated for one year, 1996, and equaled \$366 million. Total direct SAF costs were in the 6 percent range reported in previous studies (Warner et al. 1999; Max 2001). Total annual direct and indirect costs for 1995–1999 were \$157.7 billion.

These estimates vary with the methodology used to estimate costs (Chaloupka and Warner 2000). The studies described earlier emphasized current smoking history, using cross-sectional prevalence data and current year mortality data to estimate costs. The cost-of-smoking estimates were an important part of the damage claims used during negotiations of the 1998 Master Settlement Agreement between the states’ Attorneys General and the tobacco industry (American Legacy Foundation 2002). These state-specific estimates (Miller et al. 1998) addressed losses to state budgets through Medicaid and other state health program expenditures that would not “benefit” from premature deaths and reduced pensions or long-term

Table 7.8 Annual smoking attributable economic costs for adults and infants, United States, 1995–1999

Cost component	Total (\$ in millions)
Lost productivity	
Men	55,389
Women	26,483
Total	81,872
Direct medical care (adults)	
Ambulatory care	27,182
Hospital care	17,140
Prescription drugs	6,364
Nursing home	19,383
Other care	5,419
Total	75,488
Neonatal care*	366
Total costs	\$157,726

*1996 only

Source: Centers for Disease Control and Prevention 2002a.

care costs borne by the Medicare program. This agreement reimbursed the states for medical care provided by taxpayers for smoking-related diseases, resulting in annual payments through 2025 totaling \$246 billion.

In 2001, the American Legacy Foundation (2002) estimated that states had spent \$12 billion on smoking attributable diseases and that \$1.1 billion annually could be saved if the prevalence of adult smoking were 50 percent less in 2001. The cost-of-illness approach offers one perspective on the disease burden from tobacco. The cost estimates should be useful for policymakers with fiduciary responsibility to taxpayers to reduce current preventable disease burdens and the subsequent economic costs of these burdens. As economic burdens for health care increase both for governments and private individuals, such analyses might provide a stimulus to fund tobacco prevention and control programs at higher levels (American Legacy Foundation 2002).

Cost Offsets: Extended Life Expectancy for Nonsmokers and Former Smokers

The U.S. health system is based on an ethical construct that values increased life expectancy and quality of life (USDHHS 2000). However, economists have used econometric models to estimate the net effects of

prolonged life on health and social support systems, considering not only the costs of smoking but of potential economic gains from smoking.

For example, Barendregt and colleagues (1997) concluded that successful smoking cessation and health promotion activities would produce positive economic outcomes (referred to as gross outcomes) in the short run. Barendregt and colleagues (1997), however, did not consider the higher contribution made by longer living nonsmokers to pension and tax systems in making their calculations (Max 2001).

Manning and colleagues (1989) estimated the lifetime, discounted costs that smokers impose on others. Instead of total economic costs, the study focused on only those financial costs that are external to the smokers and their family members; that is, costs paid by insurance companies, the state, or public agencies in caring for smokers and borne by nonsmokers because these are the costs relevant to tax policy. Results indicate that nonsmokers subsidize smokers' medical care and group life insurance while smokers subsidize nonsmokers' pension and nursing home payments because of their shorter life expectancy. The net external financial costs that smokers impose on nonsmokers are positive at a 5 percent discount rate (\$0.15 per pack), but the excise tax revenue from cigarettes at the time of the analysis exceeded those external costs. The costs of lung cancer deaths caused by involuntary smoking and deaths caused by smoking-related fires were not included in this estimate because they were considered internal costs (costs to the individual or to his/her family unit). Costs related to maternal smoking were also omitted. With all lives lost to involuntary smoking and to smoking-related fires defined as external costs, the total external cost per pack was estimated at \$0.38 in 1986 dollars. This may be an uncertain estimate of net external costs due to imperfect data sources and unquantifiable confounding factors. In addition, there was no consideration of annoyance, pain and suffering, or other noneconomic costs (Gravelle and Zimmerman 1994). This same study found that the range of costs produced by various authors varied between net external savings of \$0.17 per pack to costs of \$2.36 per pack. These estimates depended on discount rates used in calculations, costs assigned to involuntary smoking, and various other differences, and therefore Gravelle and Zimmerman (1994) asserted that the net cost estimates produced by Manning and colleagues (1989) provided a satisfactory midpoint estimate.

In an extensive review by the World Bank (Lightwood et al. 2000), the gross health care costs of smoking for high-income countries ranged from 0.10 to 1.1 percent of the gross domestic product, and most of the net-versus-gross cost studies showed net costs for smoking.

The value of longevity and quality of life may be difficult to economically quantify. However, at least one study has discussed the issue of compression of morbidity when smoking is reduced. Using a cross-sectional study of Dutch nationals, Nusselder and colleagues (2000) found that a nonsmoking population spends fewer years with disability than a reference population of smokers and nonsmokers. The nonsmokers had lower mortality risks, but they also had a lower incidence of disability and a higher level of recovery from disability. This status resulted in reduced average time lived with disability (-0.9 years for men aged 30 years and -1.1 years for women) and increased average time lived without disability (2.5 years for men and 1.9 years for women) (Nusselder et al. 2000). Thus, with a nonsmoking population the length of life as well as the length of a disability-free life will be extended. This extension will then compress the disability for nonsmokers into a shorter period toward death; smokers, with lengthier periods of disability, will suffer earlier mortality, but they will also have more disability and certainly more medical care expenditures while disabled when compared with nonsmokers. Although the disability suffered by former smokers will be less than that of current smokers, mortality and disability risks will still be higher among former smokers than among lifetime nonsmokers.

It is clear that methodologic variability and different approaches to gross-versus-net cost estimates can lead to a wide variety of results. However, these should all be considered in the context of the public health premise that prolonging disability-free life is the goal of the health care system (Murray et al. 1994; USDHHS 2000), and thus any negative economic impacts from gains in longevity with smoking reduction should not be emphasized in public health decisions.

Other Costs

Other considerations in the net-versus-gross cost debate are presented in the following section. Previously described studies do not describe all dimensions of the impact of smoking and smoking attributable disease. For example, the pain and suffering, decreased

quality of life, and related psychosocial aspects of physical illness are not measured (Hodgson and Meiners 1982). Prevalence-based, cost-of-illness calculations do not account for economic factors such as Social Security disbursements, pension claims, changes in the demand for health specialties related to the treatment of smoking-related illnesses, and the employment by or monetary dividends from the tobacco industry (Warner 1987). Smoking can cause costs without impacting mortality or even morbidity among smokers. For example, the health or mortality of a smoking spouse may have an effect on nursing home admission rates for the nonsmoking spouse; in addition, lost income to family members who must care for smokers with prolonged disabilities is not usually measured (Max 2001). These are actually direct costs rather than indirect or human capital losses. Costs to employers for absenteeism, lost productivity, higher insurance premiums for smokers (Weis 1981; Kristein 1983), and liability incurred for exposing nonsmokers to passive smoke may also be included as an economic cost of smoking.

Several studies (Warner et al. 1999; Chaloupka and Warner 2000; Lightwood et al. 2000; Max 2001)

have reviewed these economic issues and ongoing controversies that primarily involve the net-versus-gross cost of tobacco on society. This controversy, however, ignores the main burden—that of health—when it dwells on the “benefits” of smoking that result from premature death. Generally, however, it appears that direct costs attributable to smoking comprise 6 to 9 percent of the total national health care budget. Cost estimates have tended to increase over time, reflecting improvements in methodology, increases in medical expenditures for smoking-related diseases because of inflation and/or technology, and expansion of the list of diseases caused by smoking.

Further research on the economic costs of nursing home care is needed as the impact of smoking on admissions to and utilization of nursing homes is not well described. There are also insufficient data on the costs from passive smoking-related illnesses (Max 2001). Indirect costs need more research at the national level, and costs to employers resulting from smoking by their employees should also be the subject of additional research (Max 2001).

Health Benefits of Reducing Cigarette Smoking

Premature Deaths Prevented If the *Healthy People 2010* Prevalence Objectives Are Achieved

To reduce the health consequences of smoking, the Public Health Service targeted substantial reductions in youth and adult smoking rates in the *Healthy People 2010* objectives (USDHHS 2000). The purpose of the *Healthy People 2010* goals is to reduce current smoking from 35 percent (in 1999) to 16 percent among high school youth aged 14 through 17 years, and to reduce current smoking from 24 percent (in 1998) to 12 percent among adults aged 18 years and older. Current smoking among young people was defined as having smoked on 1 or more days in the past 30 days, as reported in the Youth Risk Behavior Survey (CDC 2001e). Current smoking among adults was defined

as ever having smoked 100 cigarettes or more and currently smoking every day or some days, as reported in the NHIS (NCHS 2002).

Whether or not the necessary changes in smoking initiation and cessation are achievable has been the source of some debate. Mendez and Warner (2000) suggested that the *Healthy People 2010* objective to halve U.S. adult smoking prevalence by 2010 was unattainable, and proposed that a more realistic scenario involving a 50 percent reduction in youth initiation rates and the doubling of adult cessation rates could bring the smoking prevalence among adults to 16.7 percent by 2010. A scenario involving a gradual one-third decline in youth initiation and a 50 percent increase in adult cessation rates by 2010 would achieve an estimated youth prevalence rate of 22 percent and an estimated adult prevalence rate of 18 percent.

CDC (unpublished data) has estimated the SAM that could be averted if the *Healthy People 2010* goals for tobacco use were achieved or if the more modest prevalence reductions projected by Mendez and Warner (2000) were made. CDC used a three-step process to estimate the burden of SAM that could be prevented by reducing smoking prevalence. In step one, the number of future smokers in 2010 (by age) was projected based on current smoking prevalence estimates derived from each of three scenarios (Table 7.9): (1) youth initiation and cessation rates as well as adult cessation rates remain unchanged (status quo prevalence), (2) youth initiation declines by one-third and adult cessation increases by 50 percent by 2010 (modest reductions in prevalence), and (3) youth smoking prevalence declines from 35 to 16 percent and adult

prevalence is halved for all age groups (i.e., the *Healthy People 2010* objectives are met). For each prevalence reduction scenario, smoking prevalence rates and the number of smokers in 2010 were estimated for persons aged (in years) 10 through 17, 18 through 24, 25 through 44, 45 through 64, and 65 and older. These calculations projected overall that the number of current smokers in 2010 would be approximately 56.2 million for the status quo prevalence scenario, 49.1 million for the modest prevalence scenario, and 32.3 million for the *Healthy People 2010* prevalence reductions.

For the second step, the investigators estimated the proportion of preventable premature SAM by age through the reductions in smoking (Table 7.10). For each age, the proportion of lifelong smokers

Table 7.9 Smoking prevalence and the number of smokers in 2010 for alternative smoking reduction scenarios, stratified by age, United States

Age	Status quo prevalence*	Modest reductions [†]	<i>Healthy People 2010</i> reductions [‡]
Current smoking prevalence (%)			
10–17 years	36.0	24.4	16.0
Adults	19.5	18.1	12.0
18–24 years	26.9	22.6	14.0
25–44 years	24.1	23.8	13.8
45–64 years	17.4	15.8	12.5
65 years	9.3	7.9	5.5
Number of smokers[§]			
10–17 years	11,714,200	7,948,200	5,210,400
18–24 years	8,104,100	6,803,600	4,207,700
25–44 years	18,896,800	18,640,400	10,765,400
45–64 years	13,821,400	12,599,000	9,948,600
65 years	3,682,400	3,132,500	2,164,500
Total	56,218,900	49,123,600	32,296,600

Note: Figures for the number of smokers are rounded and hence do not add up.

*Assumes constant youth smoking prevalence of 35% (1998 data) and adult cessation rates of 0.21%, 2.15%, and 5.96% for ages 18–30, 31–50, and 51 years, respectively. Smoking prevalence estimates for adults are from the 1998 National Health Interview Survey. Data from the 1999 Youth Risk Behavior Survey were used to project the percentage of 10–17-year-olds expected to become smokers (Centers for Disease Control and Prevention [CDC] 2001b).

[†]Assumes constant annual changes: by 2010, youth initiation rates will decline by one-third and adult cessation rates will increase by 50%.

[‡]Assumes *Healthy People 2010* goals are met: reducing youth smoking prevalence among persons aged <18 years to 16% and prevalence among persons aged 18 years and for each age group by 50% overall (U.S. Department of Health and Human Services 2000).

[§]Based on U.S. Census Bureau population projections (U.S. Census Bureau 2002).

Source: CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, unpublished data.

Table 7.10 Low-, middle-, and high-range estimates of proportions of smoking-related disease (SRD) deaths and preventable deaths among current smokers, stratified by age, United States

Age	Low	Middle	High
A. Percentage of lifelong smokers expected to die from a SRD* (%)			
10–17 years	24	32	50
18–24 years	24	32	50
25–44 years	32	32	50
45–64 years	32	50	50
65 years	50	50	50
B. Expected preventable[†] SRD deaths of lifelong smokers (%)			
10–17 years	100	100	100
18–24 years	100	100	100
25–44 years	75	100	100
45–64 years	26	53	80
65 years	9	24	64
C. Percentage of future SRD deaths preventable with cessation (A x B) (%)			
10–17 years	24.0	32.0	50.0
18–24 years	24.0	32.0	50.0
25–44 years	24.0	32.0	50.0
45–64 years	8.3	26.5	40.0
65 years	4.5	12.2	32.0

*Centers for Disease Control and Prevention (CDC) 1996b; *Federal Register* 1996; Peto et al. 2000.

[†]Assumes that 100% of future SRD deaths are preventable if smokers quit before 45 years of age; the low estimate for smokers aged 25–44 years assumes that only 75% are preventable (100% for 25–34-year-olds and 50% for 35–44-year-olds). For smokers aged 45–64 years, 10% (low), 23.5% (middle), and 37% (high) of deaths among quitters are not considered preventable. For persons aged 65 years, the preventable proportion was reduced by the same percentage as the decline in the preventable proportion between the 25–44-year-old and the 45–64-year-old age groups.

Source: CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, unpublished data.

anticipated to die from a smoking-related disease was multiplied by the percentage of future deaths that are likely preventable through cessation or by preventing initiation. Between 24 and 50 percent of lifelong smokers, depending on age, are expected to die of a smoking-related disease (*Federal Register* 1996; Thun et al. 1997a; Peto et al. 2000). Depending on the age at which smokers quit, all or some of the expected future excess premature deaths are preventable. The percentages of preventable future premature deaths are listed in Table 7.10, Section B. The investigators assumed that 100 percent of future premature deaths from smoking are preventable for persons 10 through 44 years of age if they quit or if they do not initiate smoking (CDC, unpublished data), except for persons aged 25 through 44 years in the low-range column for whom

they assumed that 75 percent of future SAM was preventable (i.e., 100 percent preventable for persons aged 25 through 34 years and 50 percent preventable for persons aged 35 through 44 years).

For former smokers aged 45 years and older, the percentage of preventable future deaths was calculated using published estimates of the proportions of risk among quitters that were not preventable through cessation (i.e., the remaining risks of future deaths). An estimated 10 to 37 percent of former smokers will die of a smoking-related disease even after quitting smoking (CDC, unpublished data). This finding suggests that the percentage of deaths that are preventable ranges from as much as 80 percent (1 minus [0.1 divided by 0.5]) to as little as 26 percent (1 minus [0.37 divided by 0.5]) for former smokers aged 45 through

64 years. For the middle-range estimate, the assumption is that 23.5 percent (the midpoint of 10 to 37 percent) of former smokers aged 45 through 64 years will still die of a smoking-caused disease. Thus, 53 percent (1 minus [0.235 divided by 0.5]) of expected SAM is preventable. For smokers aged 65 years and older, the same percentage decrease in preventable SAM was assumed to occur between the ages of 45 through 64 years and 65 years and older, plus the decreases estimated for ages 25 through 44 and 45 through 64 years. For each age group and risk-of-death range, the proportion of lifelong smokers expected to die from a smoking-related death was multiplied by the percentage of preventable deaths. The results are age-specific estimates of the proportions of future SAM that would be preventable if lifelong smokers were to quit.

For the final step, the investigators calculated the number of smoking-related deaths that would be prevented as a result of a reduction in smoking prevalence in 2010 by multiplying the differences in the number of current smokers for each of the two prevalence reduction goals by the actual proportions of preventable SAM in Section C of Table 7.10. This approach produced low-, middle-, and high-range projections of the number of premature deaths avoided for each of the two levels of reduction in current smoking prevalence. The investigators then calculated how many premature deaths would be avoided by achieving the *Healthy People 2010* goals compared with meeting the modest reductions in prevalence.

The results indicate that under the middle-range preventable proportion assumptions, achieving the modest prevalence reductions by 2010 will prevent approximately 2.5 million expected premature deaths from smoking, compared with the number of projected premature deaths for the status quo youth and adult prevalence rates in 2010 (Table 7.11). The range of projected averted premature deaths is 1.7 to 4 million for the modest prevalence reductions, depending on assumptions about the proportions of future premature deaths that are preventable through quitting (Table 7.11). Compared with the status quo prevalence, achieving the *Healthy People 2010* smoking prevalence objectives will prevent approximately 7.1 million expected premature deaths from smoking, with a range of 4.8 to 11 million. Assuming that recent tobacco control efforts are able to achieve the modest reductions in smoking prevalence, meeting the *Healthy People 2010* goals will prevent an additional 5 million deaths under the middle-range preventable proportion assumptions, with a range of 3.4 to 8 million.

These results demonstrate that reducing smoking prevalence can prevent millions of the future premature deaths expected if youth smoking and initiation rates as well as adult cessation rates stay at 1998 levels. Modest reductions in youth and adult smoking prevalence by 2010 could prevent about 2.5 million deaths, compared with the status quo prevalence estimates.

Existing interventions have led to reductions in tobacco use prevalence and per capita consumption (CDC 2001b). A comprehensive review of programs in California, Massachusetts, Oregon, Arizona, and Florida by Siegel (2002) covers both the positive effects of such programs on smoking prevalence and the negative effects that follow reduced support from the states. In general, comprehensive programs have substantially reduced adult smoking prevalence and per capita consumption following their implementation in the late 1980s and early 1990s. Secular trends in California and Massachusetts before program implementation may have also contributed to reduced disease burdens attributable to smoking over time.

Nevertheless, substantial declines in the per capita use of cigarettes and in adult smoking prevalence in California through the 1990s were associated with a comprehensive program implemented in 1988 (Siegel et al. 2000). During the first years of the program (1989–1993), adult prevalence declined 1.1 percentage points per year in California, compared with 0.6 percentage points per year in the rest of the United States. Adult smoking prevalence is now 17.2 percent in California, compared with the median of 23.3 percent for all states (CDC 2002c). Moreover, there is now evidence to suggest that this reduction has contributed to a decline in the tobacco-related disease burden over time. During 1988–1997, age-adjusted incidence rates for lung cancer declined 14 percent in California, compared with only 2.7 percent in non-California cancer surveillance regions (CDC 2000). In an analysis of trends in mortality from heart disease between 1989 and 1997, there were 33,300 fewer deaths from heart disease than expected in California compared with the rest of the United States (Fichtenberg and Glantz 2000). However, lung cancer mortality will change slowly in response to population smoking prevalence changes, and thus the secular changes present in California before the start of the program contributed to the decline in lung cancer mortality. Cardiovascular mortality changes will be much more rapid, and these changes appear to be closely associated with program activity level.

Table 7.11 Estimated number of preventable smoking-related disease (SRD) deaths and *Healthy People 2010*^{*} prevalence reduction goals, stratified by age, United States

Age	Preventable number of smoking-related deaths		
	Low	Middle	High
A. <i>Healthy People 2010</i> vs. status quo prevalence[†]			
10–17 years	1,570,000	2,100,000	3,250,000
18–24 years	935,000	1,250,000	1,950,000
25–44 years	1,950,000	2,600,000	4,070,000
45–64 years	322,000	1,020,000	1,550,000
65 years	68,500	161,000	486,000
Total	4,800,000	7,100,000	11,000,000
B. Modest[‡] reductions vs. status quo prevalence			
10–17 years	904,000	1,200,000	1,880,000
18–24 years	448,000	599,000	934,000
25–44 years	164,000	219,000	342,000
45–64 years	124,000	395,000	596,000
65 years	28,000	75,000	197,000
Total	1,700,000	2,500,000	4,000,000
C. <i>Healthy People 2010</i> vs. modest reductions in prevalence			
10–17 years	657,000	876,000	1,370,000
18–24 years	623,000	831,000	1,300,000
25–44 years	1,890,000	2,500,000	3,940,000
45–64 years	220,000	702,000	1,060,000
65 years	44,000	118,000	310,000
Total	3,400,000	5,000,000	8,000,000

Note: All figures are rounded and hence do not add up.

^{*}*Healthy People 2010* goals are to reduce smoking among persons aged <18 years to 16% and among persons aged ≥ 18 years by 50% overall and for each age group (U.S. Department of Health and Human Services [USDHHS] 2000).

[†]The status quo prevalence assumes that smoking initiation and cessation rates will remain constant between 1998 and 2010.

[‡]The modest reductions in prevalence assume constant annual changes: by 2010, youth initiation rates will decline by one-third and adult cessation rates will increase by 50%.

Sources: USDHHS 2000; Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, unpublished data.

In Massachusetts, a comprehensive tobacco control program implemented in 1992 was associated with a decline of 0.43 percentage points per year in adult smoking prevalence between 1992 and 1999 (Biener et al. 2000). In Arizona, state-specific surveys following implementation of a comprehensive program in 1994 indicate that adult prevalence declined from an estimated 23 percent to approximately 20 percent between 1996 and 1999 (CDC 2001d). In Oregon, adult smoking prevalence declined from 23.4 percent in 1996 to

21.4 percent in 1999 after implementation of the 1996 tobacco control program (CDC 1999b). These changes, although modest, compare favorably with the 0.03 annual percentage point increase in adult prevalence in comparison states during approximately the same period (Siegel 2002).

Information regarding the population burden of the health effects of smoking helps to quantify the potential health and economic impacts of reduced smoking prevalence. What studies are needed to

assess the actual versus the imputed potential consequences for health of reducing smoking? PAR projections have been used to assess the impact of population-based health programs, such as in the Framingham study on CVD (Sturmans et al. 1977). In this study, a 37.3 percent attributable risk reduction in CVD mortality might have been achievable through the elimination of smoking, but because of the complex mix of strengths of association for different parts of the population, the baseline risks of the population, the proportion of the population affected by the intervention, and the degree of risk factor reduction achieved, only a few percentage point changes attributable to smoking reductions by a specific program per se were achieved. Keying interventions to specific risk groups may improve health results for these groups without necessarily reducing the population burden of mortality (Rothenberg et al. 1991). Thus, the PAR approach sets the stage for additional analyses and helps drive policies to address the population effects as well as the individual effects of smoking.

Conclusions

1. There have been more than 12 million premature deaths attributable to smoking since the first published Surgeon General's report on smoking and health in 1964. Smoking remains the leading preventable cause of premature death in the United States.
2. The burden of smoking attributable mortality will remain at current levels for several decades. Comprehensive programs that reflect the best available science on tobacco use prevention and smoking cessation have the potential to reduce the adverse impact of smoking on population health.
3. Meeting the *Healthy People 2010* goals for current smoking prevalence reductions to 12 percent

Summary

Regardless of the methodologic issues around the estimation methods, cigarette smoking remains the leading single cause of preventable mortality in the United States. This chapter reviewed various methods for assessing the disease burden of smoking-related illnesses, including epidemiologic calculations, indirect estimates, and model-based approaches for assessing smoking attributable mortality. The PAR calculation, with appropriate controls for age and gender, offers useful estimates of the mortality burden of disease attributable to tobacco use in the U.S. population. These estimates are not biased strongly by confounding factors, even though smokers, compared with non-smokers, tend to have different profiles for a number of lifestyle-related risk factors for disease and may have different costs for even the same condition. Economic disease burden estimates have been used to provide a more compelling argument as to the costs of smoking to governments and society in general, thus adding information that can be used to support comprehensive tobacco use prevention and control programs.

- among persons aged 18 years and older and to 16 percent among youth aged 14 through 17 years will prevent an additional 7.1 million premature deaths after 2010. Without substantially stronger national and state efforts, it is unlikely that this health goal can be achieved. However, even with more modest reductions in tobacco use, significant additional reductions in premature death can be expected.
4. During 1995–1999, estimated annual smoking attributable economic costs in the United States were \$157.7 billion, including \$75.5 billion for direct medical care (adults), \$81.9 billion for lost productivity, and \$366 million in 1996 for neonatal care. In 2001, states alone spent an estimated \$12 billion treating smoking attributable diseases.

Implications

Population attributable risk estimates that indicate how much of the disease burden attributable to smoking can be avoided through tobacco control interventions are an important starting point for policy development. In addition, economic cost-of-illness studies on tobacco-related diseases can help inform policymakers about the benefits of supporting comprehensive tobacco use prevention and control programs, especially at the state level. Comprehensive interventions at state and federal levels, including

educational, clinical, regulatory, and economic actions, have been shown to reduce smoking rates and to subsequently reduce the population disease burden caused by tobacco.

There is a need for additional research on the costs of illnesses related to tobacco use, the economic impact of tobacco control programs, how to quantify specific program effects on reductions in tobacco use, subsequent disease impact, and the cost and effectiveness of alternative approaches.

Appendix 7-1: Estimating the Disease Impact of Smoking in the United States

Methodology

Six approaches to calculating smoking attributable mortality (SAM) in the United States are reviewed in this section. The first approach, the population attributable risk (PAR) calculation, is the most commonly used and was the earliest method used to estimate SAM (Levin 1953). Levin originally used this approach, sometimes referred to as "Levin's attributable risk," to describe the burden of preventable lung cancer associated with smoking. The PAR and variants also have been referred to as the assigned share, excess risk, etiologic fraction, attributable proportion, attributable risk, and incidence density fraction (IDF) (Levin 1953; Walter 1976; Rothman 1986; Greenland and Robins 1988; U.S. Department of Health and Human Services [USDHHS] 1989a; Greenland 1999). These measures are basically all estimates of the total disease burden (usually mortality) or of the specific disease burden attributable to smoking. When multiplied by the reported number of deaths in these disease categories, numbers of deaths for a given time period attributable to tobacco use can then be estimated. The IDF further incorporates the concept of timing of the excess disease; that is, the onset of exposure-caused disease occurs earlier among the exposed than among the unexposed (Greenland 1999). Unless a population is in a steady state with regard to exposure and disease, estimates of attributable risk may not reflect the cumulative burden of disease for exposed cohorts (Greenland and Robins 1988). Based on this first application of the attributable risk calculation to available case-control data, Levin reported that from 62 to 92 percent of all cases of lung cancer in the study populations were caused by smoking. PAR is derived as follows:

If the excess rate (or risk) of disease (D_x) from a given exposure is the rate of death in the exposed group (D_e) minus the rate of death in the unexposed group (D_u), then

$$D_x = D_e - D_u$$

The excess proportion of the disease attributable (AP) to the exposure is

$$AP = \frac{D_x}{D_e}$$

The relative risk (RR) (or relative rate) of deaths caused by the exposure is

$$RR = \frac{D_e}{D_u}$$

and therefore the AP can be rewritten as

$$AP = \frac{RR-1}{RR}$$

The fraction (F) of all cases of the disease that occurs among exposed persons in the participant population depends on the prevalence rate (P) of the risk factor. Thus,

$$F = \frac{P(RR)}{P(RR-1) + 1}$$

If the fraction (F) of all cases occurs among exposed persons, and if the proportion of all cases attributable to the exposure is AP, then the attributable risk for all cases in the entire population (PAR) (exposed and unexposed) is

$$PAR = AP \times F$$

Thus, PAR depends on the RR of deaths or disease due to the specific risk factor (exposure) prevalence (P) in the entire population, and the formula for PAR can then be written as

$$PAR = \frac{P(RR - 1)}{P(RR - 1) + 1}$$

The PAR calculation underlies the Centers for Disease Control and Prevention's (CDC) Smoking-Attributable Mortality, Morbidity, and Economic Costs (SAMMEC) methodology. This tool was developed to assist states and other jurisdictions to estimate the burden of disease caused by tobacco in their jurisdictions (Shultz et al. 1991; CDC 2002d). SAMMEC applies the PAR calculation to men and women separately and to broad age groups (35 to 64 years and 65 years and older) to account for variability in risk and exposure according to age and gender. However, SAMMEC does not adjust the PAR estimates for other risk factors for the various smoking-related diseases.

In a second approach, Doll and Peto (1981) used the risk difference to estimate cancer deaths attributable to smoking in the United States in 1978. Excess cancer deaths attributable to smoking were computed by subtracting from the observed number of deaths (D_{obs}) for a specific diagnosis (x) the number of deaths expected (D_{ns}) if the population at risk had the same mortality rate as nonsmokers for the disease.

$$\text{SAM}_x = D_{\text{obs}} - D_{\text{ns}}$$

Cancer Prevention Study I (CPS-I), conducted during 1959–1972, provided mortality rates for cancers and other leading causes of death in lifetime nonsmokers, and these rates were then used to calculate overall expected deaths of smokers (Garfinkel 1985). This method also inherently assumes that the comparison of smokers and lifetime nonsmokers is not affected by confounding.

One methodologic concern raised with regard to PAR estimates is the potential effect from confounding by differences in other risk factors across smoking groups (Sterling et al. 1993). The third approach, a model-based approach for estimating PAR, was used by Malarcher and colleagues (2000) to develop cause-specific, age- and confounder-adjusted attributable fractions (AF_A) (as a weighted sum of the age-specific estimates from CPS-II data) and 95 percent confidence limits around these estimates. They expanded the basic formula for PAR to include adjustment for potential confounding factors, including education, alcohol consumption, hypertension, and diabetes.

$$\text{AF}_C = 1 - \frac{j}{j}$$

where j is the proportion of deaths in the j th cell in a matrix defined by exposure and confounder status

(e.g., smoking and age), and RR_j is the RR for smokers compared with lifetime nonsmokers adjusted for confounders C (e.g., age). This calculation provides an estimate of SAM that is adjusted for the selected, potential confounding factors. The estimates obtained with this model were very similar to the national SAM estimates that adjusted risks only for age and gender, as in the SAMMEC software.

In the fourth method, Thun and colleagues (2000) also used a model-based approach to evaluate SAM estimates based on the CPS-II data both with and without adjustment for possible confounders, including race, education, marital status, "blue collar" occupation, dietary factors, body mass index, and physical activity. The Cox proportional hazard model was used by the investigators to estimate the hazard ratio (HR) for various diseases for current and former smokers compared with lifetime nonsmokers, adjusting for sociodemographic factors, diet, alcohol consumption, aspirin use, physical activity, body mass index, and asbestos exposure. The authors compared the SAM estimates obtained using this adjusted HR to estimates made for current and former smokers, among men and women separately, with adjustment for age only. The HR corresponds to the RR in the PAR calculation. Only small differences were found in the SAM estimates using the confounder-adjusted risk model compared with the calculation with risks and exposures adjusted only for gender and broad age groups.

Another method for estimating disease impact among state populations uses smoking status data collected from death certificates, first implemented in 1989 by the state of Oregon (McAnulty et al. 1994). In Oregon, the physician completing the death certificate lists the primary causes of death followed by secondary conditions that may have contributed to the death. The question "Did tobacco use contribute to the death?" has four possible responses: yes, probably, no, or unknown. Comparisons of estimates based on this direct method with estimates based on the PAR approach show close similarities. Of 212,448 deaths in Oregon during 1989–1996, the PAR estimate attributed 20.1 percent (42,778 deaths) to cigarette smoking. Based on the physician assignment that attributed 27 causes of death to smoking, the corresponding estimate was 20.2 percent (42,839 deaths). Nine jurisdictions (Colorado, Louisiana, Maryland, Nebraska, North Dakota, Oregon, Texas, Utah, and New York City) now ask physicians to indicate on death certificates whether tobacco use contributed to the death (Thomas et al. 2001).

Peto and colleagues (1992) developed an approach for broad, international applications that uses the absolute rate of lung cancer mortality in a particular country as the anchoring point. The lung cancer rate is used to estimate the proportions of smokers and nonsmokers in the population and then the RR estimates from CPS-II are scaled proportionately, with a 50 percent reduction in the estimated excess risk to produce “conservative” estimates.

Key Data Sets Used to Estimate Smoking Attributable Mortality and Years of Potential Life Lost

Numerous cohort studies provide RR estimates for smoking-related diseases and mortality (Pearl 1938; Hammond and Horn 1954; Kahn 1966; Doll and Peto 1976; Garfinkel 1980a,b; Rice et al. 1986; Lew and Garfinkel 1988; USDHHS 1989a; Doll et al. 1994; Thun et al. 1997a). These studies are extensively described in several publications, including Monograph 8 of the Smoking and Tobacco Control Monograph Series published by the National Cancer Institute (NCI 1997). The RR estimates from CPS-II have been incorporated by CDC into SAMMEC for the purpose of estimating state-specific SAM, smoking attributable years of potential life lost (YPLL), and economic costs (SAMMEC, version III) (CDC 2002d).

The CPS-II data set currently used to estimate the burden of disease comes from a six-year follow-up of participants recruited by American Cancer Society (ACS) volunteers from all states and some territories in 1982. On recruitment, smoking status (current, former, or never) and other lifestyle factors (medical history, current health status, age, gender, and race) were ascertained (Stellman and Garfinkel 1986; Thun et al. 1997a). Volunteers reported the vital status of participants each year, and for participants who died, the underlying cause of death was obtained from death certificates. Information from death certificates was obtained for 94.1 percent of the deaths. The selected sample differed from the U.S. population in that participants tended to be white (93 percent), and had more education and a higher socioeconomic status than the national population (Malarcher et al. 2000). Although follow-up continues to the present, RRs from these subsequent years have not been used in SAMMEC software because smoking status (current and former) was assessed for all cohort members only on

enrollment, leading to an increased potential for misclassification of smoking status over time. National smoking prevalence data from the National Health Interview Survey (NHIS) and from various state-specific surveys (CDC 1996b) were used, along with RR estimates from CPS-II, to estimate PAR and SAM either for the nation or for individual states (CDC 1997, 2001b, 2002d).

The first ACS study (CPS-I) of one million persons in the United States provides an appropriate comparison data set for evaluating changes in RR estimates associated with smoking between the mid-1960s and the mid-1980s (Table 7-1.1) (Hammond 1966; USDHHS 1989a; Shopland et al. 1991; Thun et al. 1997a). The RRs for current smokers versus lifetime nonsmokers for lung cancer across the time periods when CPS-I and CPS-II were conducted increased substantially for both men (from 11.4 to 23.3) and women (from 2.7 to 12.7) (Thun et al. 1997a). The RRs for most of the cardiovascular diseases (CVDs) showed increases between the studies, and the RRs for all-cause mortality in smokers increased from 1.7 to 2.3 in men and from 1.2 to 1.9 in women across the interval.

Mortality rates for several smoking-related diseases have changed in recent years. Age-standardized lung cancer death rates decreased among men, and rates have begun to plateau among women (Ries et al. 2000). Cardiovascular disease and stroke mortality rates declined between CPS-I and CPS-II, regardless of smoking status, which is consistent with trends for the various CVDs in general (National Center for Health Statistics 1996). Although there was a documented decline in smoking in the United States between CPS-I and CPS-II, mortality rates reflect the effects of many factors that may change over time. For smoking, prevalence may vary and the strength of the association between smoking and particular diseases may change. There also may be changes in other risk factors for the diseases caused by smoking, and in their treatment and survival rates. Estimates of SAM at any particular point in time reflect the earlier birth cohort patterns in smoking initiation and cumulative exposures to lifetime smoking, as well as more recent patterns in cessation.

The codes from the *International Classification of Diseases, 9th Revision* (ICD-9) (USDHHS 1989b) have been changed in Web SAMMEC to reflect the newer 10th revision classifications (ICD-10) (CDC 2002b). The codes from both revisions are listed in Table 7-1.2.

Table 7-1.1 Age-adjusted relative risks of death from smoking-related diseases from the Cancer Prevention Study (CPS) I and CPS-II, stratified by gender

Disease category (ICD-9 code)*	CPS-I (1959–1965)				CPS-II (1982–1988)			
	Males		Females		Males		Females	
	CS [†]	FS [‡]	CS	FS	CS	FS	CS	FS
Neoplasms[§]								
Lip, oral cavity, pharynx (140–149)	6.3	2.7	2.0	1.9	10.9	3.4	5.1	2.3
Esophagus (150)	3.6	1.3	1.9	2.2	6.8	4.5	7.8	2.8
Stomach (151)	1.8	1.7	1	1	2	1.5	1.4	1.3
Pancreas (157)	2.3	1.3	1.4	1.4	2.3	1.2	2.3	1.6
Larynx (161)	10	8.6	3.8	3.1	14.6	6.3	13	5.2
Trachea, bronchus, lung (162)	11.4	5	2.7	2.6	23.3	8.7	12.7	4.5
Cervix uteri (180)			1.1	1.3			1.6	1.1
Urinary bladder (188)	2.9	1.8	2.9	2.3	3.3	2.1	2.2	1.9
Kidney, other urinary (189)	1.8	1.8	1.4	1.5	2.7	1.7	1.3	1.1
Acute myeloid leukemia (204–208)	1.6	1.6	1	1	1.9	1.3	1.1	1.4
Cardiovascular diseases[§]								
Ischemic heart disease (410–414)								
Aged 35–64 years	2.3	1.6	1.8	1.7	2.8	1.6	3.1	1.3
Aged 65 years	1.4	1.3	1.2	1.3	1.5	1.2	1.6	1.2
Other heart disease (390–398, 415–417, 420–429)	1.4	1.1	1.1	1.4	1.8	1.2	1.5	1.1
Cerebrovascular disease (430–438)								
Aged 35–64 years	1.8	1	1.9	1.8	3.3	1	4	1.3
Aged 65 years	1.2	1	1	1.1	1.6	1	1.5	1
Atherosclerosis (440)	3.1	2	1.9	1.5	2.4	1.3	1.8	1
Aortic aneurysm (441)	4.1	2.4	4.6	3.7	6.2	3.1	7.1	2.1
Other arterial disease (442–448)	3.1	2	1.9	1.5	2.1	1	2.2	1.1
Respiratory diseases[§]								
Pneumonia, influenza (480–487)	1.8	1.6	1	1	1.8	1.4	2.2	1.1
Bronchitis, emphysema (490–492)	8.8	10.2	5.9	5.9	17.1	15.6	12	11.8
Chronic airways obstruction (496)	5.5	9.6	5.1	5.3	10.6	6.8	13.1	6.8
Perinatal conditions								
Short gestation/low birth weight (765)			1.8				1.8	
Respiratory distress syndrome (769)			1.8				1.3	
Other respiratory conditions in newborns (770)			1.8				1.4	
Sudden infant death syndrome (798.0)			1.5				2.3	

*International Classification of Diseases, 9th Revision.

[†]CS = Current smokers.

[‡]FS = Former smokers.

[§]Among persons aged ≥ 35 years.

Perinatal relative risks for 1959–1965 are from McIntosh 1984; 1982–1988 data are from Gavin et al. 2001 and Malloy et al. 1992; see also ftp://ftp.cdc.gov/pub/health_statistics/nchs/publications/icd-9/.

Sources: McIntosh 1984; U.S. Department of Health and Human Services 1989b; National Center for Health Statistics, public use data tapes, 1995–1999; Thun et al. 1997b; National Cancer Institute 1999; Gavin et al. 2001; Hall 2001; Hoyert et al. 2001; Mathews 2001; Centers for Disease Control and Prevention 2002a,b,d; International Agency for Research on Cancer 2002; American Cancer Society, unpublished data.

Limitations of Smoking Attributable Mortality and Years of Potential Life Lost Calculations

The PAR calculation and the extension to estimate SAM and YPLL involve assumptions associated with uncertainties. These assumptions and other methodologic issues have been debated in the literature in recent years. This section addresses limitations of SAM and YPLL estimates and concerns that have been raised about these estimates.

SAM and YPLL derived from the PAR calculation may be underestimates in several respects. First, the SAM and YPLL estimates from SAMMEC are based on the prevalence of current and former smokers in the current year; however, the deaths that occur during a given year are primarily among persons who began smoking 30 to 50 years earlier, many of whom had quit smoking (Schulman et al. 1997). The prevalence of smoking among these persons 30 to 50 years ago was almost double that of similarly aged adults today, and many of the participants in CPS-II were former smokers at entry into the study. The current RRs for former smokers are lower than those of current smokers, but do not reflect the risk that was sustained up to the present age. The likelihood of dying from a smoking-related disease for those who began smoking 30 to 50 years ago and quit only recently is far higher than that for former smokers who began smoking at the same age but quit smoking earlier. Thus, the cross-sectional PAR and SAM estimates do not accurately estimate the risks of past cohorts of smokers.

The use of survey data to estimate exposure may contribute to some uncertainty in the PAR calculation. Although population-based surveys provide reasonably accurate estimates of adult prevalence, there may be some underestimation of true exposure (Caraballo et al. 2001). The degree of underestimation has likely increased in recent years.

The SAM estimates also do not include mortality caused by cigar smoking, pipe smoking, or smokeless tobacco use. Approximately 1,000 deaths in the United States were attributable to pipe smoking in 1991 (Nelson et al. 1996). Finally, diseases have now been causally associated with smoking in this report of the Surgeon General that were not included in previous estimates of SAM. Additional ICD-10 codes have now been included for RRs (Table 7-1.2) as part of the PAR calculations presented earlier in this chapter.

Previous SAM calculations have been criticized, however, for overestimating the disease burden of smoking. Estimates using PARs based on RRs that were

not adjusted for potential confounding factors have been criticized as being too high (Sterling et al. 1993; Levy and Marimont 1999). As an alternative, Weinkam and colleagues (1992) and Sterling and colleagues (1993) developed RR estimates using data from the NHIS, a cross-sectional household survey of health status with self-reported smoking status, and from the 1986 National Mortality Followback Survey (NMFS), a representative sample of all decedents aged 25 years or older in the United States. The method produced somewhat lower PARs than those incorporated into SAMMEC, and RR estimates were below 1.0 for some diseases, including some for which there is a causal association with smoking, such as cancers of the lip, oral cavity, and pharynx. Relative risk estimates must be internally valid (Greenland and Robins 1988), and strong biologic relationships between smoking and disease have been demonstrated for the diseases discussed in previous chapters of this report. Siegel and colleagues (1994) pointed out that the approach used by Weinkam and colleagues (1992) can be criticized for lacking internal validity. For example, the analysis of Weinkam and colleagues (1992) produced a RR for laryngeal cancer that was higher for men who formerly smoked than for current smokers, and a risk for lung cancer that was similar among women who were current and former smokers. These findings are not consistent with the strong evidence documented in previous reports of the Surgeon General that quitting smoking reduces the population risk for these diseases (USDHHS 1990). These surprising findings from the NMFS analyses might result from the small number of deaths from some diseases in the data Weinkam and colleagues (1992) used in their sampling process.

Two studies evaluated the methodology Sterling and colleagues (1993) used and the effects of adjusting for potential confounding factors within the CPS-II data set (Malarcher et al. 2000; Thun et al. 2000). Both analyses found that adjustment for potential confounders and consideration of effect modifiers did not appreciably alter the partially adjusted overall PAR and SAM estimates reported by CDC using the SAMMEC methodology. Thun and colleagues (2000) found that adjusting for multiple potential confounders slightly decreased the RR and PAR for current smokers among both men and women while they increased slightly for women who were former smokers. Overall, the estimated SAM for 1990 decreased by approximately 1 percent, from 401,000 to 397,000 deaths with fully adjusted rather than only age-adjusted RR estimates from CPS-II. Malarcher and colleagues (2000) found that for four of the main classes of disease (lung cancer, chronic airways obstruction,

Table 7-1.2 International Classification of Diseases (ICD) codes and comparability ratios* (CR) for smoking-related diseases, 1965–1999

Disease category	ICD-10 [†]		ICD-9 [‡]		ICD-8 [§]		ICD-7 [¶] code (1965–1967)
	code (1999)	CR	code (1979–1988)	CR	code (1968–1978)	CR	
Neoplasms[¶]							
Lip, oral cavity, pharynx	C00–14	0.960	140–149	1.012	140–149	1.060	140–148
Esophagus	C15	0.997	150	1.033	150	0.991	150
Stomach	C16	1.006	151	NR**	NR	NR	NR
Pancreas	C25	0.998	157	1.033	157	1.002	157
Larynx	C32	1.005	161	1.001	161	1.032	161
Trachea, bronchus, lung	C33–34	0.984	162	1.001	162	1.032	162–163
Cervix uteri	C53	0.987	180	1.011	180	1.003	171
Urinary bladder	C67	0.997	188	0.992	188	1.017	181
Kidney, other urinary	C64–66, C68	1.000	189	0.992	189	1.017	180
Acute myeloid leukemia	C91–95	1.012	204–208	NR	NR	NR	NR
Cardiovascular diseases[¶]							
Rheumatic heart disease	I00–09	0.821	390–398	0.665	390–398	1.152	400–402, 410–416
Ischemic heart disease	I20–25	0.999	410–414	0.878	410–413	1.146	420
Pulmonary heart disease	I26–28	0.972	415–417	2.504	426, 450	0.810	434, 465
Other heart disease	I29–51	0.972	420–429	2.504	420–425, 427–429	0.239	421–422, 430–433
Cerebrovascular disease	I60–69	1.059	430–438	1.005	430–438	0.991	330–334
Atherosclerosis	I70	0.964	440	1.065	440	0.896	450
Aortic aneurysm	I71	1.001	441	0.741	441	1.082	451
Other arterial disease	I72–78	0.850	442–448	0.741	442–444, 446–447	NR	452–454, 456, 4671–72
Respiratory diseases[¶]							
Pneumonia, influenza	J10–18	0.698	480–487	0.926	470–474, 480–486	1.044	480–483, 490–493
Bronchitis, emphysema	J40–43	0.894	490–492	0.969	490–492	1.056	501, 502, 5271
Chronic airways obstruction	J44	1.097	496	1.005	519.3	NR	5272
Perinatal conditions							
Short gestation/low birth weight	P07	1.106	765	0.963	777	NR	774, 776
Other respiratory conditions in newborns	P23–28	0.846	770	NR	776.0, 776.9	NR	762, 763
Respiratory distress syndrome	P22	1.026	769	NR	776.1, 776.2	NR	NR
Sudden infant death syndrome	R95	1.036	798.0	0.910	795.0	NR	NR

*Comparability ratios may not exactly match the included disease codes for each condition. Complete descriptions of the comparability ratios are available from the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC).

[†]ICD, 10th revision.

[‡]ICD, 9th revision.

[§]ICD, 8th revision.

[¶]ICD, 7th revision.

[¶]Among persons aged ≥ 35 years.

**NR = Data were not reported.

Sources: World Health Organization 1955, 1965; U.S. Department of Health and Human Services 1989b; Anderson et al. 2001; CDC 2002b.

CVD, and cerebrovascular disease), the CPS-II-based SAM was 19 percent larger than the estimates based on the NMFS/NHIS combined data set. The authors set any of the RR estimates that were less than 1.0 in the Sterling and colleagues (1993) study to 1.0 because RRs less than 1.0 were not plausible for diseases such as oropharyngeal cancer and CVD, for which there is sufficient evidence of causality. Fully adjusting the RRs for potential confounders in this study, including alcohol consumption, resulted in only a 2.5 percent difference in the SAM in comparison with that of Sterling and colleagues (1993). However, adjusting for alcohol consumption in the case of oral cancer is inappropriate because it is not only a potential confounding factor but also an effect modifier, acting synergistically with smoking to increase risk for oral cancer. Effect modification refers to a change in the magnitude of risk for smoking according to the presence or level of another variable (alcohol).

A second major criticism of SAMMEC involves the use of RR estimates from CPS-II because CPS-II participants were not representative of the entire U.S. population—being a cohort recruited primarily from friends and families of ACS volunteers. Differences in study populations, in the model-based versus stratified analyses, and in possible bias from the use of proxy respondents in NMFS may also contribute to the differences in SAM rates calculated by Sterling and colleagues (1993) and Malarcher and colleagues (2000). Studies have found that proxy respondents (used in NMFS) misclassify smoking by decedents more than self-reports do, thereby tending to reduce the RR of diseases associated with smoking (Lerchen and Samet 1986; Boyle and Brann 1992). A key assumption of SAMMEC is that the CPS-II RR estimates have external validity; they can be extended to the entire U.S. population. The extent of their external validity, or generalizability, is a matter of judgment based on characteristics of the CPS-II population that may modify the effects of smoking, and is based on the biologic understanding of the mechanisms underlying the causal effects of smoking on disease. Sufficient variability must also exist in both the exposure and the outcome of interest in cohort studies such as the CPS-II to assure generalizability. Szklo (1998) asserted that a cohort study need not be a representative sample of the population to develop useful *relative* measures of association, but it should be representative in order to estimate an *absolute* measure of disease frequency that can be generalized with confidence. Thus, CPS-II provides sufficient population representation for the establishment of valid RRs for the entire

population as these are *relative* and not *absolute* measures of disease occurrence.

One other major issue concerning the SAM calculation is that the results produced using any of the cited methodologies are approximations, useful for describing the magnitude of the disease burden. The input data have limitations, and there is uncertainty associated with the estimates that is only partially represented by a confidence interval (CI). For example, deaths in any given year are due to incident cases of disease in prior years, and these cases depend on a complex history of smoking exposure, including age at onset, duration, number of cigarettes smoked per day, types of cigarettes smoked, secondhand smoke exposure, age at quitting, and other risk factors for the specific disease. Relative risks are calculated for populations for a fixed period of time (e.g., 1982–1988 in CPS-II), but changes in the population exposure are difficult to capture during this fixed time period. In addition, prevalence of smoking and the RR for different smoking-related diseases vary across age groups. This variance may lead to distortions in the PAR estimation because higher smoking prevalence among younger members of the population, which contributes to a higher incidence of disease at older ages in the population, is not matched to the higher mortality among the older population.

In addition, for some of the diseases linked to smoking, for example CVD and cerebrovascular diseases, other risk factors such as hypertension, diet, and heredity add greatly to the complexity of estimating the population disease burden attributable solely to tobacco use. Varying the combinations of these contributing risk factors will alter the mortality rate and thus the preventable fraction of death from such diseases more than simply reducing the smoking prevalence (Rothenberg et al. 1991). For diseases such as lung cancer and chronic obstructive pulmonary disease (COPD), there are virtually no other risk factors, and thus the variability in these disease burdens while accounting for other risk factors would be extremely limited.

Review of Previous Estimates

Since 1964, several Surgeon General's reports have commented on the burden of smoking attributable deaths and diseases. In 1964, the Advisory Committee to the Surgeon General reviewed seven prospective cohort studies on smoking and mortality and found that the ratio of the death rate among current

smokers to the death rate of nonsmokers was 1.68 (U.S. Department of Health, Education, and Welfare [USDHEW] 1964). In 1979, the Surgeon General labeled cigarette smoking the single most important preventable environmental factor contributing to illness, disability, and death in the United States (USDHEW 1979). In 1989, the Surgeon General reported that data from CPS-II indicated a substantial increase in RRs for smoking along with an increase in the disease burden of smoking (SAM) since 1964 (USDHHS 1989a). These changes were attributed in part to birth cohort changes in smoking patterns. Several previous reports of the Surgeon General, as well as other reports, have used CPS-I, CPS-II, and other cohort study results to produce estimates of total smoking attributable deaths (CDC 1987, 1991, 1993, 1997) from cancers caused by smoking (Garfinkel 1980a; USDHHS 1982), CVD (Garfinkel 1980b; USDHHS 1983), chronic airways obstruction (or chronic obstructive pulmonary disease) (USDHHS 1984; Davis and Novotny 1989), adverse perinatal effects (Gavin et al. 2001), and other adverse effects.

Several national SAM estimates have been reported, including 270,000 deaths for 1980 (Rice et al. 1986), 314,000 deaths for 1982 (Office of Technology Assessment 1985), 320,000 deaths for 1984 (CDC 1987), 390,000 deaths for 1985 (USDHHS 1989a), 434,000 deaths for 1988 (CDC 1991), 418,690 deaths for 1990 (CDC 1993), an annual average of 430,700 deaths for 1990–1994 (CDC 1997), and an annual average of 442,398 deaths for 1995–1999 (CDC 2002a).

Rice and colleagues (1986) used the PAR calculation to estimate national SAM as well as morbidity and economic costs. Pooled RR estimates were derived from three cohort studies on smoking and health. The mathematical PAR formula was expanded to include current and former smoking separately, and CDC incorporated this stratification into SAMMEC I software (Shultz et al. 1991). States and other jurisdictions used SAMMEC I and later SAMMEC versions (II and III) to estimate the mortality and economic disease burden attributable to smoking in their populations (Nelson et al. 1994; CDC 2001b). A set of RRs from CPS-II was incorporated into the program to develop a smoking attributable fraction (SAF), and users entered mortality, prevalence, and economic cost data into the program for the jurisdiction under study. Web SAMMEC is now used extensively by states and by CDC to provide periodic estimates of SAM and YPLL for adults aged 35 years and older and, separately, for perinatal conditions associated with maternal smoking (CDC 2002d).

In 1997, CDC used national mortality data for 1990–1994 with SAMMEC II, estimating that 2,153,600 deaths (1,393,200 men and 760,400 women) were attributable to smoking over the five years (19.5 percent of all deaths), an average of 430,700 deaths per year (CDC 1997). A total of 906,600 of these deaths were attributed to CVDs, 778,700 to neoplasms, 454,800 to nonmalignant respiratory diseases, 7,900 to diseases among infants, and 5,500 to smoking-related fires. Lung cancer (616,800 deaths), ischemic heart disease (490,000 deaths), and chronic airways obstruction (270,100 deaths) accounted for most of the deaths. During 1990–1994, cigarette smoking resulted in 5,732,900 YPLL before 65 years of age and a total YPLL to life expectancy of 28,606,000. On average, each smoker who dies from a smoking-related disease forfeits 12 to 15 years of life compared with his or her lifetime nonsmoking counterparts (Peto et al. 1992; CDC 1997).

CDC later calculated annual SAM and YPLL estimates for 1995–1999 for the United States (CDC 2002a). Calculated annual estimates of deaths attributed to smoking were 264,087 in men and 178,311 in women (total 442,398) in the United States each year during 1995–1999. Excluding deaths in adults from secondhand smoke, the estimated SAM was responsible for a total annual YPLL to life expectancy of 3,332,272 for men and 2,284,113 for women. Thus, adult male and female smokers dying from smoking lost estimated averages of 13.2 and 14.5 years of life, respectively, compared with nonsmokers. The findings in this study differ from previous SAM estimates (CDC 1993, 1997) and reflect (1) the inclusion of 35,100 heart disease deaths attributable to secondhand smoke; (2) the inclusion of 966 burn deaths from cigarette-caused fires; and (3) declines in current smoking prevalence among men, women, and pregnant women since the early 1990s (CDC 2002a).

In 1996, CDC evaluated a model based on Behavioral Risk Factor Surveillance System data for the projected prevalence of smoking among young adults, the NMFS for death estimates among smokers and former smokers, and projected future SAM based on data from CPS-II. Assuming that one-third of adult current smokers and 10 percent of adult former smokers die from smoking-related diseases, and that current smoking patterns continue without a marked increase in cessation, an estimated 25 million persons (adults and children) alive in 1995 will die prematurely from smoking-related illnesses (CDC 1996a); among persons who were 0–17 years of age in 1995, more than five million are expected to die from smoking attributable causes.

Peto and colleagues (1992) estimated mortality from tobacco use in developed countries using an indirect method that was conceptually similar to the excess mortality method described previously. Using the lifetime nonsmoker lung cancer mortality rates from CPS-II (Stellman and Garfinkel 1986), they calculated the absolute excess mortality rate for lung cancer in all developed countries, and used the observed lung cancer rate in those countries as an index of overall population exposure to smoking. Smoking is the predominant cause of lung cancer, and little else contributes to lung cancer incidence (Thun et al. 1997a). Using the lung cancer rate as the anchoring point, Peto and colleagues (1992) then estimated the relative impact of smoking for several diagnostic categories other than lung cancer by age and gender. A smoking impact ratio was established for these categories (upper aerodigestive cancers, other cancers, chronic airways obstruction, other respiratory diseases, and vascular diseases). The ratio estimated the excess mortality rate for the other disease categories based on the excess lung cancer ratio, but the authors halved the apparent excess for these other categories because it would then provide a reasonable degree of protection against overestimating the epidemic. The adjusted PAR was then

calculated using the smoking impact ratio to obtain a SAM estimate for developed countries.

Using this approach, the SAM for developed countries in 1985 totaled 1.7 million (Table 7-1.3), and was projected at 2.1 million in 1995. This method has been criticized for comparing lung cancer mortality rates for the study populations in various countries with the American lifetime nonsmoker lung cancer mortality rates of participants in CPS-II (Sterling and Weinkam 1987; Lee 1996). In this analysis, the lifetime nonsmoker lung cancer rates were assumed to be similar throughout all populations.

In 2002, the World Health Organization (WHO) released *The World Health Report 2002: Reducing Risks, Promoting Healthy Life* that apportioned deaths worldwide to various risk factors including smoking (WHO 2002). This report estimated that 4.9 million deaths worldwide were attributable to tobacco (8.8 percent of all global deaths), and tobacco was also responsible for 59.1 million lost disability-adjusted life years (DALYs) (4.1 percent of the global total lost DALYs). Compared with 1990, WHO reported at least one million more tobacco-related deaths in 2000, with the highest increases in developing countries (WHO 2002).

Table 7-1.3 Smoking attributable mortality (deaths in thousands), all developed countries, 1985, stratified by age group, gender, and cause

Age/gender	Lung cancer	Upper aerodigestive cancer	Other cancers	Chronic obstructive pulmonary disease	Other respiratory diseases	Vascular diseases	Other medical conditions	All
35-69 years								
Men	203	47	64	71	14	297	78	774
Women	37	4	7	19	3	54	18	141
70 years								
Men	134	19	48	126	15	180	37	561
Women	29	4	6	42	6	72	16	175
All								
Men	338	66	112	197	30	477	115	1,335
Women	65	8	13	61	9	126	34	316

Source: Peto et al. 1992.

Infants and Children

Smoking during pregnancy has serious, adverse consequences that lead to increased risks for death in the perinatal period and to substantial YPLL. Since the early 1990s, a number of estimates have been made related to smoking during pregnancy using the parameter values from the original SAMMEC software, which were set based on the meta-analysis by McIntosh (1984). The four diagnoses and RRs used in the original SAMMEC software included the following:

ICD-9	Description	RR
765	Short gestation, low birth weight (LBW)	1.76
769	Respiratory distress syndrome (RDS)	1.76
770	Respiratory conditions in newborns	1.76
798.0	Sudden infant death syndrome (SIDS)	1.50

CDC commissioned a meta-analysis of literature published through 1999 on the risks of death to infants born to mothers who smoked during pregnancy (Gavin et al. 2001). Gavin and colleagues (2001) estimated pooled and adjusted pooled odds ratios (ORs) for infant/neonatal mortality related to smoking during pregnancy. (The RR for SAM estimates is interchangeable with the OR for rare diseases [Rothman 1986].) The pooled estimates showed a stronger effect of smoking on birth weight and intrauterine growth than on gestational age at birth: OR = 1.75 (95 percent CI, 1.39–2.19) for preterm, small for gestational age (SGA) infants; 1.84 (95 percent CI, 1.48–2.28) for LBW infants regardless of gestational age; and 1.95 (95 percent CI, 1.51–2.51) for SGA infants, including term and preterm infants. The single crude OR for mortality among short gestation, LBW infants found in the literature was in the same range (OR = 1.95 [95 percent CI, 1.29–2.95]). However, after adjustment for other factors, the 95 percent CI for this OR overlapped unity (OR = 1.52 [95 percent CI, 0.98–2.37]). The SAM estimate used the pooled OR (1.84) for LBW, regardless of gestational age, because evidence shows that smoking affects mortality at all birth weights (Wilcox 1993). Although Gavin and colleagues (2001) suggested that most neonatal mortality was captured by the excess

risk associated with LBW, excess mortality attributable to RDS and other respiratory diseases of the newborn is still evident after adjusting for gestational age, which is the major determinant of LBW. The excess risk for RDS deaths is not fully captured by the risk of death from LBW, so it is appropriate to include RDS and other respiratory diseases in assessments of neonatal mortality attributable to smoking. The most recent RRs for these conditions (1.30 for RDS and 1.41 for other respiratory diseases) are from Malloy and colleagues (1992). Although they used a predominantly white population to assess the RRs, these RRs were applied to all populations.

Compared with the quantitative review by Anderson and Cook (1997) on SIDS, the original RR of 1.50 that was used in SAMMEC appears low; a pooled adjusted OR of 2.29 (95 percent CI, 2.03–2.59) for SIDS reported by Gavin and colleagues (2001) was considered more appropriate and was used in the updated SAMMEC version. There is evidence of an increased risk of SIDS from smoking by parents and others during the postnatal period. The additional OR for maternal smoking in the postnatal period, after controlling for prenatal smoking, may be as high as 2.04 (95 percent CI, 1.56–2.68), and smoking by the father or by others in the household during the postnatal period may also increase risk. The data suggest a small independent effect from smoking by fathers or others only in addition to maternal smoking. However, the differences are not statistically significant, and they are not included in the current Web SAMMEC software. The revised RRs for perinatal mortality attributable to maternal cigarette smoking (including respiratory distress and respiratory diseases in newborns) are shown below and are included in Table 7-1.2, in addition to a comparison with ICD-9 categories. These RR values are used in the updated SAM calculations presented in this report.

ICD-10	Description	RR
P07	Short gestation, LBW	1.84
P22	RDS	1.30
P23–28	Other respiratory diseases in newborns	1.41
R95	SIDS	2.29

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