

Effects of the Antismoking Campaign: An Update

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Abstract: In the absence of the antismoking campaign, adult per capita cigarette consumption in 1987 would have been an estimated 79–89 per cent higher than the level actually experienced. The smoking prevalence of all birth cohorts of men and women born during this century is well below that which would have been expected in the absence of the campaign. As a consequence, in 1985 an estimated 56 million Americans were smokers; without the campaign, an estimated 91 million would have been smokers. As a result of campaign-induced decisions not to smoke, between 1964 and 1985 an estimated 789,200 Americans avoided or postponed smoking-related deaths and gained an average of 21 additional years

of life expectancy each; collectively this represents more than 16 million person-years of additional life. The greatest health benefit lies in the future, however, as younger individuals reach the ages at which smoking claims its greatest toll, and as middle-aged former smokers realize relative reductions in smoking mortality risks as a result of long-term abstinence from smoking. For example, campaign-induced decisions not to smoke made prior to 1986 will result in the postponement or avoidance of an estimated 2.1 million smoking-related deaths between 1986 and the year 2000. (*Am J Public Health* 1989; 79:144–151.)

Introduction

Previous papers have presented estimates of the effect of the antismoking campaign between 1964 and 1978 on two measures of smoking behavior^{1,2} and on smoking-related mortality.³ The present paper updates the findings of the earlier studies.

Throughout this paper, the term “antismoking campaign” is used not to connote a specific set of activities oriented toward smoking prevention and cessation, but rather the collectivity of all such activities and the changing social norms that have accompanied them. In essence, “antismoking campaign” serves as a short-hand expression covering the entirety of changes in the social environment spawned by scientific and social interest in the hazards of smoking. A detailed discussion of those changes—in scientific and public knowledge, smoking attitudes and behaviors, and programs and policies—is found in the Surgeon General’s 1989 report on smoking and health.⁴

Methods

Adult per Capita Consumption

An objective measure of the nation’s cigarette smoking behavior, used in numerous empirical studies, is adult per capita cigarette consumption, defined as total cigarette consumption (from the Economic Research Service of the US Department of Agriculture) divided by the population over 17 years of age; its advantages and disadvantages have been discussed elsewhere.⁵ To assess the impact of the antismoking campaign on cigarette consumption, as in the previous analysis¹ (and a predecessor study⁵), I employ ordinary least squares regression analysis on per capita consumption as the dependent variable. Independent variables include those conventionally found in demand analyses, such as price, and a series of variables representing major antismoking “events,” such as publication of the Surgeon General’s first Report on Smoking and Health⁶ and the growth of the nonsmokers’ rights movement. Time series data for the study

cover the period 1947 through 1987. Estimated regression coefficients for the antismoking variables indicate the specific association between each “event” and per capita consumption.

From the regression results, one can also estimate the aggregate impact of the antismoking campaign in any given year by adding the values of all of the relevant antismoking variables, multiplied by their regression coefficients, to the year’s actual per capita consumption. (Since the previous year’s consumption is included in the regression as an independent variable, as a measure of the addiction effect, an antismoking effect is also carried forward into future years through this variable.) The resulting estimates of per capita consumption in the absence of the campaign are then compared to the actual levels of consumption to estimate the aggregate impact of the campaign in the year in question. Details on the specifics of the methodology are found in the previous analysis.¹

Smoking Prevalence in Age- and Sex-Specific Cohorts

The second study of smoking behavior relies on *prevalence*—the most common index of the national level of smoking. In this assessment of the impact of the campaign, age- and sex-specific estimates of actual smoking prevalence derived from self-reported survey data (and therefore labeled “reported” prevalence) are compared with estimates of prevalence rates that would have been expected in the absence of the antismoking campaign (herein referred to as “expected” prevalence).

As in the earlier study,² the population born between 1901 and 1960 is divided into 12 age-sex cohorts, each incorporating 10 birth years. Cohort-specific smoking rates for the years 1964–78 are taken directly from the previous study, which employed retrospective self-report data* derived from the 1978 National Health Interview Survey (NHIS). For the years 1979–85, cohort-specific prevalence data are derived from the 1979, 1980, 1983, and 1985 NHIS.** For the years in which survey data are not available (1981, 1982, and 1984), linear interpolations from the available surveys are used.

To develop estimates of what the various cohorts’ smoking rates would have been from 1964 through 1985, in

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*Jeffrey Harris: Personal communication.

**Office on Smoking and Health, US Department of Health and Human Services: Personal communication.

the absence of the antismoking campaign ("expected" prevalence), the analysis adopts the assumption that well-established smoking patterns or trends prior to 1964 would have persisted into the future. For men, this would mean approximate continuation of the smoking frequencies of earlier cohorts, whereas for women it would mean continuation of the growth in smoking experienced primarily since World War II.

The basic formula for developing these estimates involves adding or subtracting earlier cohorts' average percentage point changes in smoking prevalence, at comparable ages, to the 1963 base rate prevalence of the cohort in question. For example, during 1964 the 1941-50 male cohort consisted of males 14 to 23 years of age. To calculate their expected smoking prevalence in 1964, in the absence of the antismoking campaign, I add to their 1963 prevalence the average of the percentage point changes in the smoking rates of the 1931-40, 1921-30, and 1911-20 male cohorts in the years in which these cohorts were 14 to 23 years old (1954, 1944, and 1934, respectively). To calculate the 1965 expected smoking prevalence for the 1941-50 cohort, I add to the 1964 estimate the average percentage point change of the three comparison cohorts in 1955, 1945, and 1935. The same procedure is repeated for 1966 and ensuing years.

This basic procedure was applied directly to the four youngest male cohorts. For the two eldest male cohorts and all of the female cohorts, variations on the basic procedure or cohort-specific ad hoc procedures had to be applied. These are described in Appendix A.

Reported and expected prevalence rates are converted into estimates of the numbers of smokers, with and without the antismoking campaign, by multiplying cohort-specific prevalence rates by the cohort's population in a given year. For the expected number of smokers in the absence of the campaign, cohort populations are adjusted downward to reflect the differential mortality rates of smokers and nonsmokers. (The needed adjustments are developed in the "deaths postponed" analysis described below.)

Deaths Postponed by Campaign-Induced Decreases in Prevalence

The third analysis estimates the number of smoking-related deaths postponed or avoided as a consequence of campaign-induced decisions to quit smoking or not to start. In the earlier analysis, this health benefit was labeled "premature deaths avoided."³ In the present updating, the terminology is changed to "deaths postponed" to clarify that the health benefit includes both complete avoidance and mere postponement of smoking-related deaths. Interest is in the loss of life expectancy of smokers compared to nonsmokers, whether smoking-related deaths occur at age 50 or age 70.

As in the previous analysis,³ the number of deaths postponed is estimated by applying epidemiologic data on age- and sex-specific mortality rates of smokers, quitters, and never smokers to the cohort-specific changes in smoking prevalence discussed in the preceding section. The reduced mortality rates of quitters (compared with continuing smokers) are multiplied by the estimated number of campaign-induced quitters and non-initiators (i.e., people influenced by the campaign not to start smoking) in each cohort and in each year from 1964 through 1985. This procedure generates estimates of the number of additional smoking-related deaths that would have occurred during those years had the anti-smoking campaign not encouraged quitting and noninitiation. To estimate the number of life-years saved associated with each death postponed, life expectancy gained is calculated by

comparing the former smoker's life expectancy with that of a continuing smoker, using life table analysis.

Much of the postponement of death due to smoking cessation and noninitiation induced by the campaign through 1985 will not be realized until later years. For example, someone who quits smoking in 1985, who would not have experienced a smoking-related death that year, might avoid a smoking-related death that would have occurred 15 years later (e.g., by avoiding the development of lung cancer). By "aging" the population of quitters and noninitiators alive in 1985 and applying to them the differential mortality rates of former smokers, one can estimate the numbers of smoking-related deaths postponed or avoided in the future as a result of quitting and noninitiation that occurred prior to the end of 1985.

Given the need to make several assumptions to develop estimates of deaths postponed, the base-case findings are subjected to four sensitivity analyses. Assumed values of each of four variables are altered in a conservative direction to determine whether the specific values selected for the base case fundamentally affect the qualitative findings of the study. The four sensitivity analyses include:

- running the estimating model with all excess smoker mortality ratios reduced by 50 per cent,
- increasing ex-smoker mortality ratios by 25 per cent,
- reducing estimates of the number of campaign-induced quitters and noninitiators by 25 per cent, and
- allowing for 50 per cent recidivism among one-year and two-year quitters and noninitiators. ("Recidivism" by a noninitiator means that someone influenced by the campaign not to start smoking in a given year decides to start smoking a year or two later.)

The precise estimating procedures for the analysis of deaths postponed from 1964 through 1985 are methodologically identical to those described in an appendix to the original study,³ subject to one refinement. When five-year population age groupings have to be broken up to provide estimates of the population at specific ages in specific years, Karup-King coefficient formulas⁷ are used; the previous analysis relied on simple averaging. Data are identical for both studies for the period through 1978. For the post-1978 period, the new age-sex cohort differences between actual and expected smoking prevalence, described in the preceding section, are used in the mortality analysis, as are more recent life table data. Other parameter values are unchanged from the original analysis.

Results

Adult per Capita Consumption

The regression model detailed in Appendix B indicates which factors reduced per capita consumption. Findings with respect to the significance of individual variables are qualitatively identical (and quantitatively very close) to those reported in the earlier study.¹ For the antismoking "events," this means that significant decreases in per capita consumption ranging from 3.5 to 7.5 per cent occurred during each year of prominent adverse smoking-and-health publicity represented by dummy variables in the regression. These include the first public smoking-and-cancer "scare" of 1953-54, publication of the Surgeon General's first Report on Smoking and Health in 1964,⁶ and the three full years of the Fairness Doctrine period, 1968-70, during which broadcasters were required by the Federal Communications Commission to donate air time to the antismoking message to

“balance” pro-smoking advertising.⁸ The regression also shows a strong inverse correlation between per capita consumption and growth in the number of states having laws restricting smoking in public places. Finally, the regression indicates a price elasticity of demand for cigarettes of -0.2 . This means that a cigarette price increase of 10 per cent is associated with a per capita consumption decrease of 2 per cent.

The aggregate impact of the campaign on per capita consumption is shown in Figure 1, which depicts both actual adult per capita consumption and two patterns of consumption estimated in the absence of the campaign. The dashed line represents an estimate in which price changes are independent of the campaign, while the dotted line reflects the assumption that the pattern of price changes, affected most strongly by state excise taxation of cigarettes, was itself a function of legislators’ reactions to the emerging evidence on smoking and health. The logic underlying the latter assumption is that, influenced by numerous state tax increases, the real price of cigarettes rose eight consecutive years following publication of the Surgeon General’s first report.^{1,9} A period of new-tax inactivity over the next decade has been attributed to concerns about interstate cigarette bootlegging in response to large tax-induced discrepancies in states’ retail cigarette prices.¹⁰ Consistently rising prices again since 1982 have been associated with increased anti-smoking sentiment in state and federal legislatures.⁴

To represent the assumption that the pattern of cigarette price was a function of the campaign, the dotted line treats price as if it had been constant (in real terms) in the years after 1963. This permits an assessment of the consumption effects of price fluctuations possibly produced as a result of the campaign. In contrast, actual experienced prices are reflected in the dashed line. The gap between the solid line (actual consumption) and dashed line (estimated consumption, actual prices) can be interpreted as a measure of the “pure publicity effect” of the campaign, while the gap between the dashed line and dotted line (estimated consumption, constant prices) measures the consumption impact of changing prices.

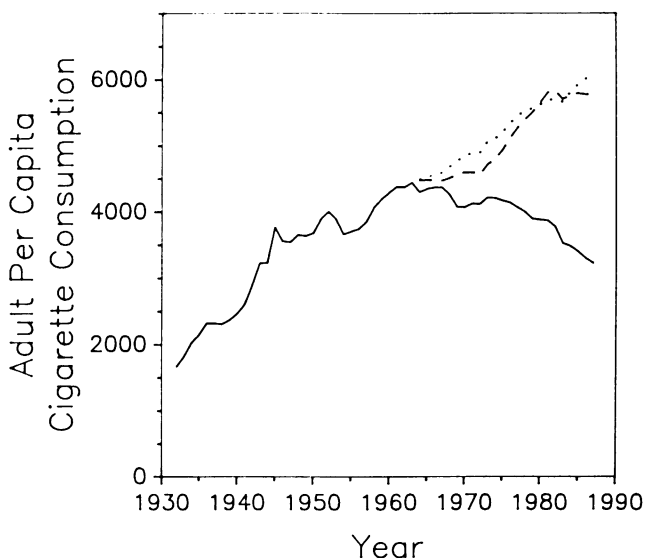


FIGURE 1—Adult per Capita Cigarette Consumption
 Solid line = actual consumption; dotted line = consumption predicted in absence of the antismoking campaign, with price held constant; dashed line = consumption predicted in absence of the campaign, with actual prices experienced

To the extent that the latter is attributable to smoking-and-health concerns of legislators, it constitutes a measure of price-related campaign-induced changes in consumption.

Each of the estimated lines exhibits a generally increasing pattern of consumption over time, in contrast to the pattern actually experienced. The analysis indicates that in 1987, per capita consumption would have been 79 to 89 per cent greater than the level actually experienced, depending on whether price changes are treated as independent of the campaign or as a function of it. The small difference suggests that, by 1987, price fluctuations contributed to decreased consumption, but to a much smaller degree than the “publicity effect.”

Smoking Prevalence in Age- and Sex-Specific Cohorts

Results of the analysis of cohort smoking prevalence are presented in Tables 1 and 2 for men and women, respectively. Each cell records reported smoking prevalence for the relevant birth cohort and the relevant year, from 1964 through 1985, and the estimate of the smoking rate that would have been expected each year in the absence of the anti-smoking campaign.

To illustrate the nature of the findings, Figures 2 and 3 plot the numbers in Tables 1 and 2, as well as pre-1964 prevalence estimates, for two representative cohorts, one male and one female. The gap between the solid and dashed lines constitutes a measure of the impact of the campaign on smoking prevalence for these two birth cohorts.

The data in Tables 1 and 2 indicate that all 12 of the age-sex birth cohorts experienced substantial quitting, or non-initiation of smoking, as a result of the antismoking campaign. By 1985, the estimated gap between actual (reported) prevalence and that which would have been anticipated in the absence of the campaign ranged from a low of six percentage points for the eldest female cohort to a high of 28 percentage points for the youngest males. The percentage point gap is larger for each male cohort than for the same-age female cohort. For women, the percentage point gap in 1985 is larger as one moves from the older to the younger birth cohorts. For men, the gap is smallest for the oldest cohort and largest for the youngest; it is similar for the remaining four cohorts.

The “expected prevalence” figures indicate that a majority of each of the four youngest male cohorts would have been expected to be smokers in 1985 in the absence of the campaign, including two-thirds of men born 1951–60. In fact, fewer than 40 per cent of each cohort reported themselves to be smokers that year. Similarly, in the absence of the campaign, smoking prevalence in the four youngest female cohorts would have been expected to include a majority or near-majority of the women (44 per cent for the 1921–30 birth cohort to 54 per cent for the 1951–60 cohort). Yet each of these cohorts reported prevalence rates of either 27 per cent (1921–30 cohort) or 32 per cent (1931–60 cohorts).

Multiplied by their respective cohort populations, the 1985 reported prevalence figures indicate a total smoking population that year of 56 million Americans born between 1901 and 1970. In the absence of the campaign, the cohort analysis indicates that there would have been 91 million smokers. (The 1961–70 birth cohorts were added to the analysis for this purpose, with prevalence figures taken from the 1985 NHIS. “Expected” prevalence was assumed to be the same as that of the 1951–60 cohorts in 1975.)

Deaths Postponed by Campaign-Induced Decreases in Prevalence

Table 3 presents estimates of deaths postponed for each of the 12 age-sex cohorts identified in Tables 1 and 2, for the

TABLE 1—Smoking Prevalence for Males (%) 1964 through 1985, Reported^a and Expected^a in the Absence of the Antismoking Campaign

Birth Cohorts	Year (19--)																				Expected minus reported 1985	
	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83		84
1901-10																						
Reported	45	45	45	43	42	39	39	36	35	34	31	30	28	27	26	20	18	18	19	19	17	16
Expected	46	46	46	45	44	42	42	40	39	38	37	37	36	36	35	34	32	31	29	28	26	25
1911-20																						
Reported	61	60	59	59	58	54	53	51	50	48	46	46	43	40	39	32	30	29	28	26	24	22
Expected	62	61	60	60	59	57	56	56	55	55	54	54	53	52	51	50	49	48	47	46	45	45
1921-30																						
Reported	63	62	62	61	60	56	55	53	52	51	48	47	47	45	44	42	41	39	36	34	33	32
Expected	66	66	66	65	65	64	64	64	63	63	63	62	62	61	61	59	58	57	56	55	54	53
1931-40																						
Reported	59	59	58	57	56	53	53	51	50	49	47	46	45	44	44	42	44	43	42	40	38	35
Expected	62	63	63	64	64	64	64	63	63	63	62	62	62	61	61	59	58	57	56	55	55	54
1941-50																						
Reported	42	46	50	54	58	58	58	57	56	55	54	53	51	49	47	44	44	43	41	40	39	38
Expected	43	48	53	57	61	61	62	62	63	63	64	64	64	64	64	63	63	62	62	62	61	60
1951-60																						
Reported	1	2	4	7	10	14	18	23	27	31	36	38	40	40	39	40	42	41	40	40	39	38
Expected	2	3	5	8	11	16	21	26	32	39	44	50	54	58	61	62	64	65	65	66	66	66

^aSee text for definition of "reported" and "expected" prevalence.

period 1964 through 1985. The estimated total number of deaths postponed as a result of campaign response is 789,200. Of these 112,400 occurred in 1985 alone (not shown in table). The distribution between men and women and across birth cohorts is seen in the individual cells of the table. All told, the 789,200 postponed deaths represented a savings of 16.3 million life-years, an average of 20.6 years per death postponed.

Between 1964 and 1985, Americans born between 1901 and 1960 experienced an estimated 5.7 million smoking-related deaths. In the absence of the campaign, 6.5 million deaths would have been expected. The estimated 789,200 deaths postponed or avoided represented 12 per cent of the anticipated mortality toll.

Table 4 indicates that 2.1 million deaths will be postponed between 1986 and the year 2000 as a result of campaign-induced nonsmoking that occurred through 1985.

The data in the table reflect the distribution between men and women and across birth cohorts.

Aggregate results of the sensitivity analyses for the two periods (1964-85 and 1986-2000) are presented in Table 5. Although they reduce the magnitude of estimated deaths postponed, the essential qualitative finding of the study is robust: as a result of campaign-related decisions to quit smoking or not to start, many hundreds of thousands of Americans have extended their lives an average of two decades each.

Discussion

Few people question the fact that the antismoking campaign has altered smoking behavior. Adult smoking prevalence has fallen from 43 per cent at the time of the

TABLE 2—Smoking Prevalence for Females (%) 1964-1985, Reported^a and Expected^a in the Absence of the Antismoking Campaign

Birth Cohort	Year (19--)																				Expected minus reported 1985	
	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83		84
1901-10																						
Reported	22	21	21	20	20	19	18	17	17	16	15	15	13	13	13	13	15	13	10	8	8	8
Expected	22	21	21	20	20	19	19	19	19	18	18	18	17	17	17	17	16	16	15	15	14	14
1911-20																						
Reported	36	36	36	36	35	33	33	31	31	30	29	28	27	26	26	25	26	24	22	20	19	18
Expected	37	37	37	37	36	36	36	35	35	35	35	35	34	34	34	34	33	33	32	32	31	31
1921-30																						
Reported	43	43	43	42	42	40	40	39	39	39	38	38	38	37	36	31	31	30	30	29	28	27
Expected	44	45	46	47	48	48	48	49	49	49	48	48	48	47	47	47	47	46	46	45	44	44
1931-40																						
Reported	44	44	44	44	44	43	43	43	42	42	42	42	41	40	39	39	35	34	34	34	33	32
Expected	45	46	46	47	47	48	48	48	49	49	50	50	50	51	51	51	51	51	50	50	49	49
1941-50																						
Reported	25	30	34	38	40	40	41	41	41	41	41	40	39	38	37	35	34	34	33	33	32	32
Expected	27	32	36	40	42	44	46	47	48	49	50	50	51	51	52	52	52	52	52	52	52	52
1951-60																						
Reported	1	1	2	3	6	10	13	17	22	27	31	35	38	38	37	34	32	33	34	34	33	32
Expected	1	2	3	5	8	12	15	20	25	31	36	40	45	47	49	50	51	52	53	53	54	54

^aSee text for definition of "reported" and "expected" prevalence.

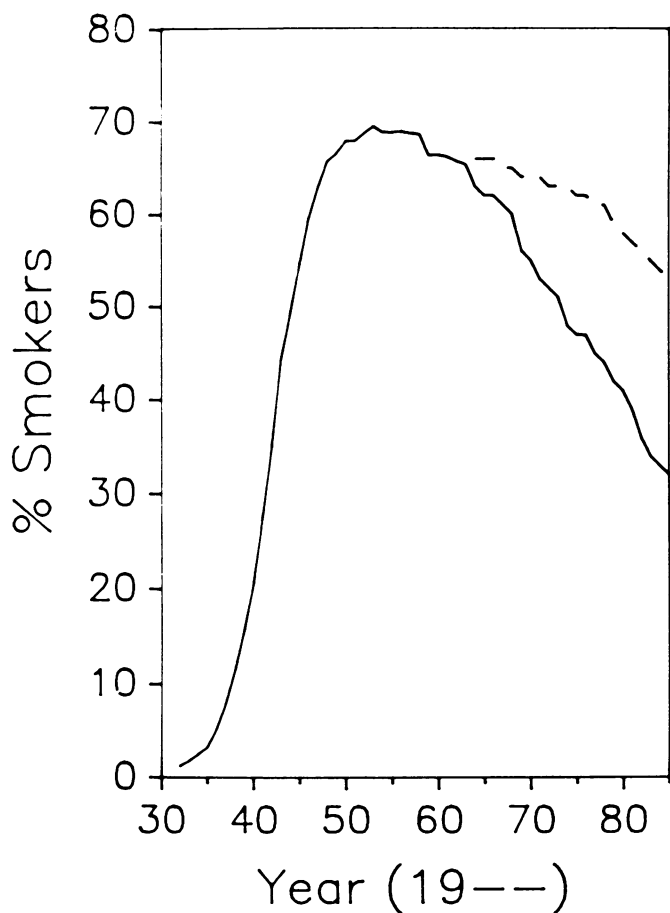


FIGURE 2—Smoking Prevalence History, 1921–30 Male Birth Cohort
Actual prevalence (solid line) and expected prevalence for 1964–85 in absence of the antismoking campaign (dashed line)

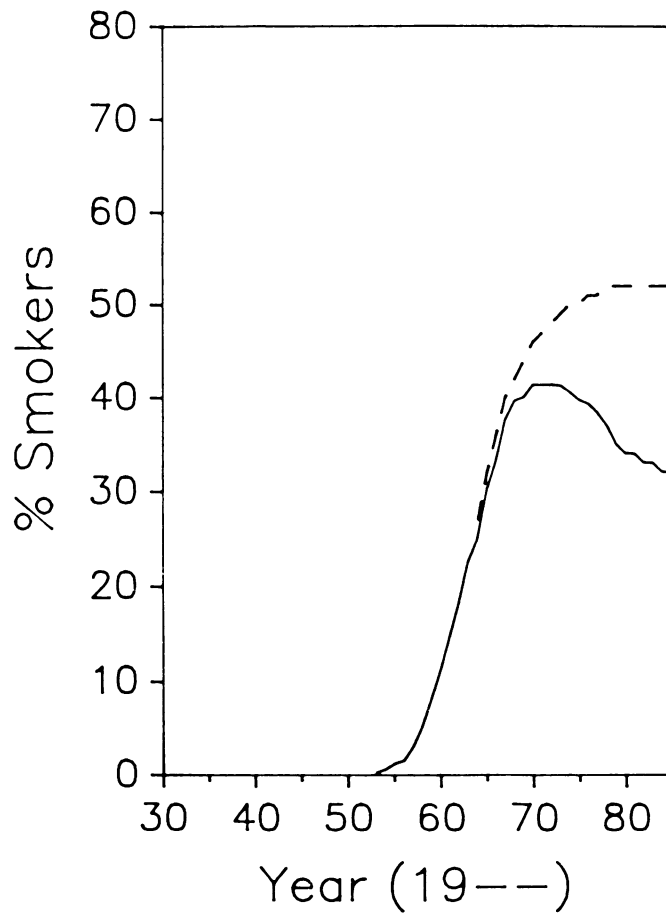


FIGURE 3—Smoking Prevalence History, 1941–50 Female Birth Cohort
Actual prevalence (solid line) and expected prevalence for 1964–85 in absence of the antismoking campaign (dashed line)

Surgeon General's first report on smoking to under 30 per cent a quarter century later; prevalence among men has almost halved. Per capita cigarette consumption has fallen annually since 1973, without exception, and is at its lowest level in 45 years; per capita tobacco consumption has reached its lowest level in a century.⁴

Yet such measures understate the impact of the anti-smoking campaign, since they miss the critical point that smoking prevalence and per capita consumption likely would have been rising in the absence of the campaign. For example, per capita consumption in 1987 was 27 per cent below the level that prevailed in 1963, the year prior to the Surgeon General's first report. But analysis of per capita consumption trends indicates that the 1987 value was actually less than half of what it would have been had the antismoking campaign never transpired.

This analytical perspective is most important in understanding the pattern of women's smoking. The relatively steady trend in adult female smoking prevalence from 1964 through the late 1970s, followed by relatively modest decreases in the 1980s, often has been interpreted as reflecting little response to the antismoking campaign. Yet as the cohort analysis demonstrates, in the absence of the campaign, women's smoking prevalence would have continued increasing, paralleling the diffusion pattern of smoking among men several decades earlier. As such, the stability of female

smoking prevalence after the mid-1960s, at a level far below that attained by men, should be interpreted as behavioral response to the antismoking campaign.

Consistent with the observation that the campaign has affected smoking by men more than by women, the cohort analysis indicates that the percentage point gap between reported smoking prevalence and prevalence expected in the absence of the campaign is larger for each male cohort than for the same-age female cohort. Missed in this observation, however, is the comparability for men and women of the numbers of campaign-related quitters and noninitiators relative to the numbers of actual smokers. In 1985, this measure

TABLE 3—Deaths Postponed by Campaign-related Smoking Cessation and Noninitiation, 1964–85 (in thousands)

Birth year	Males	Females	Total
1901–10	103.6	16.7	120.3
1911–20	182.0	46.0	228.0
1921–30	182.7	59.6	242.3
1931–40	83.2	22.7	105.9
1941–50	44.0	15.5	59.5
1951–60	29.0	4.2	33.2
Total	624.5	164.7	789.2

of relative quit-and-nonstart rate—the ratio of the percentage point gap to actual prevalence—indicates little difference between males and females.

Growth in the gap between reported and expected smoking prevalence as one moves from the older to the younger cohorts, especially among the women, is consistent with the conventional wisdom that decisions not to start smoking are easier than decisions to quit. Decisions concerning the initiation of smoking occurred after 1964 for many members of the two youngest cohorts and well before 1984 for the older cohorts.

The peak rate of prevalence of the youngest cohort of males (42 per cent in 1980) makes this cohort the first group of men born during the 20th century never to have included a majority who were smokers. Supportive of the large gap found between this cohort's reported prevalence and the estimate of prevalence in the absence of the campaign are data on the peak prevalence of each of the older cohorts reported by Harris¹¹ [and unpublished data]. In the space of a single 10-year period, peak prevalence fell 16 percentage points, from 58 per cent to 42 per cent (for the 1941–50 and 1951–60 birth cohorts, respectively). In the span covered by three 10-year birth cohorts, the maximum percentage of men smoking fell by almost 30 percentage points (from 70 per cent for the 1921–30 cohort). Preliminary data from the 1987 National Health Interview Survey, combined with other NHIS data from the 1980s, suggest that the peak prevalence for the cohort of males born 1961–70 may represent a further decrease of as much as 12 or 13 percentage points, to a rate of 29 to 30 per cent.

The 70 per cent peak prevalence rates attained by the 1911–30 male cohorts suggest that the expected peak prevalence figures for males in Table 1 may be conservative. This perception is reinforced by recognition that the 1921–30 cohort achieved its peak rate of smoking in 1953, the year of the first major public smoking-and-cancer scare, and that the succeeding cohorts reached peak smoking age during periods of concern about the health consequences of smoking.

In this context, had women's smoking patterns eventually mirrored those of men approximately three decades earlier, the expected prevalence figures in Table 2 would have to be considerably larger than they are. As such, the gaps between reported and expected prevalence for women should be considered conservative measures of the impact of the antismoking campaign on women's smoking prevalence.

The deaths postponed analysis found that, by 1985, the 789,200 smoking-related deaths postponed or avoided represented 12 per cent of the smoking-related mortality that would have occurred in the absence of the antismoking campaign. In the predecessor to this study, the comparable figure through 1978 was 5 per cent.³ The life-savings attributable to the campaign will continue to rise in the coming

TABLE 5—Sensitivity Analyses

Sensitivity Test	Deaths Postponed (thousands)	
	1964–1985	1986–2000
1. Reduce excess smoker mortality ratios by 50%	519.8	1,416.1
2. Increase ex-smoker mortality ratios by 25%	581.1	1,876.5
3. Reduce campaign-induced quitters and noninitiators by 25%	592.8	1,547.7
4. Allow 50% recidivism among 1- and 2-year quitters and noninitiators	406.0	1,044.8

decades, both in absolute terms and as a fraction of total smoking-related mortality, as the cohorts most responsive to the campaign reach the ages at which smoking claims its greatest toll; in the more immediate future, campaign-related life-savings will also rise as older cohorts achieve the health benefits of long-term abstinence from smoking. This is demonstrated by the estimate of 2.1 million deaths postponed between 1986 and the year 2000 among those who had quit smoking or not started through 1985. The benefits of the campaign through 1985 alone substantially understate the long-term health dividend of campaign-induced behavioral changes that have already taken place.

The analysis concluded that men have realized a much greater collective health benefit from their responses to the antismoking campaign than have women. This finding reflects more men than women having quit and not initiated smoking as a result of the campaign, and women having lower death rates and smoker mortality ratios than men. As women's smoking behaviors have approached those of men in recent years, the disparity between male and female smoker mortality ratios has diminished.⁴ As such, this study's conservative assumption about women's smoker mortality ratios may have produced an underestimate of the health benefit of the campaign for women. Particularly in the younger cohorts, if women respond to the relatively new emphasis on the health consequences of their smoking,¹² the male-female differential of premature deaths avoided would be expected to decrease in the future.

The analysis of deaths postponed presents an incomplete picture of the health consequences of the antismoking campaign because it ignores the smoking-related morbidity and disability avoided. Moreover, it does not address directly one of the major behavioral impacts of the campaign: the shift from the unfiltered cigarette of the 1940s and '50s to filtered cigarettes and, more recently, to the low tar and nicotine (t/n) product.⁴

On the surface, this profound change might appear to represent a positive contribution to diminishing the toll of smoking. Yet recent evidence indicates that smokers compensate for reduced nicotine yields, often unconsciously, by such methods as smoking more cigarettes, inhaling more deeply, taking more puffs per cigarette, and blocking air ventilation holes in the filters.¹³ Low-yield cigarette smokers are also exposed to additives that may introduce new health risks.¹⁴ Further, the availability of filters and low-yield cigarettes may have encouraged many smokers to continue smoking when they otherwise would have quit. It is also possible that they have encouraged certain groups to start smoking in larger numbers, perhaps most notably young

TABLE 4—Deaths Postponed 1986–2000 by Campaign-related Smoking Cessation and Noninitiation through 1985 (in thousands)

Birth year	Males	Females	Total
1901–10	18.8	5.9	24.7
1911–20	194.6	83.8	278.4
1921–30	429.5	162.6	592.1
1931–40	315.3	128.6	443.9
1941–50	290.5	109.8	400.3
1951–60	241.7	76.3	318.0
Total	1490.4	567.0	2057.4

females.¹⁵ Each of these possibilities implies increases in smoking-related morbidity and mortality relative to that which would have occurred in their absence. As such, the net health consequences of changes in the cigarette product are unknown.

The most obvious concern about smoking is the continuing population of more than 50 million smokers. While, in the absence of the campaign, the number of smokers today would exceed 90 million, the remaining population of smokers assures that the enormous burden of smoking will persist into the 21st century. The fact that tens of millions continue to smoke, when the vast majority of them wish that they did not, illustrates the tenacious grip that nicotine places on its users.¹⁴ This sobering reality places the impressive successes of the antismoking campaign in context, but it does not diminish them.

APPENDIX A

Cohort-Specific Procedures to Estimate Smoking Prevalence in the Absence of the Antismoking Campaign

The text explained the basic procedure for estimating cohorts' smoking prevalence rates in the absence of the antismoking campaign. For the two oldest male cohorts and for each of the female cohorts, deviations from the basic procedure were required. Additional minor adjustments were also required for the most recent years of the study.

For the more recent years of the study, comparison cohorts often reached comparable ages *during* the antismoking campaign. For example, the three comparison groups for the 1951–60 male cohort were the 1911–40 cohorts. To use the 1931–40 cohort in estimating the 1951–60 cohort's expected prevalence in 1984 and 1985 would place the former at comparable age in 1964 and 1965, the first two years of the campaign. Thus, for these two years, the prevalence changes of the two older cohorts alone were employed. In general, whenever a comparison cohort was of comparable age during the campaign, it was dropped from the analysis and the remaining one or two cohorts' prevalence changes were used.

When adequate comparison groups were not available, cohort-specific ad hoc procedures were adopted. In the instance of the 1921–40 male cohorts, this was needed only to estimate expected prevalence in 1984 and 1985. In both cases, the average decrease of the three preceding years was used.

For the 1911–20 male cohort, the basic procedure was followed through 1978 (using only the 1901–10 cohort as the comparison group, and making minor adjustments to compensate for the problem of understatement bias discussed by Harris¹¹). Thereafter, expected prevalence was decreased 0.924 percentage point per year, the average of the annual declines from 1950–69 for the 1901–10 cohort.

The 1901–10 male and female cohorts had no useful comparison groups, since retrospective reporting of smoking histories in 1978 by people born before 1901 produces a significant underestimation bias¹¹; in addition, the smoking histories of these earlier generations were not "models" for successor cohorts. Thus, for 1964–78, the expected prevalence rates were estimated by taking an arbitrary reduction of 50 per cent in the reported percentage point decrease in the rate of smoking. For the men, for expected prevalence after 1978, no additional differential change was assumed; i.e., an annualized rate of decline was selected to make the gap between expected and reported prevalence the same in 1985 as it was in 1978. For the women, a three-point decline

between 1978 and 1985 was assumed, as it was for the 1911–30 female cohorts during these years. This decline was more gradual than for the same-age men, but the men's prevalence figures (both reported and expected) were higher than the women's in all cases.

For the earlier years (1964–78), there was no useful comparison group for the 1911–20 female cohort, since their smoking behavior differed radically from that of their predecessors, among whom smoking was quite rare. Arbitrarily, the cohort's 1956–63 smoking rate (37 per cent) was continued through 1967 and then decreased at a decelerated rate compared to the decline in reported smoking prevalence.

For the 1921–30 female cohort, given the emergence of the women's liberation movement, with its smoking manifestations, we assumed a continuing gradual increase in smoking prevalence through 1971, at which point a peak rate of smoking 49 per cent was assigned. For the remaining years, a very gradual rate of decline in expected prevalence was assumed.

For the three youngest female cohorts (born 1931–60), the basic estimation method was employed for the years 1964–78, subject to a slight modification: we assumed that the intercohort upward trend in peak smoking prevalence would have led these cohorts, in the absence of the antismoking campaign, to reach maximum smoking rates slightly above 50 per cent. This seemed a conservative assumption, given that actual peak prevalence for males of earlier cohorts exceeded 60 (and even 70) per cent.

For the years after 1978, the basic method did not apply, for lack of adequate comparison groups. Each of these three female cohorts was treated differently, reflecting their age differences. For the eldest (1931–40), expected prevalence was decreased by a total of two percentage points. Same-age males' expected prevalence fell by seven points but remained higher (at 54 per cent) in 1985 than that of the females (49 per cent). The more gradual decline among women seemed appropriate in light of the later diffusion of smoking among women and the later age of peak prevalence for this cohort.

For the 1941–50 female cohort, expected prevalence was held constant at 52 per cent during 1979–85. For the same-age males, expected prevalence fell slightly (64 to 60 per cent). Again, the later diffusion of smoking among women and the later age of actual peak prevalence made this assumption seem reasonable and conservative, since expected prevalence generally remained about 10 points below that of the same-age males.

For the youngest group of women (1951–60), expected prevalence growth of five points from 1978 through 1985 was assumed, equaling the expected prevalence growth for the same-age men and taking expected prevalence to 54 per cent, fully 12 percentage points below that of the same-age men.

Since, of necessity, many of these patterns of expected prevalence involved arbitrary judgments, their overall reasonableness was assessed by aggregating them and comparing them with the estimates of adult per capita consumption described in the text. This procedure suggested that the assumed patterns are reasonable in their representation of smoking prevalence trends in the aggregate. Obviously, the procedure cannot be used to determine whether individual cohorts' patterns are reasonable.

The major use of this cohort analysis, in evaluation of the number of deaths postponed as a result of campaign-response, includes a sensitivity analysis in which the gaps between actual and expected prevalence are diminished by 25 per cent. As such, the principal qualitative conclusions drawn

from the analysis of prevalence effects of the campaign are unlikely to be affected by possible misjudgments in evaluation of expected prevalence rates.

A few additional details on the estimating procedures, including explanations of why they are believed to be conservative, are presented in the appendix to the predecessor study² and in a document available from the author on request.

APPENDIX B

Adult per Capita Consumption Regression

The per capita consumption regression results are as follows:

$$C_t = -3565.2 - 7.83*P_t + 0.74*C_{t-1} + 1390.7*Y_t - 153.66**D53_t - 279.37*D54_t - 181.36*D64_t - 158.58**D68_t - 278.17*D69_t - 140.63**D70_t - 23.92*NSTATES_t R^2 = .976$$

[* p < .01; ** p < .05]

where — C_t is adult per capita cigarette consumption in year t (mean = 3944)

— C_{t-1} is lagged consumption, which captures the effect of addiction

— P_t is relative real cigarette price in year t (index with $P_{1967} = 100$; mean = 92.32)

— Y_t is the natural logarithm of the last two digits of year t and reflects increases in the smoking population due to the diffusion of the behavior, particularly among women, and increases in smokers' daily consumption levels (mean = 4.18)

— $D53_t$, etc., are dummy variables, for the years indicated by the digits (except for $D54_t$), which provide estimates of the impact on consumption during these years of major antismoking activity (each = 1 in the year indicated by the digits, 0 otherwise)

— $D54_t$ is 0 before 1954 and $0.5^{(t-1954)}$ in 1954 and thereafter, a dummy which equals 1 in 1954 and has a continuing, though rapidly diminishing, additional effect that reflects additional publicity through the mid-1950s

— $NSTATES_t$ is the number of states having smoking restriction laws in effect in year t, a proxy measure for the effectiveness of the nonsmokers' rights movement (mean = 20.19)

The variables and data sources are identical to those used in the previous study,¹ except that the proxy measure of the success of the nonsmokers' rights movement in the earlier study was the percentage of the adult population residing in states with laws restricting smoking in public places, calculated by the author. The new variable was preferred because it comes from an independent source¹⁶ (updated by the Office on Smoking and Health).

As noted in the text, findings with respect to the significance of individual variables are qualitatively identical to those reported in the previous study. The relatively small differences in the estimated coefficients in the two studies primarily reflect the addition of nine years to the period studied. The most striking difference reported in the Results section is the apparent reduction (in absolute value) in the price elasticity of demand from -0.37 to -0.2 . While this

might reflect a real change in the elasticity, representing a smoking population that is older and more addicted, it could also simply reflect the vagaries of time series analysis. Both figures are within the range of price elasticities found in other studies.⁴

The Results section also reports the strong inverse correlation between consumption and growth in the number of states with laws restricting smoking in public places. As observed in the previous study, this correlation should not be interpreted as implying that passage of nonsmokers' rights laws leads to reductions in cigarette consumption.¹ In fact, a new econometric study has concluded that the direction of causality might be the opposite; that is, decreases in consumption may be causing the growth in nonsmokers' rights legislation.¹⁷

Readers interested in further details on the interpretation of the regression results should consult the previous paper.¹

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