Supplementary material for: On the Validity of Historical Smoking Data Reconstructed from Retrospective Reports

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1 Background and literature

There is a great social benefit to public health when researchers produce better data or develop methods that allow one to correct for known sources of bias in existing data. Given the lack of historical statistics on smoking prevalence, such efforts have been particularly important for research on smoking behavior. Starting with Harris [\(1\)](#page-12-0), researchers have been using data on lifetime smoking behavior that people report retrospectively to estimate historical patterns of smoking prevalence. Typically, these studies draw data from surveys that collect information about the timing of smoking initiation and, if relevant, cessation. To create life-course smoking trajectories, they assume that a person smoked in each year from the age she started until either the age she quit or, for current smokers, the year of the survey. With these data they construct a smoking-status indicator for every person-year observation. The indicator equals 1 in a year a person smokes and 0 if she does not. By averaging this smoking status indicator, they compute aggregate trajectories of smoking prevalence rates in each year for different population groups of interest (e.g. by gender, education, or birth cohort).

Using simple, often descriptive, analyses of these data, an extensive literature generates important evidence that policy makers can use to evaluate whether past and present tobacco control policies have been effective $(2-17)$ $(2-17)$ $(2-17)$. The evidence also informs the design of future tobacco control policies such as anti-smoking campaigns that may be targeted to particular demographic groups or policies such as bans on the sale of cigarettes to youth that aim to reduce rates of smoking initiation. Using somewhat more elaborate analysis, other studies have relied on retrospective data to estimate whether individual smoking behaviors vary with price $(18-23)$ $(18-23)$ $(18-23)$, whether smoking diffusion predicts mortality $(24, 25)$ $(24, 25)$ $(24, 25)$, and whether the smoking habit is determined by culture [\(26\)](#page-14-3).

Given the usefulness of historical smoking data reconstructed from retrospective reports, the research community has long grappled with the question of the potential bias that these data may carry. Such bias can be due to (i) bad recall, including heaping patterns in reporting; (ii) lack of information on temporary quits; (iii) missing data on infrequent or light smokers; and (iv) differential mortality of smokers and non-smokers. Of the four sources of potential bias, the latter is of greatest concern for descriptions of smoking trajectories because it potentially occurs in every single year. The other types of bias are mostly limited to the endpoints of each individual's smoking trajectory.

When trying to understand the validity of historical smoking prevalence rates constructed from retrospectively reported survey data one must account for the fact that smokers die sooner than non smokers. Due to this differential mortality, in any given sample of people who survive to answer a survey, one may underestimate smoking prevalence rates, especially for older cohorts. Consequently, resulting policy recommendations may be unreliable.

Harris (1) was one of the first researchers to draw attention to this issue. With standard life-table techniques, Harris adjusted smoking prevalence of eight US cohorts derived from retrospectively reported data, using relative mortality of smokers and non-smokers from a sample of US veterans. He concluded that unadjusted prevalence is biased downwards, but the extent of the bias varies with each cohort's age when surveyed. Although Harris presented confidence intervals, he did not formally test whether the unadjusted and adjusted rates statistically differ. Further, his results relied on mortality rates from an unrepresentative population (US veterans), and on the assumption that the relative mortality of smokers and non-smokers does not change over time. Given extensive evidence that relative mortality rates of smokers and non smokers vary considerably over time, by sex, and across countries [\(27\)](#page-14-4), Harris's assumptions are subject to critisism.

The substantial literature that follows Harris generally acknowledges but only sometimes corrects for the potential bias in life-course smoking trajectories of older birth cohorts. In one set of studies, researchers recognize the problem but do not document the extent of the bias or whether/how it varies across dierent birth cohorts $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$ $(5, 8-10, 13-15)$. In a second set, researchers try to reduce bias by selecting samples that are likely less to be affected by differential mortality, typically excluding the very old from the analysis $(6, 7)$ $(6, 7)$ $(6, 7)$. However, sample selection rules (age cutoffs) are ad hoc or mostly informed by Harris's results. In a third set of studies, researchers correct for differential mortality using either Harris's formula or close variants thereof $(2-4, 11, 12)$ $(2-4, 11, 12)$ $(2-4, 11, 12)$ $(2-4, 11, 12)$ $(2-4, 11, 12)$ $(2-4, 11, 12)$ $(2-4, 11, 12)$. This last set suffers the same shortcomings of Harris - they use time-invariant correction factors derived from unrepresentative populations which are often constant among people in broadly defined age-categories.

2 Correcting the record

In their recent paper, Bilal et al. [\(28\)](#page-14-5) assert that no researchers have assessed the validity of methods used to construct historical smoking prevalence rates from retrospectively reported data. If that assertion was true, the contribution of their paper to the literature would be undisputably important. However, their assertion is false and their contribution overstated.

First, there is a large literature that not only investigates this question but that also attempts to validate retrospectively reported data of all types. For example, Berney and Blane [\(29\)](#page-14-6) validate retrospectively reported data on socio-economic status as long as 50 years in the past. Researchers have been validating retrospectively reported data on alcohol consumption for more than 30 years [\(30,](#page-14-7) [31\)](#page-15-0).

Second, there is a robust literature focused precisely on the validity of retrospective data on smoking histories. Some of this literature focuses on specific populations, such as pregnant women [\(32\)](#page-15-1). Other research validates data from the broader population of smokers [\(33,](#page-15-2) [34\)](#page-15-3) and nationally representative samples of the general population [\(35,](#page-15-4) [36\)](#page-15-5). In earlier studies, researchers validated individual smoking behavior using unique samples that had measured smoking status for the same individuals in previous years. For example, Krall et al. [\(37\)](#page-15-6) validated retrospectively reported smoking status using a sample of individuals who had been surveyed 20 and 32 years prior to the survey that collected the retrospective report. Pershagen and Axelson [\(38\)](#page-15-7) validated reported smoking behavior using medical records.

More recently, researchers validate smoking status over very short periods by measuring cotinine levels in blood samples (for a review see Connor et al. [\(39\)](#page-15-8)).

Third, there are at least two previously published studies that have used the very same method as Bilal et al. to validate historical smoking prevalence rates [\(35,](#page-15-4) [36\)](#page-15-5). That is, they compare contemporaneously measured smoking prevalence rates with the rate for the corresponding year that is estimated using retrospective data. Not only do these two studies apply the method Bilal et al. later follow, they do so using data that are better in both scope and detail. Bilal et al. validate smoking prevalence in just 5 earlier years, using data from survey pairs that differ at most by 20 years. The previous studies validate historical smoking prevalence rates using retrospectively reported US data collected up to forty years after contemporaneous smoking prevalence rates were measured [\(36\)](#page-15-5). Further, Kenkel et al. [\(35\)](#page-15-4) validate historical prevalence rates against contemporaneously measured rates in 11 different prior years. Christopoulou et al. (36) do so for 27 prior years. An important feature of the prior work of Kenkel et al. and Christopoulou et al. is that those studies provide historical smoking prevalence rates by sex and birth cohort. By constructing prevalence rates at the total population level, Bilal et al. mask potentially important differences in behavior across gender and birth cohorts - differences that have important public health implications for predicting how future disease, morbidity, and mortality rates will evolve for those groups. Admittedly, Bilal et al. transcend the work by Kenkel et al. by comparing contemporaneously measured smoking prevalence rates with the equivalent rates from retrospective data after correcting the retrospective rates from smoking-related differential mortality. But this exercise is not a new contribution. Rather it replaces the method used by Christopoulou et al. Further, as we explain below, Bilal et al. use the Harris method to adjust their historical smoking prevalence rates for differential mortality rates of smokers and non smokers, a method which Christopoulou et al. have rendered obsolete.

Finally, there is also a wider literature that Bilal et al. sidestep. This literature concerns itself with issues of measurement error in general, and therefore relates to all four types

of bias that retrospective smoking data may carry. For example, an important paper by Hausman, Abrevaya, and Scott-Morton [\(42\)](#page-16-0) theoretically establishes that measurement error in a binary dependent variable leads to biased inferences when the dependent variable is measured with error. They propose a method by which one can correct for the bias. Kenkel et al. [\(43\)](#page-16-1) apply this method to data on smoking initiation that they construct using retrospectively reported smoking data. They document, using the same method of comparing retrospective and contemporanous measured smoking data, that some of the bias occurs because people who smoke few cigarettes in early life do not identify themselves as smokers later in life (light-smokers bias). Other researchers examine measurement error that occurs when people retrospectively misreport the timing of smoking cessation, most often "heaping" their reports on units evenly divisible by 5 (heaping bias). Bar and Lillard [\(44\)](#page-16-2) review the related literature, document the bias, and propose a method to mitigate it.

It is possible to defend Bilal et al.'s claim that the validity of reconstructed historical smoking prevalence rates has not been assessed before if one qualifies their statement by adding the phrase, "..., using Spanish data." We know of no other published study that has used Spanish data on contemporaneously measured smoking prevalence from some previous year to validate historical smoking prevalence rates constructed from retrospectively reported data. Another contribution of Bilal et al is that they estimate the correlation between the reconstructed prevalence rates with lung cancer mortality rates (as a proxy for smokingattributable mortality) measured 25, 30, 35, 40, and 45 years after the year of the estimated smoking prevalence. But this contribution does not test the validity of the historical smoking prevalence data. Instead it estimates the reduced form association between current lung cancer rates and past rates of smoking, as suggested by the "tobacco epidemic model" $(40, 41)$ $(40, 41)$ $(40, 41)$. The resulting correlations may be useful, though the degree of policy relevance is weakened by numerous unstated assumptions one must invoke to rationalize a reduced form model that captures an assuredly complex set of underlying structural relationships between smoking and disease. The more immediate point is that the exercise does not test the central claim of the paper - it does not test whether or by how much measurement error exists in the historical smoking prevalence rates.

3 Adjustment for differential mortality on retrospective data

3.1 The extension of the Harris method by Christopoulou et al.

We mentioned above that Bilal et al. use an outdated method to adjust their historical smoking prevalence rates for differential mortality rates of smokers and non smokers, and they restrict their analysis to smoking rates aggregated at the population level. In this section we further scrutinize these limitations by summarizing how Christopoulou and coauthors, in their 2011 article, extend the method developed by Harris to take advantage of better data that have become available since his 1983 work.

Briefly, Christopoulou et al. use age, sex, and cause-specific mortality data to relax the assumption that the ratio of the mortality rates of smokers and non smokers is constant over time and within broad age-categories. To do so, they follow the Peto et al. [\(45\)](#page-16-4) method to calculate the number of smoking-attributable deaths for each smoking-related disease by sex, year, and 5-year age category (if age>35). The Peto et al. method is straightforward to implement as it requires only widely available vital statistics, and its validity has been confirmed against other methods $(46, 47)$ $(46, 47)$ $(46, 47)$. They then compute the death rate of non-smokers as the difference between all deaths and smoking-attributed deaths divided by the total population, and the death rate of smokers as the ratio of total deaths of smokers to the total population. They use these data to calculate survival probabilities by standard life-table techniques. The Christopoulou et al. article also provides formal tests to determine for which demographic groups historical rates likely deviate from "true" rates.

The failure of Bilal et al. to reference the earlier work exposes a limitation in the design

of their and other similar studies in the "tobacco epidemic" literature. In particular, that literature (typically) proposes to use population smoking prevalence rates to identify the stage a particular country occupies in the epidemic in a given year. Setting aside the plethora of assumptions on which the tobacco epidemic model rests, the use of a population smoking prevalence rate masks important differences in rates of smoking prevalence over time, between genders, and across countries. This point has been recently acknowledged by the authors of the recently updated "epidemic" model (41) , who note the need to separately analyze smoking patterns of men and women, especially for developing countries. But this point was already made indirectly in the Christopoulou et al. not only with respect to gender but also with respect to age. They formally test, for six cohorts defined by sex and year of birth in three countries, whether the mortality adjusted prevalence rates statistically differ from the unadjusted rates each year of each cohort's life. Their main finding is that differential mortality matters only for men who are 70 and older when the retrospective smoking histories are collected. They also show that, for the US, UK, and Russian samples they use, the mortality adjusted prevalence rates do not differ from the unadjusted rates for women in any year.

Importantly, the research and policy implications one derives from the aggregate analysis of Bilal et al. and the disaggregate analysis of Christopoulou et al. are substantively different. Bilal et al find that the mortality correction has a major impact on the reconstructed smoking prevalence rates of men in the early decades of their study, and no statistically impact in all other cases. Based on these results and their perception of the complexity of the correction method, Bilal et al. conclude that it is unnecessary to correct for differential mortality in studies that reconstruct recent smoking prevalence rates. By constrast, Christopoulou et al. show that the impact of differential mortality on historical smoking rates depends on the age of respondents at interview and much less so on the time period of study. The implications of this finding is especially important for studies that rely on the growing number of surveys which focus on respondents who are 50 years old or older and ask them to retrospectively report on past smoking behavior (e.g. the US Health and Retirement Study, Survey of Health, Ageing, and Retirement in Europe, English Longitudinal Study of Ageing, Korean Longitudinal Study of Ageing, China Health and Retirement Longitudinal Study, and Longitudinal Ageing Study of India). Though Christopoulou et al. do not discuss what their findings imply for the tobacco epidemic model, it is fairly easy to recognize that policy makers will be better informed by smoking prevalence histories that are measured for much more specific groups than just gender as Thun et al. (41) suggest.

Christopoulou et al. have made freely available (i) computer codes (and detailed guidelines) that others can use to implement their algorithm, and (ii) historical smoking trajectories adjusted for smoking-related differential mortality by gender and birth-cohort for a group of ten countries (this material is available at: http://smoking-research.ehe.osu.edu).

3.2 Applying the Christopoulou et al. method to Spanish data

We next use data from the Spanish National Health Surveys to demonstrate the Christopoulou et al method and to showcase the added information one gets when one constructs sex- and cohort-specific life-course smoking trajectories. Because Christopoulou et al. describe the method in detail, we only briefly describe the data and then present results.

We compute life-course smoking prevalence using retrospective collected data from the Spanish National Health Survey (SNHS: 1995, 1997, 2001, 2003, and 2006). To correct for smoking-related differential mortality we draw cause-specific death and population data from the World Health Organization mortality database. These data start in 1951. Earlier cause-specific mortality data are not available; our calculations for the years before 1951 use overall mortality data from the Human Mortality Database (HMD). To calculate the number of smoking-attributable deaths for each smoking-related disease we use nonsmokers from the Cancer Prevention Study II as the reference population. For the years when we have only overall mortality data, we assume that the relative mortality of smokers and non-smokers is

time-invariant. We set this ratio equal to the mean relative mortality by cohort and gender derived from the cause-specific data.

In Figure 1 in the main text, we plot the adjusted and unadjusted smoking prevalence rates for the cohorts of Spaniards who were age 60-69, 70-79, and 80 and older in 2007. As explained in Christopoulou et al., across several other countries (and in Spain), the two series do not differ for younger birth cohorts. For each sub-group, we plot the prevalence derived from the retrospective data as a solid line and the prevalence adjusted using the Peto et al. calculation as a dashed line. Solid and dashed lines overlap almost completely for the 60-69 generation of men and for all generations of women (whose smoking prevalence rates never exceed 14 percent).

Figure 2 in the main text presents results from the formal test of whether or not the adjusted and unadjusted prevalence rates statistically differ. We use the standard Pearson χ^2 test of independence for binary variables. In each panel we plot three lines that represent the χ^2 -statistic in each year of life for each generation by country and gender. Values above the 5% level critical value of 3.84 identify the years in which the two rates statistically differ. The tests show that the rates statistically differ only for Spanish men who were 70-79 and 80 and older when surveyed. For women, differences are never statistically signicant. Note that for the oldest cohorts of men, the adjustments for differential mortality yield smoking prevalence rates that stastically differ in years quite close to the survey year.

In Table 1 in the main text we report sample sizes, we present details of our findings, and we highlight some patterns. The difference between unadjusted and adjusted prevalence appears largest for the oldest generation of Spanish men. At the peak prevalence rate for this group, the two rates differ by 8.9 percentage points $(13.7$ percent of unadjusted prevalence). Unadjusted prevalence for this group is signicantly underestimated in all years over 1924- 1997. The next largest difference appears for men who in 2007 were 70-79 years old, but it is sizeably smaller relative to that of the oldest cohort. At the peak smoking prevalence rate of the 70-79 year olds, the difference is 3 percentage points (4.5 percent of unadjusted

prevalence). Unadjusted prevalence for this group is signicantly underestimated in all years over 1944-1995. Notably, correcting for differential mortality affects the pattern of peak smoking-prevalence across birth cohort of men. While unadjusted peak prevalence appears to monotonically decrease with cohort age, adjusted peak prevalence monotonically increases with cohort age. Evidently, by applying an algorithm that captures more data variation relative to that of Bilal et al. we are able to derive new important insights. Specifically, we show that the adjustment alters inferences one draws about inter-generational patterns of smoking, and we can infer the year and age at which unadjusted historical smoking prevalence data stop being reliable for policy and scientific purposes.

4 Conclusion

Contrary to their claims, the analysis reported in Bilal et al. is demonstrably not the first time researchers have validated historical smoking prevalence rate data using contemporaneously measured smoking prevalence data. A long and rich literature validates social science data of all types and smoking prevalence data in particular. While it is useful to validate the Spanish National Health Interview Survey data for the five survey years they use, they employ a method to adjust for differential mortality of smokers and non-smokers that has been superseded by a superior method. Here we set the record straight on their contribution relative to the contribution of previous researchers.

The lapse of these authors is not difficult to explain. It is a perfect example of what happens when academic disciplines who work on similar (or often exactly the same) research questions grow apart, and it highlights the need for inter-disciplinary interactions. The studies that we have cited in this commentary have been written by authors in Epidemiology, Medicine, Public Health, Economics, Statistics, and Demography. These authors publish their work in journals, books, and papers in their specific fields, and follow the work of colleagues who also publish in those fields. Therefore, it may not be surprising that they miss

important contributions from researchers in other sciences. We have written this commentary with the intention to provide a general overview of the literature that validates retrospective data drawing together material from different academic fields.

Our review reveals that researchers have made impressive progress towards validating restrospective smoking data and understanding the various sources of bias. However, more work needs to be done to make these data more robust and, therefore, more useful for substantive policy questions, especially those questions that need more than just descriptions of behavior. For example, ongoing but unpublished work is exploiting the growing number of longitudinal panel studies that collect retrospective smoking data from the same individuals over multiple years. That work exploits the information embedded in multiple observations of the same (potentially) mismeasured data on one individual [\(48\)](#page-16-7). Further, the method described in Bar and Lillard [\(44\)](#page-16-2) for correcting for heaping in retrospective reports can be extended and enriched to control for factors that systematically predict recall errors. The Christopoulou et al. correction for differential mortality bias can also be extended to relax the embedded assumption that, while smokers who survive and do not survive to answer a survey differ in terms of mortality, they do not differ in terms of their smoking behavior (i.e. they have the same rates of smoking initiation and cessation). This assumption likely produces conservative estimates of adjusted smoking prevalence, since non-survivor smokers may be less likely to quit smoking and might also start smoking earlier than survivors. These and other issues are at the forefront of the literature that attempts to validate data on historical smoking behavior generated with retrospectively reported data.

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