# A new perspective on the evolution of cigarette consumption

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## Abstract

**Objectives:** Population exposure to smoking health-risks depends on the popularity and duration of the smoking habit and the quantity and quality of consumed cigarettes, all of which vary dramatically over time and across generations. We propose the first smoking indicator that combines these co-variates.

**Methods:** Using retrospective reports on smoking behavior, a time-series of cigarette tar yields, and novel methods, we create a standardized indicator of smoking prevalence. Our data produce nationally representative life-course smoking trajectories of eleven generations of US men and women, spanning more than 120 years of history. We also use detailed mortality data to construct, for each generation and gender, the corresponding trajectories of smoking-attributable mortality and we examine their association with the smoking patterns.

**Results:** Relative to unstandardized measures, our data describe a different history of smoking diffusion in the US and better predict later-life mortality. We show that, among recent cohorts, smoking health-risk exposure is at a historic low and will account for less than 5% of deaths.

**Conclusion:** We produce a more exact smoking indicator to better monitor smoking patterns, forecast mortality effects, and inform public health policies.

## 1. Introduction

The last two decades have seen a global tobacco control treaty and enormous sums spent on anti-smoking campaigns worldwide. Despite these efforts, the estimates of smoking-attributable mortality increased from 3.5 (1) to 5 million deaths annually(2), and the latest forecasts suggest that this toll will exceed 8 million by 2030. Progress in reducing smoking prevalence has been generally slow. For example, since the US launched its high profile initiative *Healthy People* in 1990, it has frequently set ambitious targets to limit tobacco consumption but has consistently failed to fully meet those targets. Public health officials are especially concerned because per capita consumption is now greater in developing than developed countries and continues to increase further, creating expectations of waves of smoking-caused deaths in the decades to come(3).

To frame policy discussions about the relationship between current smoking and future health, researchers commonly rely on the "cigarette epidemic" model. That model describes stylized patterns of aggregate smoking prevalence and its co-evolution with smoking-related mortality(4,5). It suggests that cigarette demand and supply initially increase with economic development but, as the population becomes more educated, health information spreads, and governments adopt anti-smoking policies, cigarette consumption eventually declines. Importantly, the hump-shaped pattern in overall smoking prevalence is followed by a similar pattern in smoking-attributable mortality a few decades later. This observation suggests that policy makers can minimize the long-run costs of smoking if they limit its uptake before it becomes a widespread social habit. However, the model yields no detailed policy prescriptions.

On a more practical level, researchers study the smoking-mortality relationship by generating smoking histories of different demographic groups and using them in micro-simulation models to estimate health costs. Among the most state-of-the-art models are those developed by the Cancer Intervention and Surveillance Modeling Network of the National Cancer Institute, e.g. to test the impact of tobacco control policies on smoking-related deaths(6-8). Albeit more useful for policy planning, these models suffer three limitations. First, they rely on retrospectively reported data to generate smoking histories but use an outdated method to account for the differential mortality bias inherent in this type of data. Second, none of the models take into account temporal changes in cigarette quality. Third, no model combines all available information on smoking into one comprehensive measure that can be used as a macro-level monitoring device of smoking diffusion. Although some research describes concepts of smoking intensity(9,10), no available indicator accounts for cigarette quality or has extensive sample coverage.

We address all three limitations. While we use retrospectively reported smoking data, we process those data with a novel technique that uses time-varying cause-specific mortality data. We also account for temporal changes in cigarette quality with time-series on average tar yields per cigarette. While some public health researchers discount this indicator, it is the only available indicator that is consistently measured and, in fact, it is understudied. With the data at hand, we produce nationally representative life-course smoking trajectories that are standardized across eleven generations of US men and women and cover more than 120 years of history. Our standardized rates are directly comparable across time and capture the four main aspects of individual smoking behavior that contribute to smoking-related mortality:

the popularity and duration of the smoking habit, and the quantity and quality of consumed cigarettes. We also use widely available age, sex, and cause-specific mortality data to construct, for each generation and gender group, the corresponding trajectories of smoking-attributable mortality. Next, we contrast smoking patterns and the smoking-mortality association between the naive and standardized data; we identify the duration of cigarette consumption associated with the highest probability of premature death; we explore changes in the smoking-mortality relationship over time; and we project smoking-attributable mortality forward.

## 2. Standardized Smoking Prevalence Rates

We start from an established approach in the public health literature that studies inter-generational smoking patterns from a life-course perspective(6,7,11-19). Using retrospectively collected smoking data and techniques described in (20,21), we calculate the share of smokers in successive 10-year birth-cohorts of men and women and in every year of each cohort's life. Our calculations account for the fact that smokers are less likely to survive for interview than non-smokers, and measure this differential mortality using cause-specific death rates that vary over time, across generations, and between genders (see supplementary text). We plot the resulting data in fig.1A-B which shows that, as cohorts grow older, smoking rates increase to a peak and then fall, reflecting dynamics in smoking initiation and cessation.

Overall, the trajectories match well with previous evidence from US data adjusted for differential mortality bias in a similar, albeit more restricted, technique; i.e., by relying on external but time-invariant mortality rates by smoking status (11,12). Our trajectories are less consistent with evidence presented in studies that correct for differential mortality indirectly, by relying on model calibrations, without

using actual data on smoking-related deaths(6,7,13). These studies also assume that differential mortality is time-invariant and, in comparison to our results, they over-estimate smoking prevalence rates by as much as 10 percentage points.

Despite differences in level estimates, all studies describe smoking patterns that follow the narrative of the cigarette epidemic model. They show that the popularity of smoking rose across successive generations and peaked for men born between 1915-1924 and women born between 1935-1944. Smoking prevalence fell among subsequent cohorts, though in the youngest cohort of women smoking rates still exceed those of the oldest cohort.

An advantage of the cohort-specific smoking trajectories is that they convey information about the duration of the average person's smoking habit. In cohorts whose trajectory has higher kurtosis, people smoked fewer years. Clearly, the average smoker in the older cohorts smoked more years than the average smoker in younger cohorts. This inter-generational differential in smoking persistence creates reasonable expectations of a corresponding differential in smoking-attributable mortality, which one misses when pooling all cohorts together. However, cohort-specific rates are not comparable across years and generations because they do not reflect the changing quantity and quality of cigarettes. To address this problem, we use data on the number of cigarettes each person (retrospectively) reported having smoked each day and data on the sales-weighted average tar yield of cigarettes sold in each year. With these data, we re-define a smoker to be a person who smokes 20 cigarettes per day, each of which contains 37 mg of tar, and we reconstruct cohort smoking trajectories using this standardized measure of smoking status (see supplementary text).

Although some researchers assert that the cigarette tar content data are a noisy (and biased) measure of the tar smokers actually inhale and, therefore, a flawed

measure of cigarette quality, these data serve our intended purpose. Criticisms derive from two independent observations: first, that the relative health-risk from smoking increased over the period that machine-measured tar content of cigarettes was falling; and, second, that smokers of "light" or "low-tar" cigarettes increase the intensity, duration, and frequency of puffs to compensate for the lower nicotine or tar content - a fact not captured by machine measurements(22). But these separate observations do not establish that, faced with declining tar yields, smokers adjusted their smoking behavior by so much that they caused relative health-risk from smoking to increase. In fact, no study explicitly shows that changes in the tar content of cigarettes (or any other indicator of cigarette quality) has caused higher smoking-related mortality (we discuss this issue in more detail in the supplementary text). Given that the tar yields are measured with a consistent method over time and absent strong evidence to the contrary, we use these data as a proxy for the true underlying data on delivered tar. At the very least, we assert that the data reflect a trend towards less harmful cigarettes. A priori, we are agnostic as to whether this assumption is correct and to what degree this indicator will predict smoking-related mortality. Our analysis in Section 3 tests that empirical question.

In fig.1C-D we show that naive and standardized rates narrate substantially different smoking histories. The standardized smoking rates of the four older cohorts of men and all cohorts of women are substantially lower because they typically smoked fewer than 20 cigarettes per day. In contrast, the standardized smoking rates of men born between 1915-1934 are higher because they smoked more than 20 cigarettes per day. Finally, the standardization causes the right tail of the smoking trajectories to shrink to the left in all cohorts alive after 1950 because starting in that year cigarette manufacturers began to reduce cigarette tar content. The variation in the

standardized data better reflects developments that influence cigarette demand and supply; e.g. the substantial reduction in the cost of cigarettes that occurred after 1881, when James Bonsack invented the cigarette rolling machine, changes in social norms about women smoking, and the arrival of scientific evidence of the health consequences of smoking which, among others, pushed manufacturers to supply "healthier" cigarettes (see (23) and references therein).

To illustrate differences more clearly, in fig.2 we plot the peak unstandardized and standardized smoking prevalence rate by gender and cohort (and by education level in fig.S1 and fig.S2). Although the correspondence is not exact, a cohort's peak smoking prevalence approximates that cohort's share of ever-smokers. Thus, one can read the lines plotted here as describing smoking diffusion across successive generations instead of calendar years. Compared to the unstandardized data, the standardized rate peaks higher for men and lower for women by 7 percentage points. But while unstandardized rates peak for men two cohorts sooner than for women, standardized rates reach a maximum in the same cohort for both genders; i.e., those who were born over 1925-1934 and who became adults in the decade following World War II (WWII). In subsequent cohorts, standardized rates fall much faster than unstandardized rates, and eventually achieve closer convergence across genders. Most importantly, standardized peak prevalence falls to levels lower than those observed at the start of the 20th century, showing that population exposure to smoking-health risks of recent US cohorts is largely overstated by unstandardized data. For example, among men born over 1975-1984 there are three times fewer (standardized) smokers compared to men born over 1987-1884. We find this pattern even though we do not measure consumption of tobacco products other than cigarettes. Such products were especially popular until around 1940 when cigarettes began to dominate the

market(13). This implies that the difference in the exposure to tobacco health-risks between the youngest and oldest cohorts is in fact larger than that shown in fig.2.

## 3. The smoking-mortality relationship

While increasingly fewer people smoke, the health consequences of smoking are still unfolding. We demonstrate this in fig.3 by plotting gender-cohort trajectories of smoking-attributable deaths as a proportion of total deaths. We construct these data using an established method which relies on widely available vital statistics(24). Like smoking rates, smoking-attributable mortality in each cohort also follows a hump-shaped pattern but that pattern occurs a few decades later. In our sample, individuals born after 1965 are still younger than 65 - the age smoking-attributable mortality rates start to emerge. Consequently, smoking-attributable mortality is virtually zero for that group. Sex-differences in death rates are less pronounced than those in the corresponding smoking rates due to the widely-documented gender differential in overall mortality.

With these data we explore how fast and to what degree smoking translates to mortality and whether the standardized prevalence better predicts this relationship. First, we correlate the cohort-specific smoking-related mortality with the corresponding standardized and unstandardized smoking prevalence 10-40 years earlier. In fig.4 we show that this correlation is always higher for women relative to men, again reflecting that women are less likely than men to die from causes unrelated to smoking. As the temporal distance grows between the years mortality and smoking are measured, the correlation increases to a maximum value, after which it gradually falls. For both genders, standardized prevalence more strongly predicts smoking-attributable mortality than unstandardized prevalence. We find that a 1

percentage point increase in the fraction of men who smoke (20 cigarettes per day, each containing 37 mg of tar) in a given cohort is associated with an increase in smoking-attributable death rates of that cohort that peaks at 0.64 percentage points 21 year later. The corresponding peak for women is at 1.15 percentage points 24 years later.

Although we cannot claim that these correlations are causal, they are net of several other measurable influences. Throughout the period of study, the US has seen an impressive improvement in overall quality of life, an expansion in the coverage of the health-system, and progress in medical knowledge and technology with differential impact across types of disease, including several milestones in cancer treatment (25). These factors have affected smoking-related mortality both in absolute terms and relative to total mortality, irrespective of smoking rates. To account for such factors, when we derived smoking-mortality correlations we controlled for gross domestic product (GDP), population, government spending on health care as a share of GDP, and government spending on health care per capita (plotted in fig.S3). Inclusion of such controls plausibly causes the correlations to peak and then fall faster across different temporal lags (fig.S4). Regardless, relative to the unstandardized measure, the standardized prevalence more strongly predicts smoking-related mortality. In fact, this result is robust to even more restrictive model specifications that include time/age trends and fixed-effects and, therefore, may over-control for unobserved variation (figs.S5-S6).

An alternative way to explore the effect of unobserved mortality determinants is to test whether the smoking-mortality correlation varies over time. We test this hypothesis by using every year from 1945 to 1965 as a threshold to repeatedly break our sample into two periods. For each of the two samples we then estimate separate

correlation coefficients. Tab.1 shows that, relative to the later period, in the early period the correlation reaches its maximum sooner (i.e. when prevalence and mortality are separated by fewer years). Stated differently, smokers in the later period take longer to die and the delay increases somewhat with the threshold year. Importantly, the delay is either smaller or zero when one uses the unstandardized prevalence.

For example, consider the estimates for 1964 - the threshold year associated with the longest delay. These estimates suggest that, around 1964, a change in some (unobserved) factor caused the gap between smoking prevalence and the onset of smoking-related mortality to widen by 8 years for men and 4 years for women (fig.S7). At the same time, this change also significantly increased the strength of the correlation between the standardized prevalence and smoking-related mortality (rising from 0.61 to 0.83 for men and from 0.91 to 1.13 for women). We identify three events that could relate to this result. In 1965, Congress created Medicare and Medicaid (26), which guaranteed access to health insurance for individuals age 65 and older, low-income families, and vulnerable population groups. In the same year, researchers discovered robust anti-cancer effects of combination chemotherapy(27-29), which later developed into a standard treatment for lung cancer(30). Finally, in the late 1960s, the long-term decline in total mortality rates began to accelerate dramatically(31). By giving a larger share of smokers access to better treatment, it is plausible that the two former events caused smoking-related mortality to take longer to unfold (given that many of the smoking-attributable diseases, including lung-cancer, are treatable but not curable). All else equal, the latter event caused mortality risk of smokers relative to non-smokers to increase. The fact that these patterns are

not apparent in the unstandardized smoking data adds further evidence that the standardized measure better describes the US smoking history.

To highlight the policy relevance of our results, we use them to predict future smoking-attributable mortality. For simplicity, we rely on the maximum estimates of the smoking-mortality correlation, as indicated by the vertical lines in fig.4. Under a number of alterable assumptions (see supplementary text), these estimates allow us to predict mortality rates both during the period in which smoking data are available and for over two future decades; in this case, until the late 2020s. Focusing on the six cohorts that may survive until that time, fig.5 shows that their predicted mortality trajectories replicate the observed trajectories fairly accurately. The forward projections show that the smoking-attributable mortality rates of women will exceed those of men starting with the cohort born in 1955-1964 and that, for both genders, smoking-attributable mortality will fall dramatically across generations. In fact, we project that fewer than 5% of individuals who are currently in their mid 30s will die from smoking-attributable causes.

## 4. Discussion

We propose the first standardized indicator of smoking prevalence that reflects not only how popular smoking is but also how long smokers smoke, and the quantity and tar content of the cigarettes they smoke. Our indicator suggests that US cigarette consumption spread, peaked, and contracted faster than people commonly perceive. In fact, among the youngest cohorts, exposure to smoking health-risk is at a historic low.

Our analysis highlights the shortfalls of the cigarette epidemic model (see notes of fig.1). We show that the usefulness of this model is limited because it relies on a single dimension of smoking: its popularity. It ignores the duration, frequency,

and quality of smoking that also increase smoking health risks. This model is also based on stylized facts derived from the US experience alone, which reflects specific market and social conditions that could be much different in other countries and contexts. For example, governments in less developed countries sometimes close their markets to imports and/or try to delay the flow of information on smoking health-risks. In such countries, smoking rates may form a plateau that persists for several decades instead of the more narrow hump-shaped pattern suggested by the US experience. It is also possible that countries undergo a revival of smoking popularity after a period of decline due to social changes that may even be sex or group specific (e.g. a wave of feminism). We discuss such examples in other work(32).

Whenever possible, a policy maker should be guided by analysis of detailed country-specific data which can accurately predict mortality of specific demographic groups. Our paper also contributes to the efforts made to this direction via micro-simulation models that use smoking histories reconstructed from retrospective reports. First, we show that when researchers do not adjust smoking histories for smoking-related differential mortality with external time-varying data, they significantly over-estimate smoking rates. Second, we show that a richer measure of smoking prevalence, one that recognizes temporal variation in smoking participation, smoking duration, cigarette consumption, and machine-measured tar yields, better predicts future smoking-attributable mortality than unadjusted smoking prevalence data. Our measure also reveals a delay in premature death from smoking that is consistent with the increasing population access to progressively more effective treatments.

Although here we use the US context to showcase our approach, one can apply it for every country that has the necessary data. Promisingly, retrospective reports on smoking are regularly collected in a growing number of countries, including emerging

economies which are at the forefront of policy discussions (e.g. Korea, Indonesia, China, India, Russia, Ukraine, and South Africa). Information on the tar content of cigarettes is also publicly available in many countries, as this is often regulated centrally (e.g. starting in 1979 Spain began to regulate the advertising of tar and nicotine content, imposed a limit of 15 mg per cigarette in 1992 and lowered it to 12 mg per cigarette in 1997). Given data availability, our proposed standardization offers scientists and officials in both developed and developing countries the opportunity to better monitor smoking rates and associated mortality effects of different cohorts and, thus, to design more appropriately targeted anti-smoking interventions.

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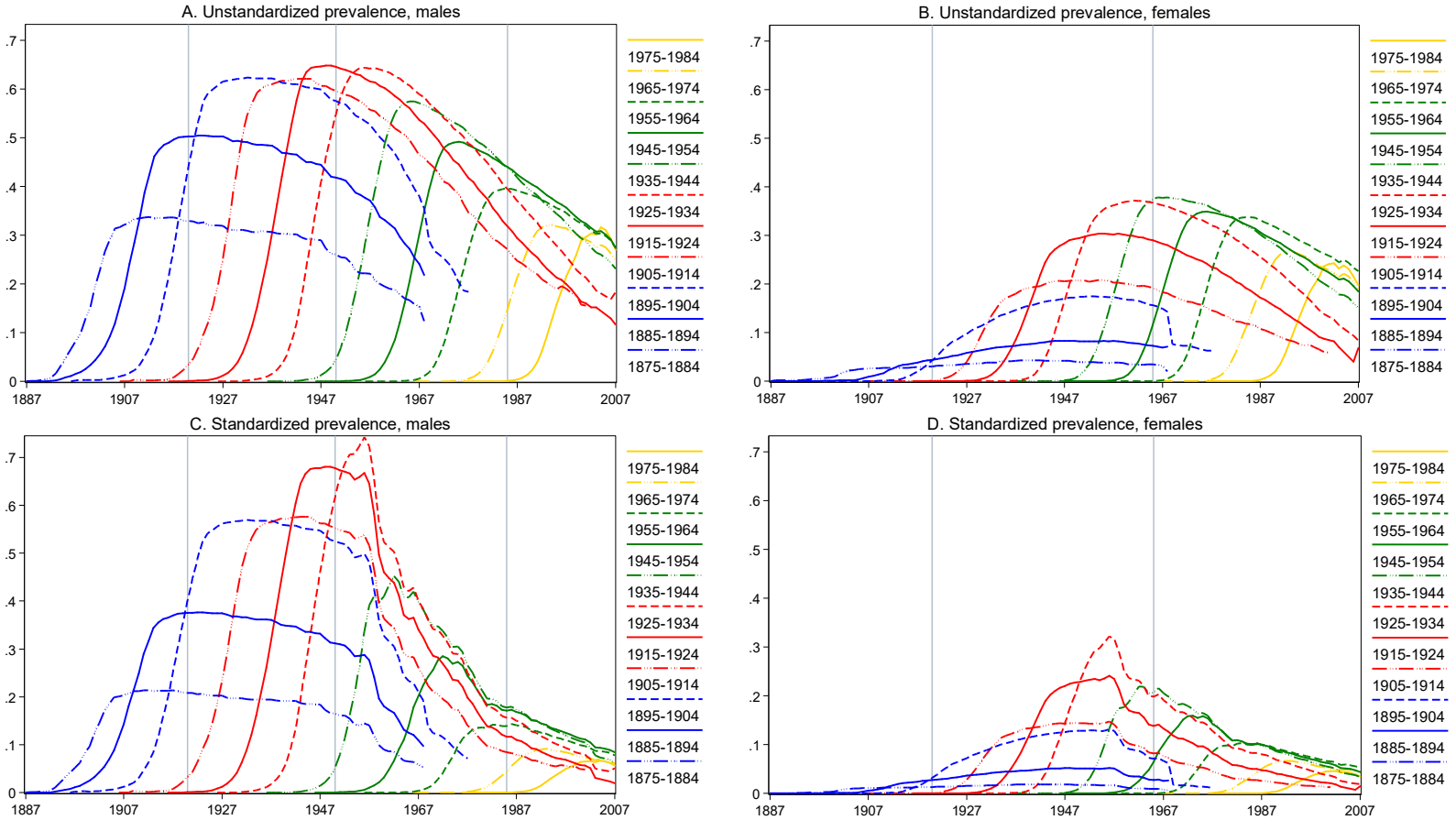
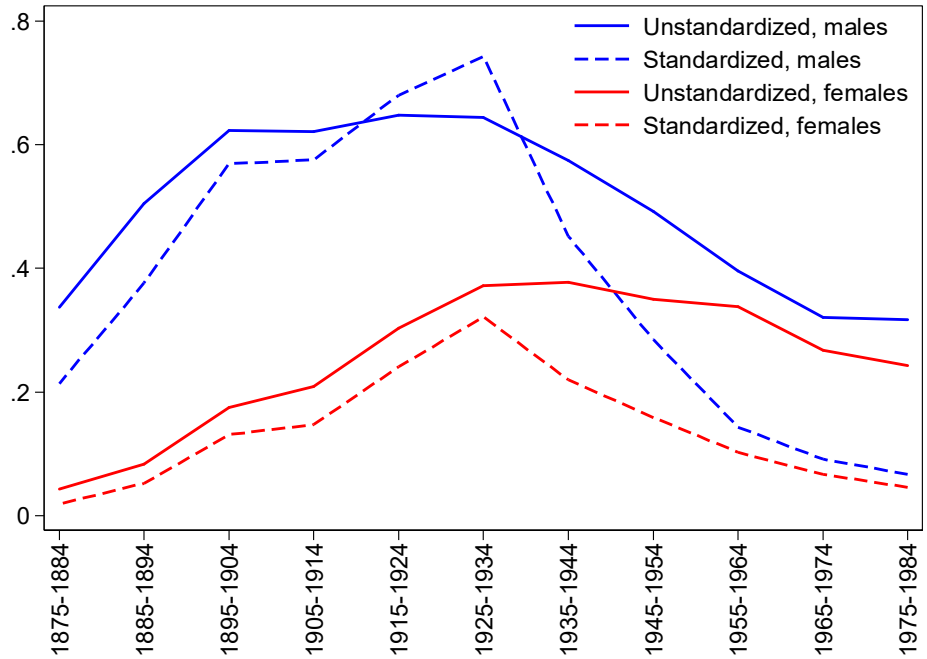
Figure 1: Life-course trajectories of unstandardized and standardized smoking prevalence by gender and birth-cohort



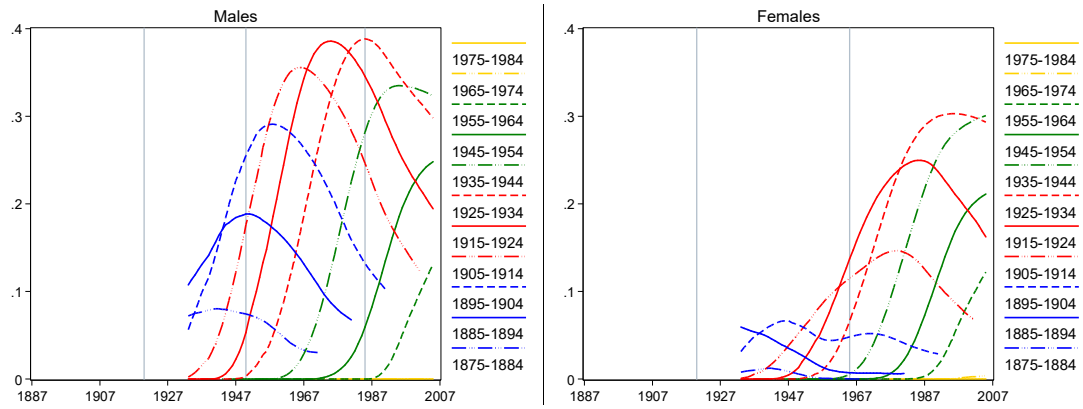

Note: For each cohort-year observation, standardized prevalence=unstandardized prevalence*(cigarettes/20)*(tar density/37), where cigarettes is the average consumption by cohort-year based on retrospective reports and tar density is the US year-specific average. For more information on data and methods see the supplementary text. Vertical lines represent the boundaries between the stages of the cigarette epidemic, as defined by Thun et al. (5). Those stages are timed in specific periods across which aggregate smoking prevalence and smoking-attributable mortality co-evolve. In stage I smoking diffusion is low and smoking-related mortality almost zero; in stage II smoking prevalence escalates rapidly but related mortality is still low; in stage III smoking prevalence starts falling but mortality spirals to its peak; and, finally, in stage IV both smoking and mortality rates fall to lower levels. It is clear that standardized smoking rates do not follow these stages for women, for whom stage III starts after smoking rates have been falling for a decade.

Figure 2: Peak standardized and unstandardized smoking prevalence by gender and birth-cohort



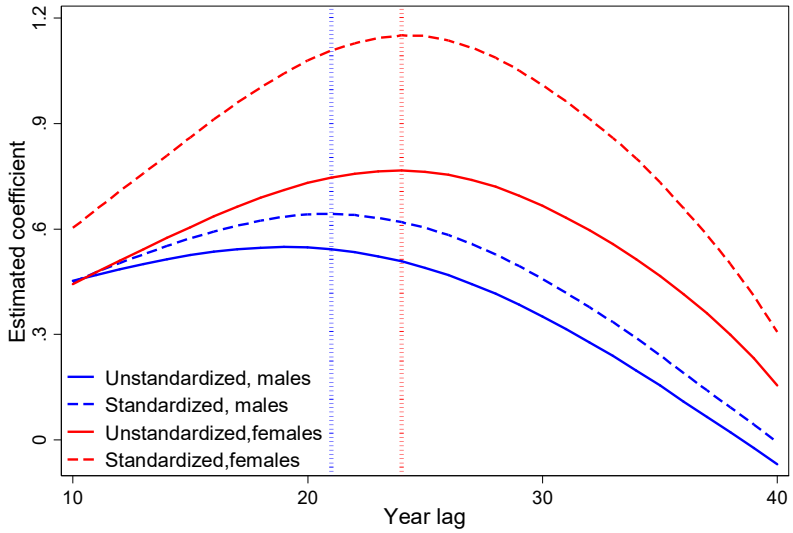

Notes: Differences between standardized and unstandardized prevalence rates are always significant at the 1% level. See tab.S1 for descriptive statistics and sample sizes.

Figure 3: Life-course smoking-attributable deaths (as a % of total deaths) by gender and birth cohort



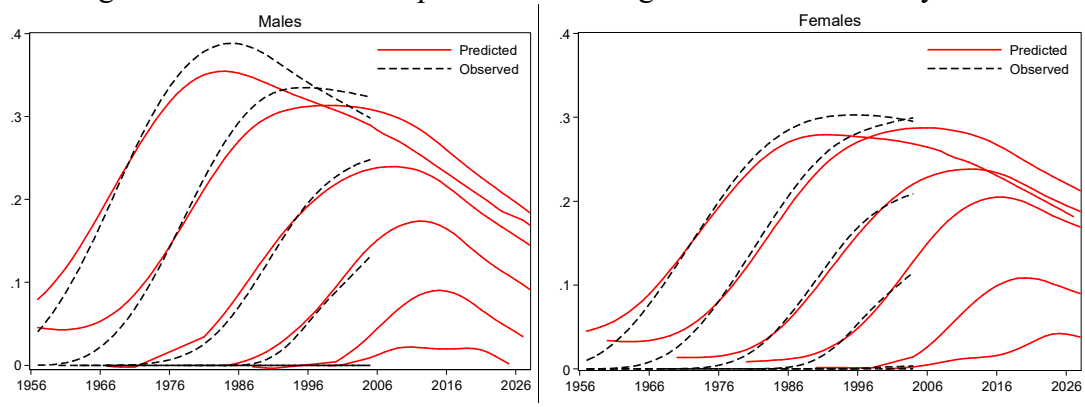

Note: Vertical lines represent the boundaries between the stages of the cigarette epidemic, as defined by Thun et al.(5). For additional information on data construction, see the supplementary text and tab.S4.

Figure 4: Estimated coefficients from regressions of smoking-attributable mortality rates on standardized and unstandardized smoking prevalence at different temporal lags




Note: Each point of each line represents a coefficient from a different regression. All regressions use year-cohort observations and control for spending as a share of GDP, GDP, spending per capita, and population, all observed at t and t+lag (see also tab.S2-S3).

Figure 5: Observed versus predicted smoking-attributable mortality rates




Notes: Lines from left to right correspond to cohorts born in 1925-1934, 1935-1944, 1955-1964, 1965-1974, and 1975-1984, respectively.

Table 1: Temporal gap separating mortality and smoking prevalence data that maximizes the mortality-prevalence correlation by sub-period

| | Men | | | | | | Women | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Temporal gap using unstandardized smoking prevalence | | | Temporal gap using standardized smoking prevalence | | | Temporal gap using unstandardized smoking prevalence | | | Temporal gap using standardized smoking prevalence | | |
| Year Y | Before Y | After Y | Delay | Before Y | After Y | Delay | Before Y | After Y | Delay | Before Y | After Y | Delay |
| 1945 | 23 | 23 | 0 | 23 | 28 | 5 | 27 | 24 | -3 | 27 | 28 | 1 |
| 1946 | 22 | 23 | 1 | 23 | 27 | 4 | 27 | 23 | -4 | 27 | 28 | 1 |
| 1947 | 22 | 22 | 0 | 22 | 27 | 5 | 27 | 23 | -4 | 27 | 27 | 0 |
| 1948 | 22 | 22 | 0 | 22 | 26 | 4 | 27 | 24 | -3 | 27 | 27 | 0 |
| 1949 | 21 | 23 | 2 | 23 | 28 | 5 | 27 | 24 | -3 | 27 | 29 | 2 |
| 1950 | 22 | 23 | 1 | 22 | 28 | 6 | 26 | 24 | -2 | 26 | 28 | 2 |
| 1951 | 22 | 23 | 1 | 22 | 27 | 5 | 26 | 23 | -3 | 26 | 28 | 2 |
| 1952 | 22 | 23 | 1 | 22 | 27 | 5 | 26 | 25 | -1 | 26 | 27 | 1 |
| 1953 | 21 | 25 | 4 | 22 | 26 | 4 | 25 | 25 | 0 | 26 | 27 | 1 |
| 1954 | 21 | 24 | 3 | 23 | 29 | 6 | 26 | 24 | -2 | 26 | 29 | 3 |
| 1955 | 22 | 24 | 2 | 22 | 28 | 6 | 25 | 24 | -1 | 26 | 28 | 2 |
| 1956 | 21 | 23 | 2 | 22 | 27 | 5 | 26 | 24 | -2 | 26 | 28 | 2 |
| 1957 | 21 | 26 | 5 | 22 | 27 | 5 | 26 | 26 | 0 | 26 | 27 | 1 |
| 1958 | 21 | 25 | 4 | 21 | 27 | 6 | 25 | 25 | 0 | 26 | 27 | 1 |
| 1959 | 21 | 25 | 4 | 21 | 28 | 7 | 25 | 25 | 0 | 26 | 28 | 2 |
| 1960 | 21 | 24 | 3 | 22 | 28 | 6 | 25 | 24 | -1 | 25 | 28 | 3 |
| 1961 | 21 | 24 | 3 | 22 | 27 | 5 | 25 | 23 | -2 | 26 | 27 | 1 |
| 1962 | 21 | 26 | 5 | 22 | 27 | 5 | 25 | 26 | 1 | 26 | 27 | 1 |
| 1963 | 21 | 25 | 4 | 22 | 26 | 4 | 25 | 25 | 0 | 25 | 26 | 1 |
| 1964 | 20 | 25 | 5 | 21 | 29 | 8 | 24 | 25 | 1 | 25 | 29 | 4 |
| 1965 | 20 | 24 | 4 | 22 | 28 | 6 | 24 | 24 | 0 | 25 | 29 | 4 |

Notes: Results are from regressions of smoking-attributable mortality rates on yearly unstandardized and standardized sex-cohort-specific smoking prevalence rates with controls for period-specific cohort intercepts.