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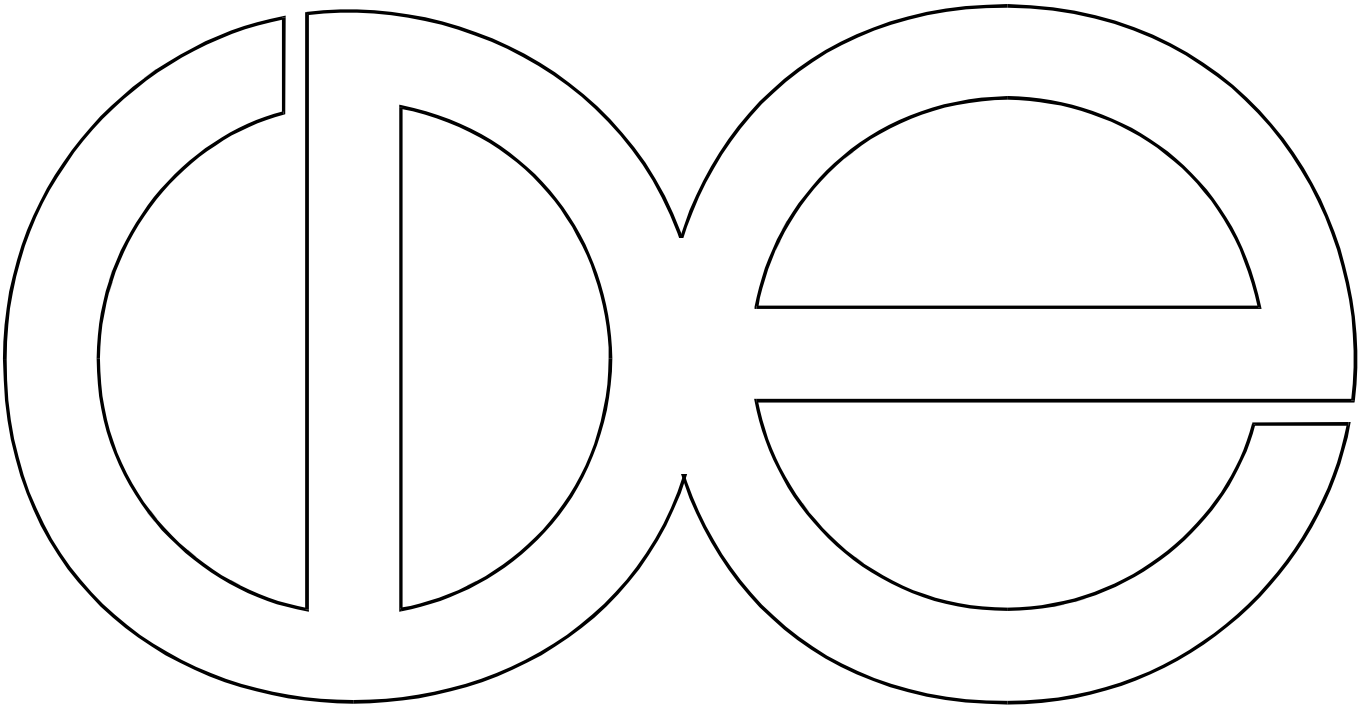

Forecasting Effects of Smoking on Latin American Mortality

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Introduction

In a recent paper (Palloni, Novak and Pinto, 2012) we showed that the tug of past smoking weighs heavily on observed mortality levels and patterns in a handful of countries of the Latin America and Caribbean region (LAC). The effects are felt mostly at ages above 60, precisely the age segment within which future changes in mortality risks could alter future life expectancy more than trivially. We examined the experience of five countries spanning a broad range of manifestations of the smoking epidemic and estimated that smoking attributable mortality is equivalent to losses of life expectancy at age 50 of the order of 4 to 5 years. In this paper we fine-tune the methodology to derive estimates, generalize their application to a set of set of 15 countries from 1980 to 2008, and formulate a flexible procedure to forecast short-to-medium run expected years of life lost attributable to past smoking.

The plan of the paper is as follows: in section I we provide background and summarize what we know about smoking behavior in LAC. In Section II we fine-tune a technique to estimate mortality attributable to smoking and (a) make it suitable to LAC countries with deficient vital statistics and (b) explicitly account for model uncertainty. In Section III we derive flexible procedures to forecast mortality attributable to smoking. In section IV we describe the data. In Section V we apply the estimation procedure and generate forecasts. In Section VI we derive implications, summarize our findings, and conclude.

Background

As a response to the increasing vigilance and massive public health campaigns against tobacco consumption that began in the US after the mid sixties, the tobacco industry initiated an overt program to open new markets in Europe, Asia and Latin America (Bianco et al. 2005). Four socio-demographic factors contributed to the expansion of the market of potential smokers in LAC since the 1950s: the explosive growth in the populations of adolescents and young adults who are highest risk to initiate smoking, the spread of urban life style and the accelerated growth of cities, greater access to education, and the entry of women into the labor market (da Costa e Silva and Koifman 1998). Furthermore, sharp drops in the price of market-ready tobacco products and the unabated assault of a sophisticated publicity machine contributed further to broaden the appeal of cigarettes and transform potential customers into habitual consumers. As a

result of these changes the prevalence of cigarette consumption increased first among forerunners of the mortality decline in the region (Argentina, Uruguay, Cuba, and Chile) and with a lag it spread in countries such as Brazil, Colombia, Mexico, Panama and Venezuela. Laggards in the mortality decline (Peru, Paraguay, Ecuador, Bolivia and the bulk of Central America and the Caribbean) have followed suit as they begin to experience the initial stages of a 'smoking epidemic'[1] (Champagne et al. 2010; Menezes et al. 2009).

a. Preliminary evidence: the footprints of smoking

There is considerable empirical evidence suggesting that at the very least two diseases will become more prevalent among those who smoke: lung cancer and chronic obstructive pulmonary disease (COPD) (Doll et al., 2005; Glei et al. 2011, Streppel et al. 2007, Menezes et al. 2005, Bosetti et al. 2005). This evidence also implicates smoking as a significant causative factor of other types of cancers (Doll et al. 2005) and cardiovascular diseases (CVD) (Chen and Boreham 2002). CVD declines over the last two decades in high and middle-income countries is largely attributable to medical and technological innovations, widespread screening, and massive use of pharmaceuticals all of which offset or postpone the deleterious effects of smoking. Similar, improvements in the screening/treatment of other smoking-related diseases, particularly COPD and lung cancer, have been slow and hard to achieve.

Evidence from mortality rates over age 50 due to smoking-related causes of death (lung cancer, other forms of cancer, COPD, and diseases of the circulatory system) in LAC countries indicates that smoking has already left a scar in some countries and will, in all likelihood, influence similarly the mortality experience of countries where trends in smoking prevalence are on the raise. The issue is not if the impact of smoking will be felt but rather when and how large it will be.

Figures 1a to 1d display male and female mortality rates, adjusted for completeness, over age 50 due to lung cancer as well as to diseases of the circulatory system in selected countries of LAC[2]. Lung cancer rate for males is high and declining in Uruguay followed closely by Cuba and Argentina, where trends are flat or slightly upward. Brazil, Chile Colombia and Venezuela experience lower rates and slightly flat. In contrast, female mortality rates due to lung cancer are

[1] We use the term 'smoking epidemic' as is used in the standard literature on the subject, as a shortcut rather than to refer to a process that can be legitimately thought of as an epidemic.

[2] We include five countries for which we have abundant information on smoking prevalence (Argentina, Brazil, Chile, Mexico and Uruguay) and two countries located at the extremes of the smoking epidemic, Cuba and Colombia.

lower than those for male rates but increasing over time. Cuba has the highest rate followed by all the remaining countries, which also show an increasing trend. In opposite direction, mortality rates due to circulatory diseases are on the decline everywhere[3]. As shown below, the ranking order of countries by these causes of deaths is consistent with the stage they are experiencing in the smoking epidemic.

b. The diffusion of smoking

What should we expect future mortality patterns to look like? How harsh could the impact of smoking be and how heterogeneous across countries? Section IV addresses this issue directly by generating empirical forecasts into 2020. To put forecasts in context as well as to provide guidance to judge their accuracy, we characterize the LAC smoking epidemic in comparative perspective.

Lopez et al. (1994) proposed a stylized representation to describe the stages that the tobacco epidemic typically undergoes in developed countries. This model identifies four stages or phases. The initial phase is one where smoking prevalence is low for both men and women. In the second stage smoking among men begins a steep increase while among females smoking uptake lags behind by one or two decades but catches up rather rapidly. In stage three, male smoking prevalence initiates a decline at all ages and the drop is particularly marked among younger cohorts. Women smoking prevalence peaks throughout this stage whereas in the last stage it begins to decline as sharply as it did among males in the preceding phase. Gender differentials are an important feature of this process but so are social class differentials: during the initial stages male and female smoking uptake occurs first and more rapidly among members of the upper classes. As tobacco prices drop, facilitating adoption among lower classes and as the impact of public health campaigns erodes the behavior among higher classes, the social class gradient changes direction and reverses much as the gender gap does as one stage supersedes the previous one.

The health damage induced by smoking requires some time to fully manifest itself. Under average smoking intensity and distribution of ages of initiation, its mortality impact takes at least 20 to 30 years to become detectable. As a result, during the first and second stages there are no significant impacts on mortality. Smoking attributable mortality begins to surge after the onset of

[3] The reduction in mortality due to circulatory diseases is due mostly to the attenuation of risk factors that more than compensate for the reinforcement of risks associated with smoking.

the third stage and throughout the fourth stage before dipping down sharply. The bulk of smoking attributable mortality is associated with lung cancer, COPD, cancers of selected sites, and some forms of circulatory and heart disease (in order of importance) and should attain a peak 20 to 30 years after the onset of[4].

Table 1 identifies and describes the main traits of the stages proposed by Lopez et al. (1994) and Ezzati and Lopez (2004). Table 2a classifies LAC countries according to the stage of the tobacco epidemic in which they can be situated (see Table 2b). While the US and other developed countries are exiting stage four (Edwards 2004), most countries in LAC are in stage two and some are sufficiently advanced and are classified in stages three or four. This is the case of Argentina, Uruguay, Chile and Cuba where tobacco consumption is very high in capital cities and with steady increases of smoking prevalence in the periphery (Champagne et al 2010). Other countries such as Brazil, Colombian, Mexico, Venezuela and Panama follow closely behind whereas the remaining countries are navigating through the first two stages.

c. *Cross sectional prevalence of smoking*

Recently collected information on individual smoking behavior enables us to approximately size the magnitude and diversity of the smoking epidemic in LAC. Figures 2a to 2c display estimates of smoking prevalence by age and gender and Figures 3a to 3c show prevalence by coarse age groups and levels of education in five LAC countries with requisite data[5]. First, these figures suggest that smoking behavior must have already left an imprint in these countries, one that that will be augmented in the future as younger cohorts of smokers make their way into older ages. At the very least two conditions will become more prevalent among those exposed to smoking: lung cancer and COPD (Glei et al. 2010; Streppel et al. 2007; Menezes et al. 2005; Bosetti et al. 2005) and possibly other types of cancers (Doll et al. 2005). Second, age, gender and education patters are roughly consistent with the stage-based model and classification of countries introduced before (see Tables 1, 2a and 2b).

Assessing past and future impact of smoking on mortality would be rather simple if we were in possession of not just of cross sectional smoking prevalence as shown above but of more detailed longitudinal information about smoking behavior. In the absence of such information,

[4] The foregoing scheme or ideal type of stages in the smoking epidemic has been recently improved to broaden its applicability and to increase its descriptive power. Ezzati and Lopez (2004) added a fifth stage to the conventional four-stage model to fine tune the description of the last stage. They propose a distinction between an early and late period of decline each following the early, rising and maturity periods included in the original scheme.

[5] A more detailed examination of these data is in Palloni et al. 2013

we will employ approximations to evaluate the magnitude of the impact of smoking on LAC adult mortality in the most recent past and in the near future.

Methods of estimation

In the absence of suitable longitudinal information about smoking behavior, health, and mortality, we resort to indirect methods to estimate the fraction of adult deaths attributable to smoking. We do this with different versions of a procedure well-suited in countries with good vital statistics but much less robust when vital statistics and population census counts are deficient. We first discuss the data set, formulate the core of the estimation procedure and, finally, define the variants we apply to LAC countries.

a. Methods I: the core procedure

We adopt a modified technique suited in contexts where vital statistics are defective and cause of deaths information less reliable than in high-income countries. Modifications are introduced to resolve two problems. The first has to do with the class of regression models we estimate in a pooled cross-section and time series dataset. The second is to take seriously the distinction between a fine-tuned categorization of causes of death and an alternative one that lumps all causes of deaths, other than lung cancer, into a single category

We start from first principles (Preston et al., 2011) and pose a relation between the lung cancer mortality rate in the *total population* and among nonsmokers[6]

$$M_L = \lambda^N_L (1+\theta_L) \qquad (1)$$

where M_L is the observed lung cancer mortality rate in *the population* and λ^N_L is the mortality rate among non-smokers. In this expression θ_L is a measure of the excess mortality risk (EMR) associated with smoking. It is NOT the lung cancer relative risk of smoking. The relative risk is given by

$$\rho_L = \lambda^S_L/\lambda^N_L \qquad (2)$$

[6] To avoid confusions, and whenever possible, we use the same notation utilized by Preston and colleagues (2011). Furthermore, to avoid cluttering we ignore subscripts for age, time and gender. But, unless noted, all expressions are age-time-gender specific.

where the term in the numerator is the lung cancer mortality rate among smokers. From the canonical definition of relative and population attributable risk we obtain

$$\theta_L = s*(\rho_L - 1) \qquad (3)$$

where s is the fraction in the population who smoke. Thus, whereas ρ_L is a pure measure of the impact of smoking on lung cancer mortality, θ_L is an indirect measure of this impact that *depends on the population prevalence of smoking*. The indirect measure can increase or decrease as a function of smoking prevalence even if the relative risk remains invariant. Later in the paper we exploit this relation to develop a consistency test. A longitudinal survey such as CPS-II provides high quality information on both quantities, but under ordinary circumstances we only observe M_L. If, in addition, we secure an estimate of λ^N_L we can compute an estimate of θ_L.

At this point we could formulate a model analogous to (1) for any arbitrary cause of death

$$M_J = \Gamma_J*(1+\eta_J) \qquad (4)$$

where M_J is the observed mortality rate due to cause of death J (also associated with smoking), Γ_J is the hypothetical (unknown) mortality rate due to cause J among nonsmokers, and η_L is the J cause of death-specific EMR associated with smoking. This leads to a tractable expression *only* under some additional assumptions. However, one can turn to a proportional hazard functional form to arrive at a more practical expression:

$$M_J = \Gamma_J*\exp(\beta_J*\theta_L) \qquad (5)$$

where M_J , Γ_J , and θ_L are as before and $\beta_J >= 0$ is a multiplying factor that expresses by how much more (how much less) smoking produces excess mortality due to cause J relative to lung cancer mortality[7]. Note that, here again, β_J,is a multiplier of θ_L and not a relative risk: it is the excess mortality risk due to cause J among those who smoke relative to the excess of lung cancer. Replacing (1) in (4) leads to

[7] It is straightforward to show that if $\eta_J = \beta_J*\theta_L$ is small enough, (4) and (5) are equivalent. The "small enough" is the problem.

$$M_J = \Gamma_J * \exp(\beta_{J*}(M_L/\lambda^N_L - 1)) \qquad (6)$$

We normally observe (frequently with error) the quantities M_J and M_L but none of the other quantities.[8] Assume there is a standard population from which we retrieve λ^N_L as well as estimates of M_J and Γ_J, M^*_J and Γ^*_J respectively. In this standard population expression (6) also holds. If one assumes the ratio of total mortality due to J in the observed to the standard population, M_J/M^*_J, to be the same as the ratio among nonsmokers of the death rate due to cause J in the observed to the standard population, Γ_J/Γ^*_J, then we could easily solve for β_J in (6). Alternatively one can leave the baseline mortality function for cause J as a free function and estimate it from a pooled cross-section time series data in which enough additive and interaction terms are entered to consistently estimate both Γ_J and β_J. This is the strategy followed by Preston and colleagues. Although to estimate Γ_J and β_J there is no need to assume that λ^N_L is known, this quantity is required to compute total smoking attributable lung cancer mortality from estimates of β_J and Γ_J . The smoking attributable rate associated with lung cancer is $\Delta_L = (M_L - \lambda^N_L)$ and the smoking attributable rate associated with cause J is $\Delta_J = (M_J - \Gamma_J)$.

b. Methods II: variants of the core procedure

Before applying it to LAC data, the core method requires fine-tuning to circumvent difficulties absent or unimportant in contexts with high quality vital statistics.

i. Completeness of death registration and population age misstatement

Relative completeness of deaths registration is problematic even if it does not vary by causes of death. Not only will the estimated coefficient of M_L be biased but all parameters involving interaction terms with M_L will be biased as well. When completeness is imperfect but invariant across causes of deaths the biases will be of moderate magnitude as they affect both the dependent and the independent variable in equal measure. To resolve this problem we use a data base that is adjusted for relative completeness and age misstatement among the older population. But even if adjusted for completeness residual errors in the data may remain. In particular if

[8] Lung cancer mortality rates among smokers are usually obtained from CPS-II and usually employed as benchmarks. Of course this can lead to errors since the fraction of all deaths due to lung cancer among smokers depends on many factors that could be very different in populations other than that in CPS-II. However, in this paper (as well as in Preston et al.) departures from the assumption of identical values of λ^N_L in all population considered leads to only small errors).

completeness varies across causes of deaths we will produce underestimates of the effects (and standard errors) of smoking due to mortality of lung cancer.

ii. Defective distribution of causes of deaths

The derivation above assumes that classification of causes of deaths is error-free. This is unlikely to be the case anywhere but particularly unlikely in low to middle-income countries. The most important problem is the fraction of death with an ill-defined cause and the changing propensity of some causes of deaths to end up in this category.

The solution to this problem is remarkably simple. Suppose that the true level of mortality due to lung cancer is $M_{Lo}(1+\sigma_L M_{Io}/ M_{Lo})$ and the true mortality due to cause J is $M_{Jo}(1+\sigma_J M_{Io}/ M_{Jo})$, where M_{Lo} and M_{Jo} are the observed mortality rates due to lung cancer and cause J respectively, M_{Io} is the mortality rate due to ill-defined conditions, and σ_L and σ_J are the fraction of ill- defined deaths that correspond to lung cancer and cause J respectively. It can be shown that, under some simplifying but not overly restrictive assumptions, the appropriate regression model is (6) but with two extra additive terms and associated parameters: $\sigma_L(M_{Io}/ M_{Lo})$ and $\sigma_J (M_{Io}/ M_{Jo})$. The expression can be made even more general if one deems appropriate interaction terms to represent possibly changing values (over time, by gender or across units of observation) of the parameters σ_L and σ_J.

iii. Model specification

Equation (6) can be estimated consistently only if all the covariates on which Γ_J depends are included. In particular, there should be enough observations and time specific variables to capture sample variability in Γ_J. Furthermore, since the imprints of smoking on mortality by cause of death vary by age, time periods, and countries we should include two and three way interaction terms to secure a precise specification.

Taking logs on both sides of equation (5) leads to:

$$\ln M_J = \ln\Gamma_J + (\beta_J / \lambda^N_L)*M_L - \beta_J \qquad (7)$$

Since this is the logarithm of a rate (or of a count variable per exposure) the assumption of normality is inappropriate. Nor is the assumption of a Poisson distributed count pertinent as there is almost certainly overdispersion. For this reason, Preston and colleagues suggest to use a negative binomial distribution.

Regrettably, identification of the correct functional form is not the only difficulty related to the definition of the model. Here we depart from the formulation by Preston et al. Robust estimation of parameters from a cross section and pooled time series conventionally requires to pose and test for autocorrelation processes to represent the behavior of the errors over time. To account for this and for suboptimal estimation of standard errors, we estimate alternative ARIMA models for the error terms. More importantly, however, if there are omitted variables that do not change over time and are correlated with one or more of the variables included in the model, the estimates of the target quantities will be inconsistent. In order to test for this possibility we estimate average, fixed and random effects models and test for equality of coefficients in the last two using Hausman's test. In virtually all cases the fixed effects model yields estimates for varying covariates that are indistinguishable from those of the random effects model but the latter produce somewhat different estimates than the population average model. We retain both sets to compute a range of estimates of smoking attributable mortality[9].

iv. Grouping causes of deaths

While distinguishing mortality due to lung cancer from mortality due to other diseases is an easy way to estimate parameters, the distinction is too coarse and could be inappropriate. There is reason to believe that smoking is more likely to leave marks on some causes of deaths more than in others: diseases such as cancers and circulatory diseases are likely to be differentially associated with smoking in the sense that cumulative health effects translate into different relative risks. Smoking is known to be related to bladder and pancreas cancers as well as to some forms of circulatory and heart disease but less so to other chronic conditions. If so, each of these conditions is associated with a distinct parameter β_J. Neglecting this type of heterogeneity leads to biased estimates of smoking-attributable mortality. The direction of the bias is difficult to ascertain *a priori* and its magnitude depends on the empirical distribution of causes of death and on the relative impact of smoking on each group of causes.

To address this issue we use two alternative estimation strategies. The first is one where we only distinguish lung cancer and all other diseases as do Preston and colleagues. The second distinguishes lung cancer, cancers of other sites, circulatory diseases and all other diseases. In the first case there is only one equation (7) to estimate whereas in the second we estimate three

[9] We also estimated population average models with plausible autocorrelation schemes. But these do not yield different estimates of regression coefficients (and, hence of smoking-attributable mortality), only different standard errors

equations, one for each group of causes of deaths, and then must aggregate results across them to compute total smoking attributable mortality.

In summary, a final model accounting for ill-defined causes of deaths for five year age groups from 50 to 85+ over the period 1980-2007 is

$$\ln M_J = \Sigma_i \varphi_{iJ} Z_i + \gamma_J M_L + \Sigma_i \alpha_{iJ} * I_I + \sigma_J * M_{Ill}/M_J + \sigma_L M_{Ill}/M_L + \varepsilon_J \qquad (8)$$

where Z_{iJ} is a set of dummy variables that include age, country and calendar time, the I_i's are first order interaction terms between M_L and age, country and calendar time and ε_J is an error term. There are two extra terms involving the mortality rates due to ill-defined causes (M_{ill}) to control for differential propensity for lung cancer and cause J to be explicitly classified as such.[10]. We generate three sets of estimates: two from fixed and random effects models and one from a population average model. Finally, to simplify estimation we set the origin to January 1980.

For each set of parameter estimates we calculate two sets of predicted values of M_J: one corresponding to observed values of M_L and the other corresponding to a counterfactual scenario where the rates of lung cancer equal those that would be observed if the smoking prevalence were 0, namely, λ^N_L. The difference between these two predicted values, $\Delta_L = M_J - \Gamma_J$, is an estimate of the smoking attributable mortality associated with cause J. This quantity added to the difference $\Delta_L = M_L - \lambda^N_L$ yields an estimate of the total smoking attributable mortality.

The combination of two alternative model specification (average and random effects) and two strategies to handle causes of deaths, two versus four groups, leads to four sets of alternative estimates of smoking-related attributable mortality. The variance of these estimates is a measure of model uncertainty that must be explicitly considered.

Forecasting smoking-attributable mortality

Is the information generated thus far is sufficient to produce short term forecasts of smoking related mortality. The answer would be simple if we knew the composition of current cohorts by

[10] In a preceding paper (Palloni et al., 2012) we found only small differences between alternative ways of handling the grouping of causes of deaths. But that exercise was carried out in only four of 20 possible countries. In the larger sample of countries the two alternative groupings of causes produce different results.

age and smoking status or, better yet, their history and appropriate relative risks. What is available to us is much less than this. Below we suggest four different procedures none of which requires information on smoking histories.

a. Single-step forecasting model

The simplest forecasting model is one that focuses directly on the main quantity of interest, namely, the fraction of deaths (or death rates) attributable to smoking. We estimate alternative ARMA models for the trends in smoking-attributable deaths choose the most parsimonious among the best fitting ones. This type of model does not require assumptions beyond those inherent in the identification of a time trend. The ARMA models that fit better are different across countries but they all share the same properties: the best fitting model is one with a moving average parameter (p) equal to 1 or 2 and an autocorrelation parameter (q) equal to 0 or 1. Other models overfit the data in all cases.

While simplicity is appealing, it is also deceiving. The quantity we forecast, the fraction of smoking-attributable mortality, is the product of two quantities: the death rates due to lung cancer, M_L, and the excess relative risk (EMR) retrieved from equation 8. In turns this is a function of the regression coefficients and interaction terms associated with M_L. A more powerful, though a more complex, strategy is to forecast separately each of these components in two steps.

b. Two-step forecasting models

The next three forecasting models proceed in two stages. In the first we use full information to forecast country-specific trends in lung cancer death rates. In the second we combine the lung cancer death rates forecasts with forecasts of the parameters in (8) that translate lung cancer mortality into smoking-related mortality due to other causes of deaths. This is easy to do since model 8 contains, by definition (interaction term between year and M_L) the influence of time on the effects of M_L on M_J .The combination of these two pieces of information is enough to compute forecasts of smoking related attributable mortality. Three assumptions must be made: (a) trends in lung cancer deaths among nonsmokers remain the same; (b) mortality rates due to ill-defined causes of death and their effects remain constant throughout the forecasting period; (c) past trends in *changes over time in ERM (as reflected in the regression coefficients of the additive and interaction terms in model)* remain invariant. The most influential; of these assumptions is (c).

The three forecasting methods differ in the strategy to forecast lung cancer death rates. We briefly describe each of these in order of complexity.

i. ARIMA models M_L

The simplest solution is to identify the most parsimonious among the best fitting ARMA model for the detrended series of lung cancer death rates. It is well known that multiple ARMA model may fit the data well but they do (or can have) different implications for forecasts. We fit alternative ARMA models in each country and chose the two best fitting ones and compute two alternative forecasts of lung cancer death rates. We then combine these with forecasts of the estimated regression coefficients in model 8 and the implied ERM.

The drawback of this strategy is that the ARMA models for fitting the data are atheoretical and do not express any interpretable representation of the processes that generates the lung cancer death rates.

ii. Lee-Carter models for M_L

The standard formulation for the Lee-Carter model is as follows

$$\ln M_L(x,t) = A(x) + B(x) * K(t) + \varepsilon(x,t) \qquad (9)$$

where $M_L(x)$ are observed lung cancer mortality rates, A(x) is an arbitrary scaling age-specific mortality schedule (the "level" or general age pattern or profile), B(x) is a standard schedule of age-specific *changes* in $M_L(x)$ over time, and K(t)is the trend in the magnitude of mortality changes. Model 9 is underidentified and restrictions need to be imposed. The most commonly adopted is to set A(x) to equal the average of ln $M_L(x)$ observed over the time period examined[11] . What we propose here is to specialize (9) to lung cancer death rates: we let A_x be the average pattern of the log of lung cancer death rates between 1980 and 2007 or so, B(x) is the average pattern of age-specific changes over time and K(t), the time dependent magnitude of those changes. To identify the unknown quantities in 9 we need to use Singular Value Decomposition (SVD) to minimize squared deviations once the effect of the average level (A(x)) has been accounted for. Under regular conditions SVD yields a unique solution. Finally, we forecast $M_L(x)$ into the future using simple standard ARIMA models.

iii. A Brass logit model for M_L

[11] Or, equivalently, that the sum of the B(x) values over x is 1 and the sum of K(t) values over t is 0

We start with the assumption that age patterns of mortality rates due to lung cancer can be represented as a function of a standard age pattern of lung cancer mortality rates. A very general expression (specific for country and gender) is

$$\varphi(M_L(x,t))=\alpha(t) + \beta(t) *\varphi(M_L^s(x)) + \varepsilon(x,t) \qquad (10)$$

where $M_{L(}x,t)$ and $M^s_L(x)$ are, respectively, the observed lung cancer mortality rate at age x and calendar year t and the standard lung cancer mortality rate at age x[12]. Although this is a two-parameter formulation, we allow each parameter to change over time thus introducing extra-parameterization. If we choose a logit transform the constant in (10) will capture the level of mortality due to lung cancer whereas slope parameter will operate to tilt the age pattern: when it increases above 1 it will elevate mortality rates at older ages relative to mortality rates at younger ages. When the value of the parameter descends to zero from 1mortality rates at younger ages will increase relative mortality rates at older ages to older ages. This representation is remarkably convenient because mortality rates due to lung cancer must respond differently by age and with different lags to the timing of the smoking epidemic: the age pattern of mortality rates due to lung cancer will experience a first impact at older ages when the cohorts who first take up smoking feel the brunt of their cumulated exposure to smoking. But then it should gradually tilt around itself when the younger cohorts who abandon smoking shed excess mortality risks as they reach older ages. Allowing both the constant and the slope to vary over time provides a very flexible representation:

$$\varphi(M_L(x,t))= [\alpha_o + \beta_o * \varphi(M^s_L(x))] + \mathbf{\Sigma_{j=1,k}}\, \alpha_j(t) + \Sigma_{j=1,k}\, \beta_j(t)*\varphi(M^s_L(x))+ \varepsilon(x,t) \qquad (11)$$

where $\varphi(.)$ is the logit transform of the rates[13] . The terms in squared parentheses represent the canonical formulation of a logit model whereas the remaining terms identify the effects of time on both the constant and the slope of the logit model.

The usefulness of Model 11 depends on the choice of standard and on proper identification of time trend of $\alpha(t)$ and $\beta(t)$. To simplify calculations, and also because it is a less

[12] Instead of rates one can also use the survival function of the associated single decrement table.
[13] logit y=ln(y/(1-y))

error-prone choice, we choose country-specific standards defined as the average schedule of lung cancer death rates over the period considered. To identify the time trends in the logit model parameters we adopt a "non-parametric" [14] procedure that assigns differential weight to more recent and more distant past values of the time series for the production of a forecast. In particular we choose to use the Holt-Winters (HW) smoother with its, albeit limited, forecasting capabilities (Winters, 1960). This is a class of exponentially weighted moving average smoother that computes fitted values of a time series, v(t), as an exponentially weighted average of estimates of the past mean and past trend of the series. As all exponentially weighted moving averages, the HW estimator requires selection of an arbitrary weight and in the HW estimator we need to do so both for the term reflecting the past trend and the term representing the past mean. A Brass logit model with two parameters, $\alpha(t)$ and $\beta(t)$, requires that we choose four different weights, a pair for each parameter. To make computations feasible we chose pairs of weights for $\alpha(t)$ and $\beta(t)$ from within the range .1 and .9 in steps of .2 and then chose the subset of fitted trends that best represent the observed trends with an R-square metric. In the end we chose 9 different HW estimates of trends for α and 9 for β yielding a total of 81 possible different trends in lung cancer death rates[15]. As we show below, all models we so choose fit the *observed* trends well but they do have different implications for the future since different combinations of weights in the Holt-Winters computations weight differently the recent and more distant past. This flexibility is crucial when modeling trends in lung cancer death, a phenomenon that reflect waves of disease prevalence created as different cohorts pass through ages at higher risk as they enter and exit the smoking epidemic.

iv. Forecasting strategies: similarities and contrasts

While the first (ARMA) strategy presupposes only minimal theorization about the quantity being modeled (lung cancer death rates), there are striking similarities between the premises and implications of strategies (2) and (3). To begin with in both cases one starts with the assumption that there is an age-pattern for death rates due to lung cancer: in the Lee-Carter model this is contained in the vector of values Ax. In the case of the Brass system the age pattern is the standard. In both cases estimation is possible only if these are fixed *ex ante*. The second

[14] Non-parametric is perhaps a misnomer as the HH estimator depends on arbitrary weights. But its values do not depend on a model for the trend itself.

[15] This calculation assumes that the two parameters are independent of each other. A more complex but highly taxing strategy is to estimate and forecast the matrix of variance covariance of the logit model parameters and use it to constrain the number of forecasts.

commonality is that both the Lee-Carter and the Brass system assume an age pattern of mortality changes. The main difference is that whereas the age pattern of changes in the Lee-Carte model is fixed over time, the one allowed in the logit model can vary over time with changes in the parameter $\beta(t)$. In contrast to the Lee-Carter model, these changes are not age-free and are constrained to pivoting the standard around a fixed age (e.g. to changes of mortality at the upper ages relative to mortality at younger ages). Finally, in both models there is a parameter that controls the levels (of the trend of mortality or of age-specific changes): in the logit model this is the role of α (t) whereas in the Lee-Carter model the level of age-specific changes are captured by K_t.

Modeling lung cancer death rates time trends with ARIMA models constrains us to represent only the direct dependency of age specific rates over time, e.g. via lagged values. But since the number of lagged terms we can use is limited, the relational part of lung cancer mortality rates that reflects an age-cohort smoking pattern cannot be properly represented. As a consequence the forecasts, even over a short horizon of a few years, could be off target and differentially so by age. The Lee-Carter model improves this by allowing both an age pattern of mortality and a pattern of changes across ages. While this is an improvement it contains a flaw that limits its applicability to lung cancer mortality: the pattern of changes by age is fixed over time. This is a shortcoming for modeling mortality due to lung cancer because the pattern of changes by age shifts over time reflecting the entrance/exit of cohorts with different smoking histories. This is partially improved in the logit model where the age pattern of changes by age is allowed to vary over time, albeit in a limited form and with extra parameters[16].

From this reasoning we anticipate that the logit model will perform better than the Lee-Carter model and either will perform better than simple ARMA models.[17]

Data

The quality of vital statistics and population census counts in LAC has varied over time from poor to mediocre to good. Completeness of death registration and population counts,

[16] Appendix 3 contains an ad-hoc description of each model's properties, their similarities and differences.

[17] Di Cesare and Murphy (2009) were the first to identify this shortcoming of the Lee-Carter model as a representation of time trends of some cause-specific causes of deaths. The authors suggest the use of age-period-cohort models as optimum tool. However, APC models require a much larger number of parameters and highly precise age-specific death rates

inaccuracies in the classification of death by cause, and age overstatement at adult ages are likely to be more severe than in high-income countries. To minimize the impact of errors of coverage and age misstatement we use a new database for Latin American mortality created at the Center for Demography and Ecology over the last ten years (LAMBdA). This database contains national life tables (2 for every decade) from 1850 to 2010 for 18 countries of the LAC region. They were obtained combining vital statistics and censuses for the period 1930-2010 with uniform and standardized adjustment (indirect) procedures to correct for completeness and age overstatement. A summary of methods used to adjust the data, comparison with alternative estimates and a preview of their usefulness appears elsewhere (Palloni and Pinto, 2004; Palloni and Pinto 2011).

Data on causes of deaths starting in 1945 were obtained from WHO databases and modified to conform to corrected total mortality. These data have a shortcoming in that we cannot correct for misclassification of causes of death, a piece of information that is badly needed by the method described below. Information on causes of deaths is notoriously sensitive to classification schemes, delays in adopting standard classificatory principles, medical practices and definitional idiosyncrasies. Some of these problems can be overcome with suitable groupings and we do this below. But the most important difficulty, and the one that is more relevant in some countries of LAC, is the magnitude and changes over time of deaths assigned to "ill-defined" group. In lieu of an allocation of these deaths via convenient but *ad hoc* (and *ex ante* defined) procedures we use the model proposed below to adjust for biases induced by the variable magnitude of death rates due to ill-defined categories. This is a more appropriate correction as it is derived internally from the model itself rather than imposed exogenously.

More details about the database spanning the period 1980-2007 is described in Appendix I and II. Because mortality associated with smoking is very low below age 50 we confine our analyses to five-year age groups between 50 and 85+.

Estimation and forecasts

We first discuss results of smoking-attributable mortality and examine counterfactual life expectancies at age 50 from our models. We show that model uncertainty is important and that a great deal of it is due to alternative groupings of causes of death. Despite this uncertainty, male trends of years lost due to smoking are uniformly high and growing among forerunners of the

smoking epidemic and lower but growing in all other countries. With the exception of a handful of countries, females losses are considerably smaller than among males but trends are turning upwards even in those countries where we suspect there is low levels of female smoking.

In this section we also discuss results from all four strategies to forecast smoking attributable mortality and end the section with a couple of consistency tests

a. Alternative estimates of smoking-attributable mortality

First off, we address the issue of model uncertainty. Altogether for each country and gender we estimate four alternative models: two different model specifications (random and average) and, in each case, two groupings of causes of death (one versus three residual groups). Figure 4 displays the mean values of the (uncertainty) range for Argentina (males). This figure alone encapsulates three important features we find replicated in all countries and both genders: (a) heterogeneity of model-specific estimates is moderate to high: it can lead to differences representing up to 30% of the mean value across models; (b) the variation of estimates induced by different grouping of causes of death is larger than that induced by different model specifications; and, (c) there is an interaction effect as the variability associated with model specification is larger when using a more detailed classification of causes of deaths (grouping #1). The latter feature ought to be expected since in this case we must estimate three (not just one) models. The importance of this finding is obvious: increased estimation precision demands careful consideration of the grouping of causes of death other than lung cancer.

Tables 3a and 3b contain the mean, lower and upper bounds of estimates of proportionate excess mortality attributable to smoking for males and females respectively[18]. These figures are years of life lost at age 50 (relative to those observed) and are calculated for the period 2000-2005. Figures 5a and 5b displays the mean values by selected countries for males and females respectively. In the absence of smoking, Argentinian males in 2005 would have experienced a life expectancy over age 50 about 10% higher than what they did. This is the expected value several across models: the bounds of uncertainty place the figure between 7% and 13%. First, note that there are a handful of countries where smoking attributable morality is quite high: Cuba, Argentina, Uruguay and Chile followed closely by Colombia, Panama, and Venezuela. In virtually all countries the trend is upward, even in cases where smoking prevalence is so low that

[18] In each case the mean, lower and upper bounds are from four different models fitted to the data (see text for explanation)

estimates of means of bounds of uncertainty are near zero or within negative territory[19]. Everywhere females lose a smaller number of years of life, reflecting a lesser load of past smoking. Yet, in countries where male smoking is highest, there is an upward progression of years of life lost among females as well. This is particularly the case in Chile and Cuba but also in Argentina and Uruguay, the four countries considered to be the forerunners in the smoking epidemic.

b. Alternative forecasts of smoking attributable mortality

Previously we introduced two major classes of forecasts: the one-step and the two-step. Although each of these classes is legitimate and plausible, the first one does not require any theory to be deployed and it is just an exercise in "best fitting". The second class of strategies contains alternatives that require *ex ante* representation of at least age-patterns of lung cancer mortality.

i. Modeling directly the quantity of interest

The simplest forecasting model is one designed to represent the 'estimated' smoking attributable mortality using all possible model variants described before. There will be four possible time trends, one associated with each model/grouping of causes of death. Although there are multiple ARMA models that could fit these trends equally well we choose the most parsimonious among those that fit best[20]. To simplify calculations we fit time trends to the mean of the range and, independently, to the lower and upper bounds. Figure 6 displays results for the case of Argentina (males). The figure clearly shows that uncertainty over future trends has more to do with the treatment of groupings of causes of deaths (and less so with model specification) than it does with the actual time series fitted to the data. In fact the differences between lower and upper bounds remain fairly constant across time, at least in the case of Argentina[21]. In all cases there is one inference that can be made, namely, that foregone years of life expectancy at age 50 are expected to increase slowly and stop growing altogether after attaining a level between 6 and 10 % (or between 2 to 3 years of current life expectancy at age 50) in the next five to ten years.

The remaining three forecasting strategies require two steps: one requires forecasting lung cancer death rates and the others involve forecasting the effects of lung cancer death rates

[19] Negative estimates are a result of imprecision and very low values of observed lung cancer death rates

[20] We used a combination of BIC and Akaike criterion to choose the most appropriate models. In all cases both of these lead to the same choice

[21] Other countries (and females) show similar patterns. The main differences across countries are the magnitude of model uncertainty due to treatment of causes of deaths and the time trends in that uncertainty. Differences between lower and upper bounds are smaller and steadier in countries with more accurate vital statistics

on other causes over time. The second step is easy as we do have estimates of interaction of estimated EMR effects with time. The first step requires additional modeling decisions. We discuss below the suitability of the Lee-Carter and the Brass logit system approach to perform this step.

ii. The Lee-Carter and Brass logit model in the two-step approach

Figure 7 (and Figure 7a and 7d) summarizes the contrasts between these two strategies using as examples four age groups and Chilean males[22]. The figures display observed and fitted values from Lee Carter and Brass logit model. While the Lee-Carter model reproduces fairly accurately time trends in lung cancer for some ages, it cannot do this for all ages it simultaneously. Thus for example it captures nicely the time trends for the youngest age group but misses completely the trajectory of the oldest group. By contrast the logit model follows in lock-step the time trend and it does not smooth over irregularities at all. Some of these irregularities are surely noise produced but death registration delays, variable completeness over time and oscillations in classifications. and are better eliminated or smoothed out.

The logit model is better behaved but only at the expense of estimating extra-parameters which then we need to forecast to produce values of lung cancer death rates and associated smoking attributable mortality. By contrast, the Lee-Carter model only requires forecasts of the function K(t).

The behavior of the Lee-Carter model is not unexpected. It was confirmed with totally different and independent data sets (Di Cesare and Murphy, 2009) and was anticipated more formally in Appendix 3. Because its results are considerably off-target for some age groups, we exclude it from consideration in the forecast exercise that follows and instead we only employ the Brass logit model.

c. Review of alternative forecasts

The Brass logit model depends on the values of two parameters and a standard age pattern of lung cancer death rates. In each country, and separately for each gender, we estimate a range of non-parametric Holt-Winters $\alpha(t)$ and $\beta(t)$ estimates fitting time trends before 2005 and then compute associated short-run forecasts for each. We then combine these with the standard age

[22] Analogous results and inferences apply to all other countries and to females as well.

pattern to produce forecasts of lung cancer death rates between 2005 and 2020 approximately[23]. In the second step these are combined with forecasts of the EMR from equation 9 to produce forecasts of smoking-attributable mortality. Tables 4a and 4b display the results for males and females respectively. Figure 8a and 8b show results of the fitting and forecast for Cuba males and females respectively.

In all country/gender pairs we obtain estimates from four combinations of model-specification /cause of death groupings. In each case we estimate fitted values using 81 different specifications of the Holt-Winters estimator and compute associated forecasts. This produces a total of 324 alternative forecasts. To summarize this information we compute the mean and upper and lower bounds. In addition to these, we estimate the two the best fitting/most parsimonious ARMA models for each combination of model/grouping. The forecasts from these plus the means, upper bounds and lower bounds are displayed in figures 8a and 8b. We show the results for Cuba since they are undoubtedly the most dramatic of all: the losses of life expectancy at age 50 are expected to growth continuously at least until 2018 when they will amount to between 22% and 32% of life expectancy at age 50. This is equivalent to the loss of a whooping 6.8 years of life from age 50 on up. The singular, most striking feature in this Figure holds in all countries and both genders alike: this is that model uncertainty surrounding the expected losses is quite small relative to the magnitude of the losses. Irrespective of the nature of the model, losses of life expectancy will continue to mount everywhere among males and females and, with a handful of exceptions, the magnitude of these losses will be at a minimum 3-4% and could attain values as high as 30% .

Estimates and forecasts for females are more fragile than for males: the small values of lung cancer death rates reflecting earlier stages of the epidemic, translate into forecasts that even if not containing more model uncertainty, are contaminated by noise which reflects in very small and even negative values in some places. However, trends are as expected everywhere else, particularly in countries that have led LAC tobacco consumption for longer than three decades.

d. Validity tests

How can we gauge the validity of these estimates (and associated forecasts)? In a previous paper (Palloni et al., 2012) we formulated a procedure that could be applied only in countries with

[23] The latest data for which we have information is 2010 but these dates varies by country Since we only focus on forecasts at most ten years after the most recent observation, the final date of forecasts is in all but one case earlier than 2020. To display uniform information for all countries we only show forecasts up to 2018

information on smoking prevalence by age at least once during the recent past. In essence we combined our estimates of EMR with data on age-specific smoking prevalence and computed an estimate of lung cancer (and other diseases) relative risk among smokers (see expressions 1-4). The results we obtain were satisfying since the estimates thus obtained were close to those computed from the CPS-II, the only longitudinal study of smokers where the quantity can be calculated directly.

What about the forecasts? How consistent are they with established forecasts of mortality? The Population Division of the United Nations has been producing mortality projections well into the XIXth century. Our methods for forecasting lung cancer mortality and from this smoking/nonsmoking attributable mortality contain embedded in them estimates of total mortality by age. If these are contrasted with those of the UN we should expect important agreement. After all the UN projections too contain embedded in them assumptions about time trends that must apply to smoking and non-smoking related diseases. A comparison of the two sets should at least help us judge the reasonableness of our lung our cancer mortality forecasts. Figures 9a-9c display the age-specific death rates for 2015-2020 from the UN projections and the ones associated with our forecasts in 2018 (Chile, males). There is close agreement for the youngest age group (the figure does not do justice to the remarkably small differences). The small discrepancies that do exist cannot be due to the modeling of smoking since this behavior should make little difference at young ages. These differences are more likely due to adjustments and corrections present in the original data-base we use in this paper. The discrepancies increase at older age groups (see figure9b and 9c) to the point that in the oldest group shown our forecasts imply that mortality should stop declining altogether from about 2010 onward. Since we do yet have estimates of observed mortality for years past 2008, it is difficult to tell whether the stalling of mortality decline at older ages is an observed feature or a result of incorrect modeling.

The second validity test is harsher but requires more rigid assumptions. Assume that the relative risks of lung cancer and other illnesses estimated for the years (and countries) for which they can be calculated (when surveys of smoking prevalence took place) remain constant over time into 2020. Assume further that nonsmokers lung cancer mortality rates remain constant over time. It is straightforward to show that the age specific smoking prevalence for all years up until 2020 should be given by

$$S(x,t) = (M(x,t)^{TOT} - M(x,t)^{N})/(M_K^{N}(x,t) * (RR_K(x)-1) + M_L^{N}(x,t) * (RR_L(x)-1))$$

where S(x,t) is the estimated prevalence of smoking at age x and time t, $M(x,t)^{TOT}$ is the total mortality rate in the population at age x and time t, $M(x,t)^{N}$ is the counterfactual total mortality at age x and time t, $M_K^{N}(x,t)$ is the non smokers mortality due to group of causes K at age x and time t, $RR_K(x)$ is the relative risks due to group of cause K associated with smoking, $M_L^{N}(x)$ is the lung cancer mortality rate among non smokes at age x (assumed to be constant over time), and $RR_L(x)$ is the relative risk of lung cancer among smokers.

If estimation and forecasting were reasonably accurate we expect that the estimates of smoking prevalence derived from the forecasts should follow the movements of cohorts as they pass through critical ages when mortality risks begin to be felt. In Figures 10a-10c we display observed and "estimated" smoking prevalence for three years: 2000, 2010 and 2018. The expectation is that the derived estimates of prevalence should pivot over observed prevalence and the pivoting must reflect the passage of cohorts. The sequence of Figures 10a through 10c shows exactly this. Furthermore, Figure 10c contains the expected prevalence in 2018, roughly 15 years after the survey. Individual who will be aged 65 in 20018 were aged about 50 in 2004 and the derived prevalence should be closer to the observed prevalence among those who are 50 than among those who were 65 in 2004. The same applies to those aged 70 and 75 in 2018: the "forecast" of smoking prevalence should be closer to (but lower than) the smoking prevalence among those aged 55 and 60 in 2004. The agreement is purely informal but satisfying. The discordant note is that the smoking prevalence at age 50 in 2018 is about .20 and this is about 25% lower than the smoking prevalence assessed among those aged 35 in 2004 and about 50% lower the smoking prevalence detected in the same age group in the 2006 survey. We do not know whether these discrepancies are a product of failure of our forecasts or simply of sampling/reporting errors in the survey or both[24].

Summary and discussion

The application of a modified technique to retrieve estimates of smoking-attributable mortality is only partially new. In this paper we introduce variants to the method to make it suitable for

[24] As we did in the previous test, the second test was replicated in other countries with requisite data (in the case of the second test, smoking prevalence). The results we obtain are similar across countries and we chose to illustrate them with the case of Chile

applications in contexts with less than ideal quality vital statistics. The results we obtain from this application confirm for the entire LAC region findings uncovered with a similar application to a handful of countries. In particular, the damage of past smoking is detectable in several cohorts, is growing over time, and behaves in agreement with the stage theory of the smoking epidemic. An important finding is that our estimates are sensitive to both the model specification, and more so, to the definition used to group causes of death. This finding was somewhat obscured in our previous paper and deserves notice.

A newer development of the paper is the blending of the aforementioned technique with time series analysis to generate short/medium term forecasts of smoking-attributable mortality. We assign heavy emphasis to model uncertainty particularly because in many cases we cannot discriminate between different models that fit equally well but entail potentially different estimates and forecasts. The consequence of our agnostic treatment is a proliferation of forecasts that can be summarized using means and ranges of estimates. We show, here again, that the uncertainty bandwidth is driven by model uncertainty and grouping of causes of deaths. Uncertainty regarding the modeling of trends is less influential as long as one restricts attention to extra-parameterized models such as the logit model. Even in the presence of model driven uncertainty, the results suggest that the impact of smoking will grow among males and females, will spread across all countries, and will attain sizeable magnitudes.

Finally, we made an effort to at least provide informal testing of the underlying assumptions on which the forecasts rests. The first test, a comparison of UN mortality projection and the total levels of mortality implied by our forecasts of lung cancer and of future EMR suggest that there is reasonable agreement at younger ages but much less so at older ages. In fact the total levels of mortality embedded in our forecasts of lung cancer death rates and of EMR imply a deceleration and halting of mortality decline.

The second test is informal and suggests that the estimates of smoking prevalence implied by our forecasts roughly behave according to expectations but depart somewhat from them at the youngest ages. The test is coarse and although agreement is rewarding it does not constitute a water prove of validity.

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Table 1. Stages of the Tobacco Epidemic

	Lopez et al 1994	Ezzati & Lopez 2004
	Stage I	**Early**
Males Smoking Prevalence	<15%	10%
Females Smoking Prevalence	<5%; rarely >10%	<10%
Cigarette Consumption	<500, mostly by men	Ratio Male/Female: 1/1.75
Deaths due to Smoking	Not evident	
Span	1-2 decades	
Tobacco Control	Undeveloped	
	Stage II	**Rising**
Males Smoking Prevalence	50-80%	From 25% (age 15) to 35% (age 40); from 35% (age 50) to 20% (age 80)
Females Smoking Prevalence	Lagging behind men 1-2 decades. Rapidly increasing.	From 20% (age 15) to 25% (age 20); 25% (ages 20-40); from 25% (age 40) to 10% (ages 80)
Yearly Cigarette Consumption	1000-3000, mostly by men (probably 2000-4000)	Ratio Male/Female: 1/1.75
Deaths due to Smoking	Males: 10%; lung cancer rate has risen 10-fold (from 5 to 50/100000). Females: <<10%; lung cancer rate has risen from 8 to 10/100000	
Span	2-3 decades	
Ex-smokers	%very low	
Tobacco Control	Not well developed	
	Stage III	**Peak or Maturity**
Males Smoking Prevalence	Starts to decline probably after being 60% for a long period (goes to 40%). Prevalence lower among middle age & older.	From 40% (age 15) to 60% (age 30); 60% (ages 30-60); from 60% (age 60) to 40% (age 80)
Females Smoking Prevalence	35-45%. Young 40-50%; 50-60: <10%. At the end of Stage III starts after a plateau.	From 30% (age 15) to 40% (age 20); 40% (ages 20-50); from 40% (age 50) to 20% (age 80)
Yearly Cigarette Consumption	3000-4000 males; 1000-2000 females	Ratio Male/Female: 1/1.50
Deaths due to Smoking	Males: 25-30%; lung cancer rate: 110-120/100000). Females:5%; lung cancer rate: 25-30/100000	
Span	Around 3 decades	
Tobacco Control	Favorable conditions.	
	Stage IV	**Declining**
Males Smoking Prevalence	33-35%	From 30% (age 15) to 40% (age 30); 40% (ages 30-60); from 40% (age 60) to 30% (age 80)
Females Smoking Prevalence	30%	From 25-30% (age 15-30); 30% (ages 30-50); from 30% (age 50) to 15% (age 80)
Yearly Cigarette Consumption		Ratio Male/Female: 1/1.25
Deaths due to Smoking	Males: <30%, 40-45% at middle age, falling to 30% in 1 or 2 decades. Lung cancer rate falling 20% in 1-2 decades. Females: 20-25%	
Tobacco Control	Smoke free environment an issue	
		Late
Males Smoking Prevalence		From 20% (age 15) to-25% (age 20); 25% (ages 20-50); from 25% (age 50) to 15% (age 80)
Females Smoking Prevalence		From 15% (age 15) to 20% (age 20); 20% (ages 20-50); from 20% (age 50) to 5% (age 80)
Yearly Cigarette Consumption		Ratio Male/Female: 1/1.25

Table 2.a. Latin American and Caribbean Countries: Stages of the Tobacco Epidemic

	Epidemic Stage	Reference
Central & North America		
Belize	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Costa Rica	Ending 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
El Salvador	Late 1 - Early 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Guatemala	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	1 (Rural) 3 (Urban)	Barnoya in Drope et al. 2007
Honduras	Late 1 - Early 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Mexico	Late 1 - Early 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	Ending 3	Franco-Marina 2007[3]
	2	Champagne et al. 2010[4]
Nicaragua	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Panama	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
South America		
Argentina	Late 2	da Costa e Silva & Koifman 1998[2]
	3.5-4 (Males)/ 2.5-3 (Females)	Ezzati & Lopez 2004[1]
	3	Martínez et al. 2006[5]
	3	Mejia in Drope et al. 2007[6]
	3 (Buenos Aires)	Champagne et al. 2010[4]
Bolivia	Late 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Brazil	Late 2	da Costa e Silva & Koifman 1998[2]
	3.5-4 (Males)/ 2.5-3 (Females)	Ezzati & Lopez 2004[1]
	3 (Brazilian Capitals)	Correa et al. 2009[7]
Chile	Late 2	da Costa e Silva & Koifman 1998[2]
	3.5-4 (Males)/ 2.5-3 (Females)	Ezzati & Lopez 2004[1]
	Early 3 (Santiago)	Champagne et al. 2010[4]
Colombia	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	2 (Bogota)	Champagne et al. 2010[4]
Ecuador	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	2 (Quito)	Champagne et al. 2010[4]
Guyana	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Paraguay	1	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]

Table 2.a Latin American and Caribbean Countries: Stages of the Tobacco Epidemic – Cont.

Peru	Late 1 - Early 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	2 (Lima)	Champagne et al. 2010[4]
Suriname	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Uruguay	Late 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Venezuela	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	2 (Barquisimeto)	Champagne et al. 2010[4]
Caribbean		
Antigua & Barbuda	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Bahamas	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Barbados	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Cuba	Ending 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 2.5-3 (Females)	Ezzati & Lopez 2004[1]
Dominica	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Dominican Republic	Late 1 - Early 2	da Costa e Silva & Koifman 1998[2]
	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
	2	Dozier et al. 2006[8]
Grenada	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Haiti	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
Jamaica	2.5-3 (Males)/ 1.5-2 (Females)	Ezzati & Lopez 2004[1]
St. Kitts & Nevis	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
St. Lucia	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & López 2004[1]
St. Vincent and the Grenadines	2.5-3 (Males)/ 0.5-1 (Females)	Ezzati & Lopez 2004[1]
Trinidad & Tobago	2.5-3 (Males) 1.5-2 (Females)	Ezzati & Lopez 2004[1]

[1] Ezzati and Lopez (2004) evaluation based on data drawn from the Global Burden of Disease (GBD) mortality database and the American Cancer Society Cancer Prevention Study, Phase II (CPS-II).
[2] da Costa e Silva & Koifman (1998) evaluation based on data drawn from the 1997 WHO database.
[3] Franco-Marina (2007) evaluation based on data from the Mexican National Addiction Surveys 1988, 1993, 1998, and 2002.
[4] Champagne et al. 2010 evaluation based on data from the Cardiovascular Risk Factor Multiple Evaluation in Latin America (CARMELA) study.
[5] Martínez et al. 2006 evaluation based on data from the study Monitoring and Evaluation of Social Programs System (SIEMPRO) 2001.
[6] Mejia in Drope et al. 2007 evaluation based on data from the study National Survey of Risk Factors (ENFR) 2005.
[7] Correa et al. 2009 evaluation based on data from the study Brazilian Household Survey on Non Communicable Diseases Risk Factors (2002–2003) and the Brazilian Mortality System (2003).
[8] Dozier et al. 2006 evaluation based on their own data.

Table 2.b. Characteristics of the Tobacco Epidemic in Argentina, Brazil, Chile, Mexico and Uruguay

	Argentina 2005	Brazil 2008	Chile 2006	Mexico 2009	Uruguay 2009
Males					
Smoking Prevalence (%)	38	24	42	26	33
Mean Number Cigarettes per Day	12	15	5	10	11
Mean Number Cigarettes per Year	4539	5564	1889	3548	4117
Proportion of Deaths (all Causes) Attributable to Tobacco (%) [1]	19	15	11	7	24
Death Rate (per 100000) Attributable to Tobacco (Trachea, Bronchus, and Lung Cancers) [1]	75	35	32	71	92
Females					
Smoking Prevalence (%)	26	15	31	8	21
Mean Number Cigarettes per Day	9	13	4	8	11
Mean Number Cigarettes per Year	3378	4731	1507	2865	3836
Yearly Consumption Ratio Female/Male	0.74	0.85	0.80	0.81	0.93
Proportion of Deaths (all Causes) Attributable to Tobacco (%) [1]	6	6	8	6	5
Death Rate (per 100000) Attributable to Tobacco (Trachea, Bronchus, and Lung Cancers) [1]	12	6	10	37	46

[1] WHO (2012) Estimated proportion deaths attributable to tobacco and deaths rates correspond to 2004 and are totals for individuals aged 30 and over.

Table 3a
Means and Lower and Upper Bounds of Relative Differences between Observed and Counterfactual Life Expectancies at Age 50 (E50), 2000-2005. Males

Country	Year	Mean	Lower	Upper		Country	Year	Mean	Lower	Upper
Argentine	2000	0.11	0.08	0.14		**Cuba**	2004	0.19	0.17	0.21
Argentine	2001	0.10	0.07	0.13		**Cuba**	2005	0.20	0.18	0.22
Argentine	2002	0.10	0.07	0.13		**Dominican Rep.**	2000	0.01	0.01	0.01
Argentine	2003	0.10	0.07	0.13		**Dominican Rep.**	2001	0.02	0.02	0.03
Argentine	2004	0.10	0.07	0.13		**Dominican Rep.**	2003	0.01	0.01	0.01
Argentine	2005	0.10	0.07	0.13		**Dominican Rep.**	2004	0.02	0.02	0.03
Brazil	2000	0.05	0.02	0.08		**Dominican Rep.**	2005	0.02	0.01	0.02
Brazil	2001	0.05	0.02	0.08		**El Salvador**	2000	0.00	0.00	0.00
Brazil	2002	0.05	0.02	0.08		**El Salvador**	2001	0.00	0.00	0.01
Brazil	2003	0.05	0.02	0.08		**El Salvador**	2002	0.01	0.00	0.02
Brazil	2004	0.05	0.02	0.08		**El Salvador**	2003	0.01	0.00	0.01
Brazil	2005	0.05	0.02	0.09		**El Salvador**	2004	-0.02	-0.02	-0.01
Chile	2000	0.10	0.10	0.11		**El Salvador**	2005	0.00	0.00	0.00
Chile	2001	0.10	0.09	0.10		**Mexico**	2000	0.03	0.02	0.04
Chile	2002	0.10	0.09	0.10		**Mexico**	2001	0.03	0.03	0.04
Chile	2003	0.10	0.09	0.10		**Mexico**	2002	0.03	0.03	0.04
Chile	2004	0.11	0.10	0.11		**Mexico**	2003	0.03	0.03	0.04
Chile	2005	0.11	0.10	0.11		**Mexico**	2004	0.03	0.03	0.04
Colombia	2000	0.11	0.07	0.14		**Mexico**	2005	0.03	0.03	0.04
Colombia	2001	0.09	0.06	0.12		**Panama**	2000	0.03	0.01	0.04
Colombia	2002	0.10	0.06	0.13		**Panama**	2001	0.04	0.02	0.06
Colombia	2004	0.12	0.08	0.15		**Panama**	2002	0.03	0.02	0.05
Colombia	2005	0.12	0.08	0.16		**Panama**	2003	0.05	0.03	0.08
Costa Rica	2000	0.05	0.03	0.08		**Panama**	2004	0.07	0.04	0.10
Costa Rica	2001	0.06	0.03	0.10		**Uruguay**	2000	0.10	0.09	0.11
Costa Rica	2002	0.05	0.03	0.08		**Uruguay**	2001	0.11	0.10	0.13
Costa Rica	2003	0.05	0.03	0.08		**Uruguay**	2004	0.11	0.10	0.12
Costa Rica	2004	0.04	0.02	0.06		**Venezuela**	2000	0.05	0.03	0.07
Costa Rica	2005	0.05	0.02	0.07		**Venezuela**	2001	0.06	0.04	0.08
Cuba	2000	0.16	0.14	0.18		**Venezuela**	2002	0.05	0.03	0.07
Cuba	2001	0.16	0.14	0.17		**Venezuela**	2003	0.06	0.04	0.08
Cuba	2002	0.17	0.16	0.19		**Venezuela**	2004	0.06	0.04	0.08
Cuba	2003	0.18	0.17	0.20		**Venezuela**	2005	0.06	0.04	0.08

Table 3b
Means and Lower and Upper Bounds of Relative Differences between Observed and Counterfactual Life Expectancies at Age 50 (E50), 2000-2005. Females

Country	Year	Mean	Lower	Upper		Country	Year	Mean	Lower	Upper
Argentine	2000	0.00	0.00	0.00		**Cuba**	2004	0.08	0.07	0.09
Argentine	2001	0.01	-0.01	0.01		**Cuba**	2005	0.09	0.07	0.10
Argentine	2002	0.00	-0.01	0.01		**Dominican Rep.**	2000	-0.02	-0.04	-0.01
Argentine	2003	0.01	-0.01	0.01		**Dominican Rep.**	2001	-0.02	-0.03	-0.01
Argentine	2004	0.00	0.00	0.00		**Dominican Rep.**	2003	-0.02	-0.04	-0.01
Argentine	2005	0.00	0.00	0.00		**Dominican Rep.**	2004	-0.02	-0.03	-0.01
Brazil	2000	-0.01	-0.02	-0.01		**Dominican Rep.**	2005	-0.02	-0.02	-0.01
Brazil	2001	-0.01	-0.02	-0.01		**El Salvador**	2000	-0.03	-0.04	-0.02
Brazil	2002	-0.01	-0.02	-0.01		**El Salvador**	2001	-0.02	-0.03	-0.02
Brazil	2003	-0.01	-0.01	-0.01		**El Salvador**	2002	-0.05	-0.07	-0.04
Brazil	2004	-0.01	-0.01	0.00		**El Salvador**	2003	-0.03	-0.04	-0.03
Brazil	2005	0.00	-0.01	0.00		**El Salvador**	2004	-0.03	-0.04	-0.03
Chile	2000	0.01	0.01	0.02		**El Salvador**	2005	-0.04	-0.05	-0.03
Chile	2001	0.01	0.01	0.02		**Mexico**	2000	-0.01	-0.01	0.00
Chile	2002	0.02	0.01	0.03		**Mexico**	2001	-0.01	-0.01	0.00
Chile	2003	0.02	0.01	0.03		**Mexico**	2002	-0.01	-0.01	0.00
Chile	2004	0.02	0.01	0.03		**Mexico**	2003	-0.01	-0.01	0.00
Chile	2005	0.02	0.01	0.04		**Mexico**	2004	-0.01	-0.01	0.00
Colombia	2000	0.01	0.00	0.01		**Mexico**	2005	-0.01	-0.01	0.00
Colombia	2001	0.01	0.01	0.02		**Panama**	2000	-0.01	-0.01	-0.01
Colombia	2002	0.01	0.01	0.02		**Panama**	2001	-0.02	-0.02	-0.01
Colombia	2004	0.02	0.01	0.03		**Panama**	2002	-0.03	-0.04	-0.02
Colombia	2005	0.02	0.01	0.03		**Panama**	2003	0.02	0.02	0.03
Costa Rica	2000	0.02	0.01	0.02		**Panama**	2004	0.00	0.00	0.00
Costa Rica	2001	-0.02	-0.02	-0.01		**Uruguay**	2000	0.01	0.02	0.05
Costa Rica	2002	-0.02	-0.03	-0.01		**Uruguay**	2001	0.03	0.04	0.08
Costa Rica	2003	-0.01	-0.01	-0.01		**Uruguay**	2004	0.01	0.01	0.03
Costa Rica	2004	-0.01	-0.01	-0.01		**Venezuela**	2000	0.01	0.00	0.01
Costa Rica	2005	-0.02	-0.03	-0.01		**Venezuela**	2001	0.01	0.00	0.01
Cuba	2000	0.06	0.05	0.07		**Venezuela**	2002	0.01	0.00	0.01
Cuba	2001	0.06	0.05	0.07		**Venezuela**	2003	0.01	0.01	0.02
Cuba	2002	0.07	0.06	0.08		**Venezuela**	2004	0.01	0.00	0.01
Cuba	2003	0.07	0.06	0.08		**Venezuela**	2005	0.02	0.01	0.02

Table 4a
Forecast Means and Lower and Upper Bounds of Years of Life Lost at Age 50, 2010-2018. Males

Country	Year	Mean	Lower	Upper		Country	Year	Mean	Lower	Upper
Argentine	2010	0.09	0.08	0.10		**Ecuador**	2010	0.03	0.02	0.03
Argentine	2015	0.10	0.09	0.11		**Ecuador**	2015	0.05	0.02	0.07
Argentine	2018	0.11	0.10	0.12		**Ecuador**	2018	0.06	0.03	0.10
Brazil	2010	0.05	0.05	0.06		**Guatemala**	2010	0.00	0.00	0.00
Brazil	2015	0.06	0.05	0.07		**Guatemala**	2015	0.00	0.00	0.00
Brazil	2018	0.07	0.05	0.08		**Guatemala**	2018	0.00	0.00	0.01
Chile	2010	0.11	0.10	0.11		**Mexico**	2010	0.02	0.01	0.02
Chile	2015	0.12	0.11	0.14		**Mexico**	2015	0.02	0.01	0.03
Chile	2018	0.15	0.13	0.17		**Mexico**	2018	0.02	0.00	0.03
Colombia	2010	0.16	0.12	0.20		**Panama**	2010	0.03	0.03	0.04
Colombia	2015	0.19	0.16	0.24		**Panama**	2015	0.04	0.03	0.05
Colombia	2018	0.23	0.19	0.27		**Panama**	2018	0.04	0.03	0.05
Costa Rica	2010	0.02	0.01	0.03		**Peru**	2010	0.02	0.01	0.03
Costa Rica	2015	0.01	0.00	0.03		**Peru**	2015	0.04	0.03	0.05
Costa Rica	2018	0.01	-0.01	0.03		**Peru**	2018	0.09	0.03	0.11
Cuba	2010	0.20	0.18	0.22		**Uruguay**	2010	0.15	0.13	0.17
Cuba	2015	0.24	0.21	0.27		**Uruguay**	2015	0.18	0.15	0.23
Cuba	2018	0.26	0.22	0.31		**Uruguay**	2018	0.20	0.17	0.27
Dominican Rep.	2010	0.02	0.01	0.03		**Venezuela**	2010	0.05	0.05	0.05
Dominican Rep.	2015	0.02	0.00	0.04		**Venezuela**	2015	0.06	0.06	0.07
Dominican Rep.	2018	0.02	0.01	0.05		**Venezuela**	2018	0.08	0.07	0.10

Table 4b

Forecast Means and Lower and Upper Bounds of Years of Life Lost at Age 50, 2010-2018. Females

Country	Year	Mean	Lower	Upper		Country	Year	Mean	Lower	Upper
Argentine	2010	0.03	0.02	0.04		**Ecuador**	2010	-0.01	-0.01	-0.01
Argentine	2015	0.05	0.02	0.08		**Ecuador**	2015	0.00	-0.01	0.01
Argentine	2018	0.06	0.03	0.10		**Ecuador**	2018	0.01	-0.01	0.02
Brazil	2010	0.03	0.01	0.04		**Guatemala**	2010	0.00	0.00	0.00
Brazil	2015	0.04	0.02	0.06		**Guatemala**	2015	0.00	0.00	0.00
Brazil	2018	0.05	0.03	0.07		**Guatemala**	2018	0.00	-0.01	0.00
Chile	2010	0.03	0.02	0.03		**Mexico**	2010	-0.01	-0.01	0.00
Chile	2015	0.04	0.04	0.05		**Mexico**	2015	-0.01	-0.02	0.00
Chile	2018	0.06	0.05	0.07		**Mexico**	2018	-0.01	-0.03	0.00
Colombia	2010	0.07	0.03	0.10		**Panama**	2010	-0.01	-0.02	-0.01
Colombia	2015	0.09	0.04	0.13		**Panama**	2015	-0.02	-0.04	0.00
Colombia	2018	0.10	0.06	0.17		**Panama**	2018	-0.03	-0.06	0.00
Costa Rica	2010	-0.02	-0.02	-0.01		**Peru**	2010	0.00	-0.01	0.01
Costa Rica	2015	-0.03	-0.03	-0.02		**Peru**	2015	0.04	0.02	0.05
Costa Rica	2018	-0.03	-0.04	-0.02		**Peru**	2018	0.06	0.04	0.09
Cuba	2010	0.12	0.09	0.15		**Uruguay**	2010	0.00	-0.01	0.01
Cuba	2015	0.16	0.11	0.22		**Uruguay**	2015	0.02	0.01	0.06
Cuba	2018	0.19	0.12	0.28		**Uruguay**	2018	0.06	0.04	0.09
Dominican Rep.	2010	0.01	0.00	0.01		**Venezuela**	2010	0.01	0.00	0.01
Dominican Rep.	2015	0.00	0.00	0.01		**Venezuela**	2015	0.01	0.01	0.03
Dominican Rep.	2018	0.01	-0.01	0.02		**Venezuela**	2018	0.04	0.02	0.06

Figure 1a. Mortality Rates: Lung,Trachea, Bronchus Cancer

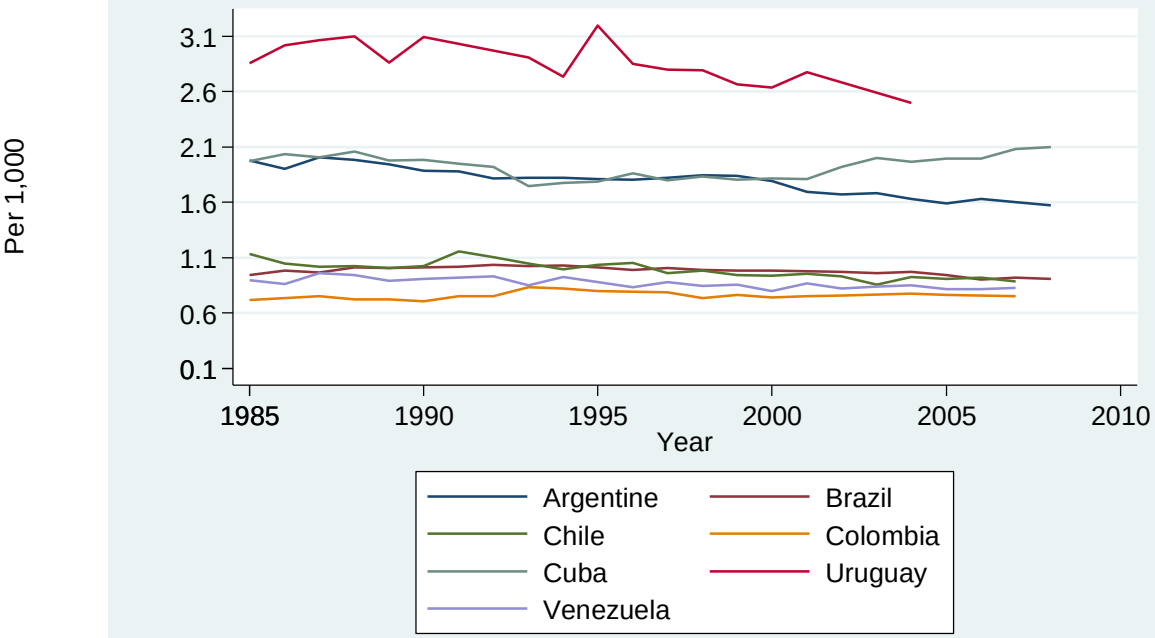


Figure 1b. Lung Cancer Mortality Rates by Country and Year

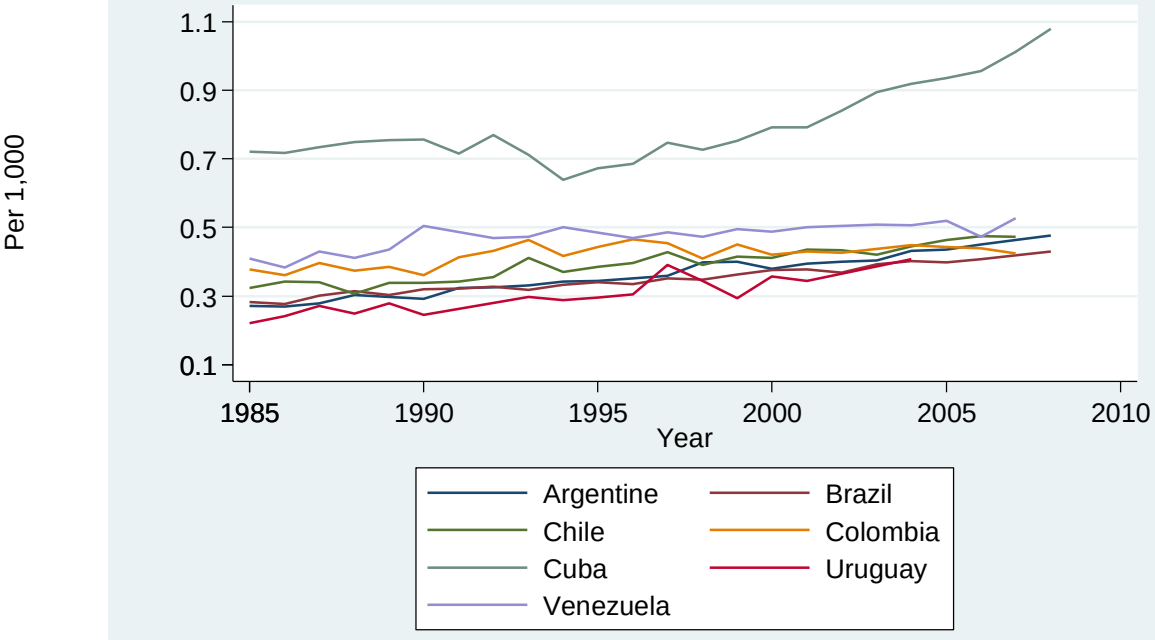


Figure 1c. Circulatory Diseases Death Rates by Country and Year

Males

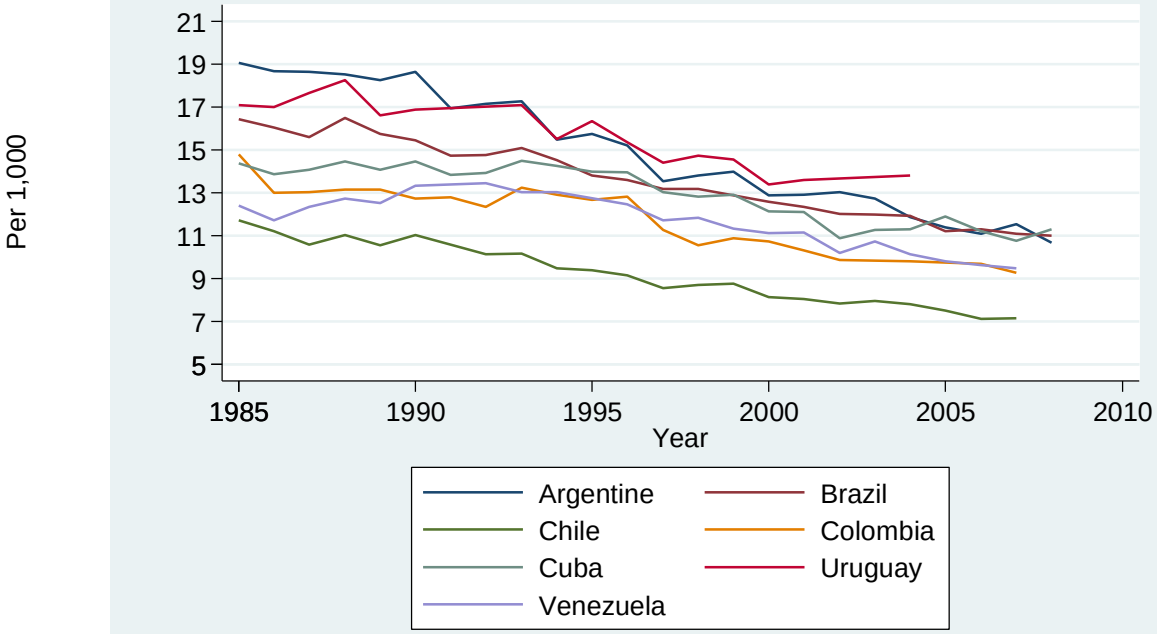


Figure 1d. Circulatory Diseases Death Rates by Country and Year

Females

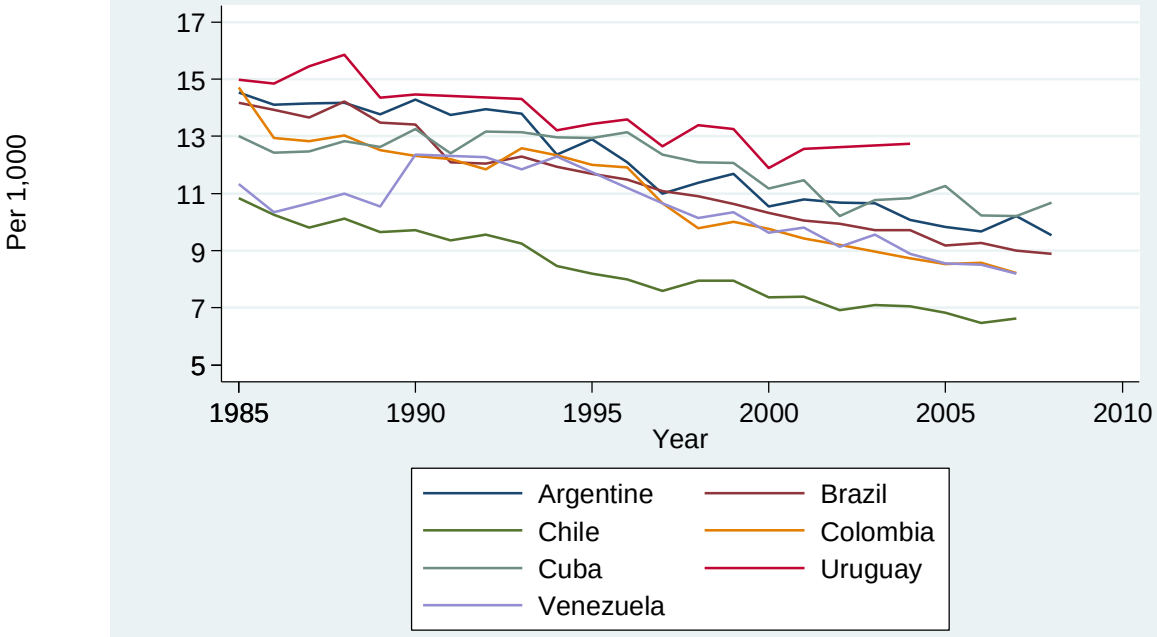
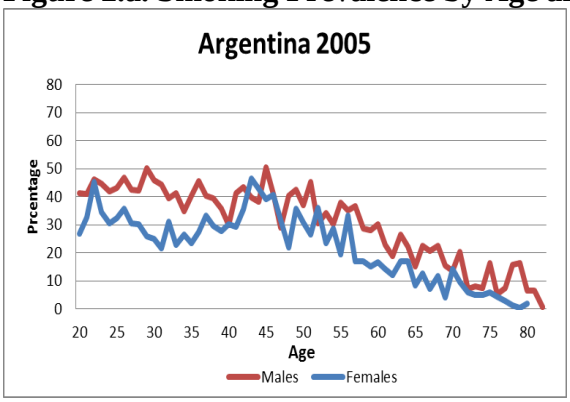


Figure 2.a. Smoking Prevalence by Age and Gender: Argentina and Brazil



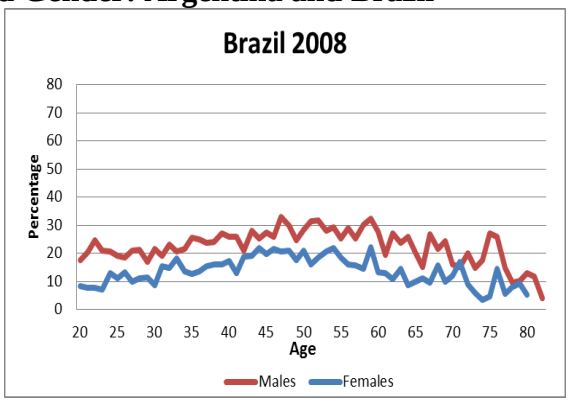
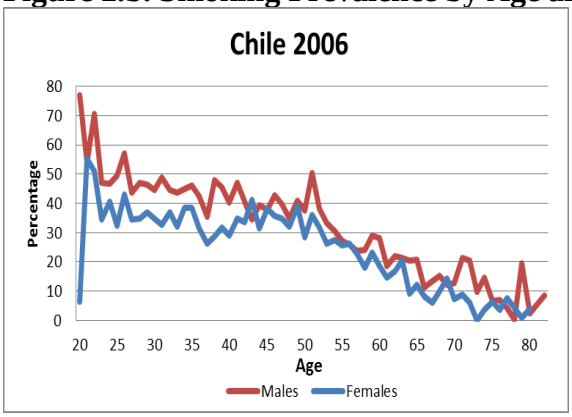


Figure 2.b. Smoking Prevalence by Age and Gender: Chile and Mexico



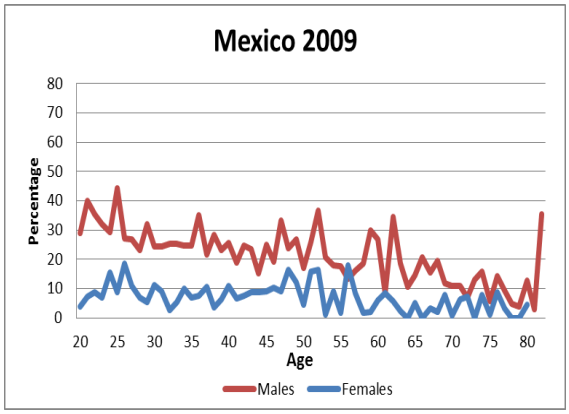
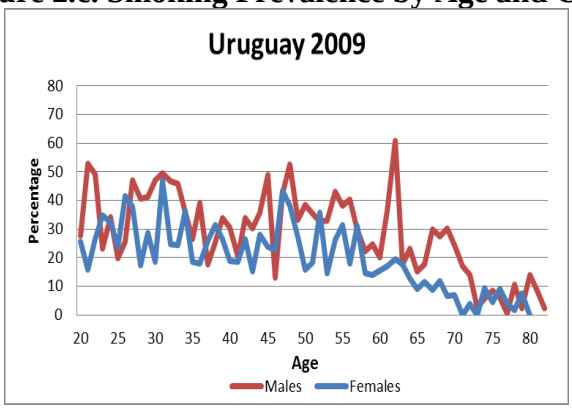


Figure 2.c. Smoking Prevalence by Age and Gender: Uruguay and the US



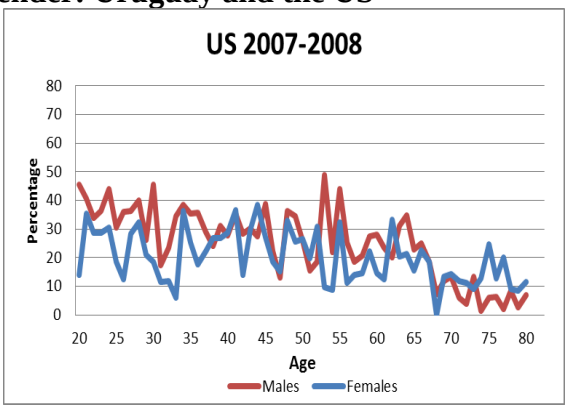
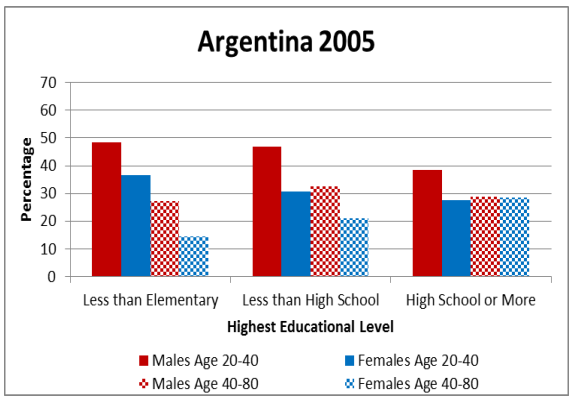


Figure 3.a. Smoking Prevalence by Age, Gender and Educational Attainment: Argentina and Brazil



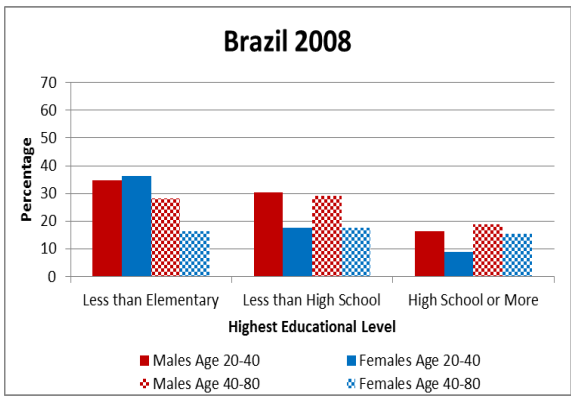
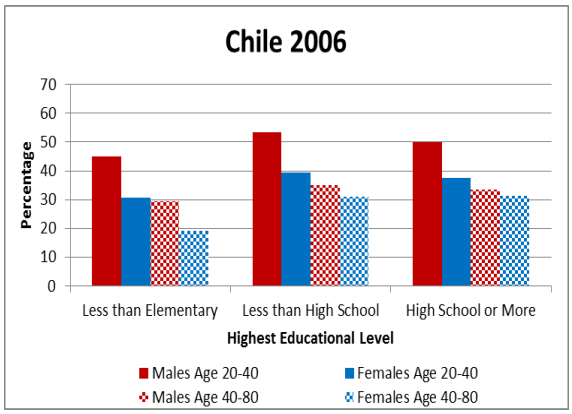


Figure 3.b. Smoking Prevalence by Age, Gender and Educational Attainment: Chile and Mexico



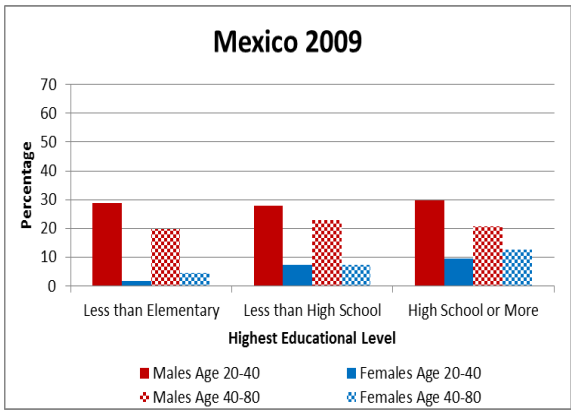
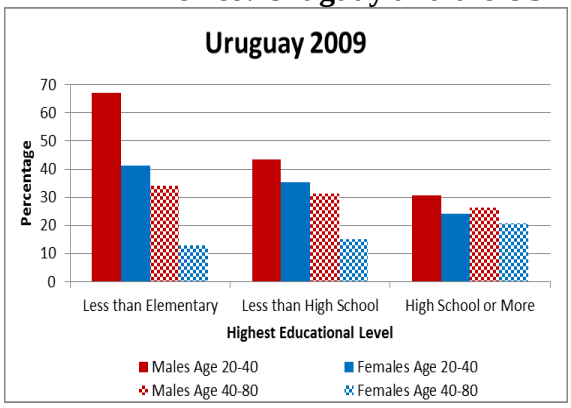


Figure 3.c. Smoking Prevalence by Age, Gender and Educational Attainment: Chile and Mexico: Uruguay and the US



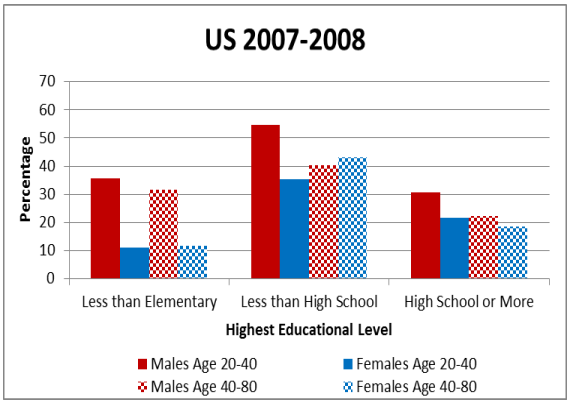


Figure 4:Alternative Estimates of Years of Life Lost (E(50))

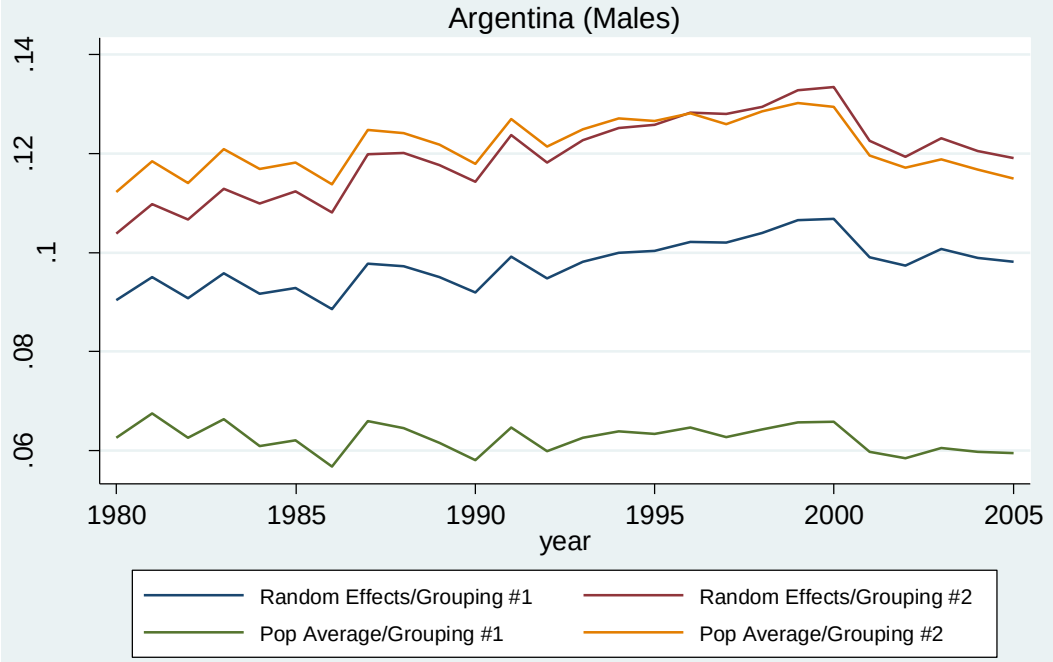


Figure 5a: Relative (mean) differences counterfactual and observed E(50)

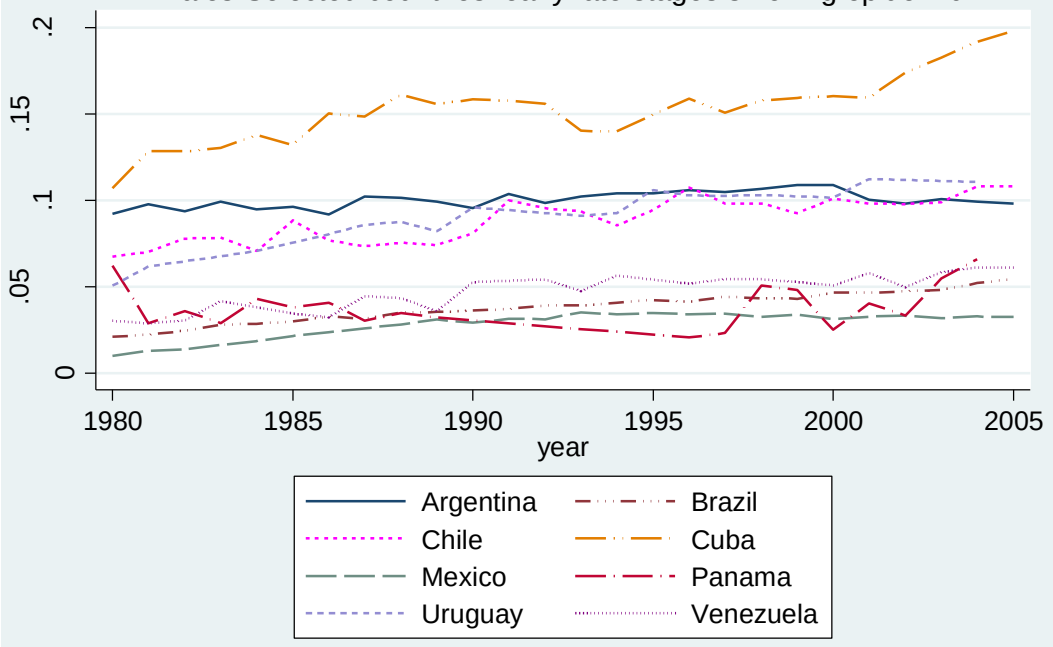


Figure 5b: Relative (mean) differences counterfactual and observed E(50)

Females-Selected countries early-late stages smoking epidemic

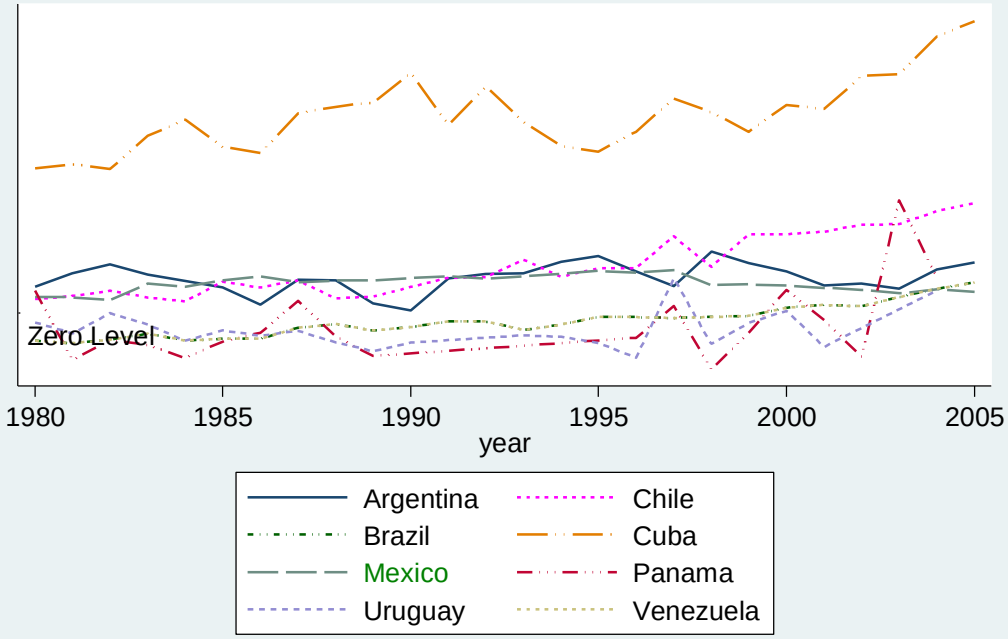
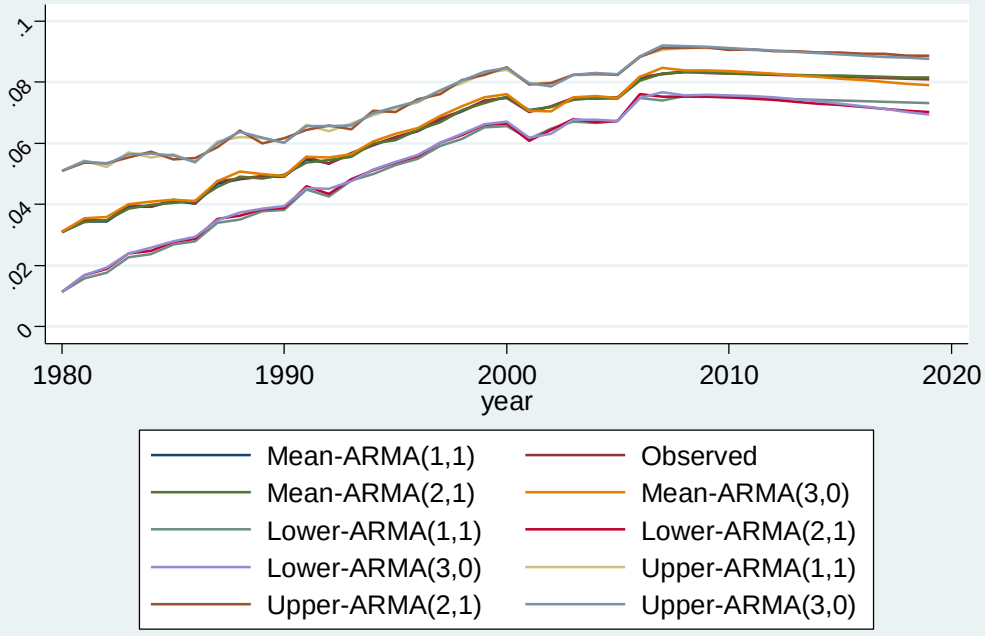


Figure 6: Alternative Forecasts of Relative Years lost at age 50

Argentina, Males



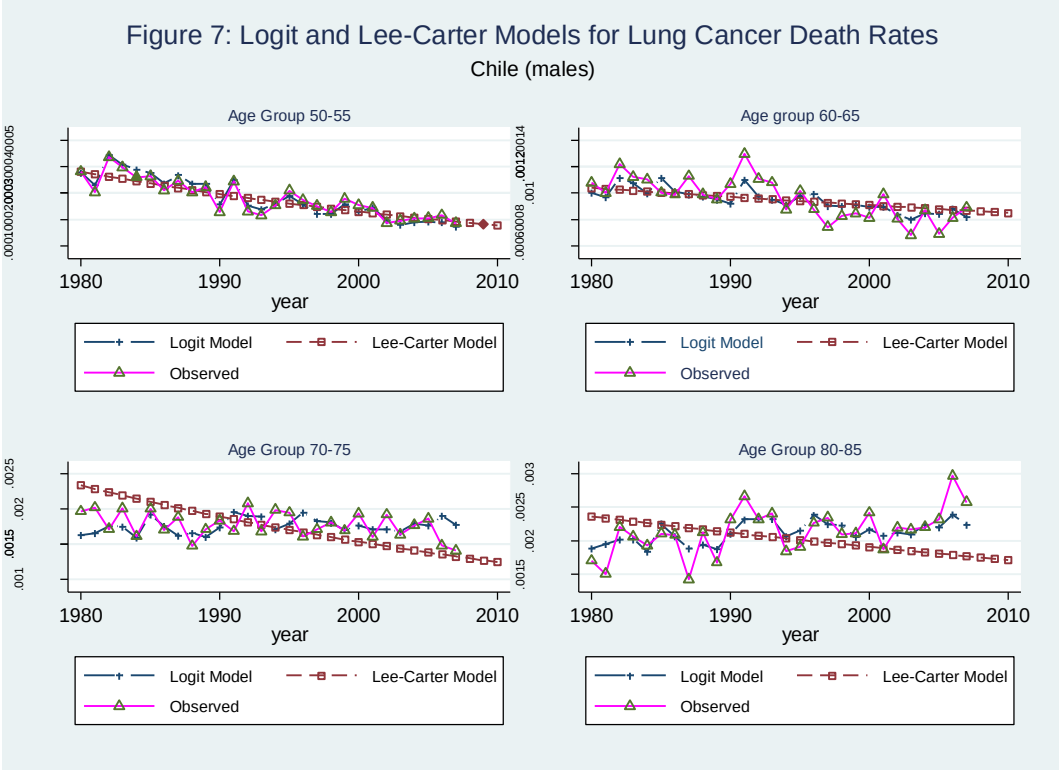


Figure 7: Logit and Lee-Carter Models for Lung Cancer Death Rates
Chile (males)
Age Group 50-55
Age group 60-65
Age Group 70-75
Age Group 80-85
year
1980
1990
2000
2010
Logit Model
Lee-Carter Model
Observed

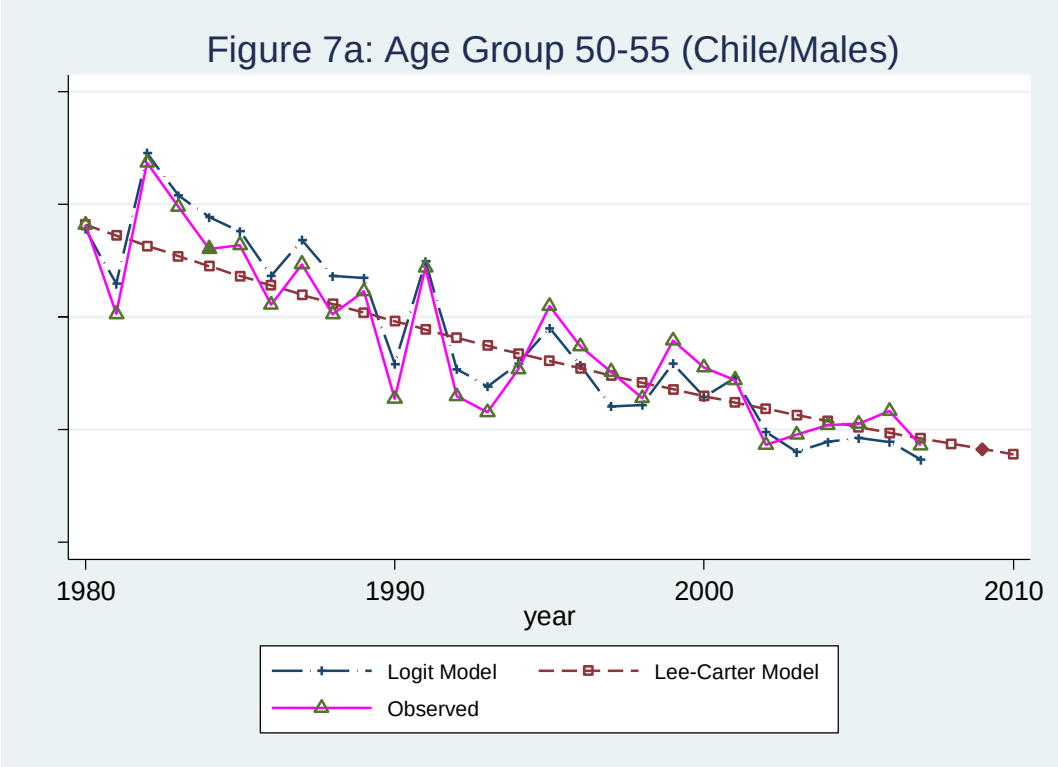


Figure 7a: Age Group 50-55 (Chile/Males)
.0001
.0002
.0003
.0004
.0005
1980
1990
2000
2010
year
Logit Model
Lee-Carter Model
Observed

Figure 7d: Age Group 80-85 (Chile/Males)

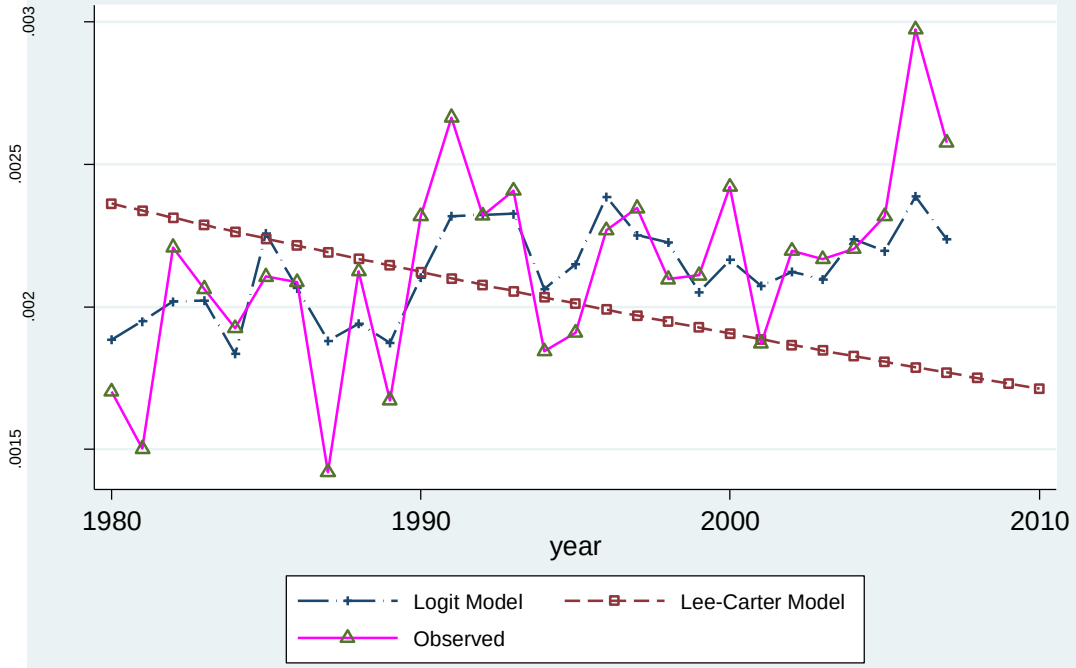


Figure 8a: Alternative Forecasts of Proportionate Years of Life Lost (E(50))

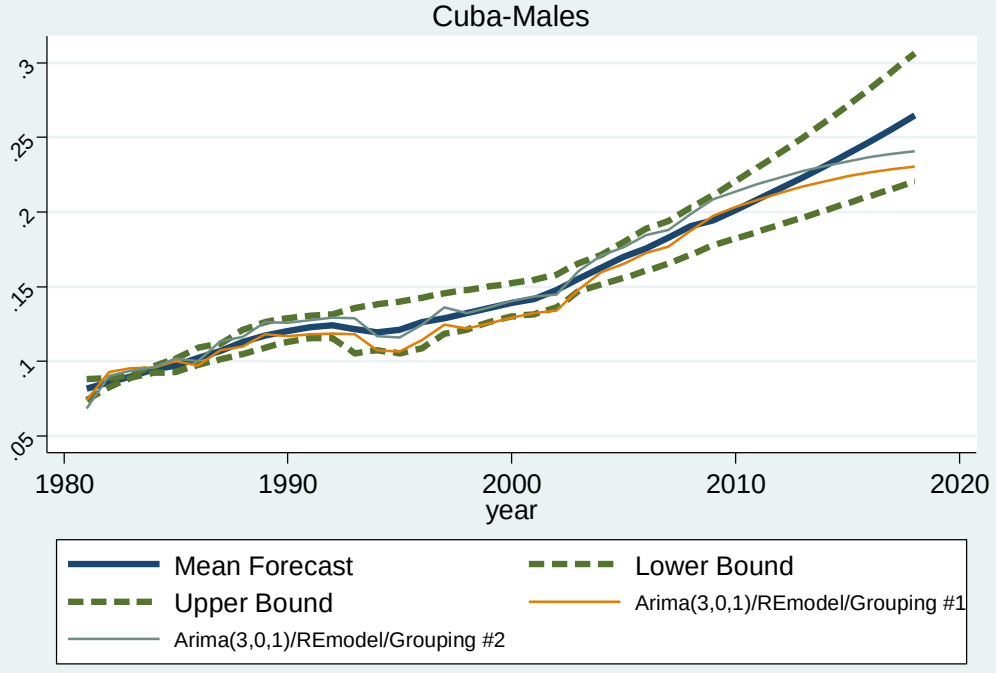


Figure 8b: Alternative Forecasts of Proportionate Years of Life Lost (E(50))

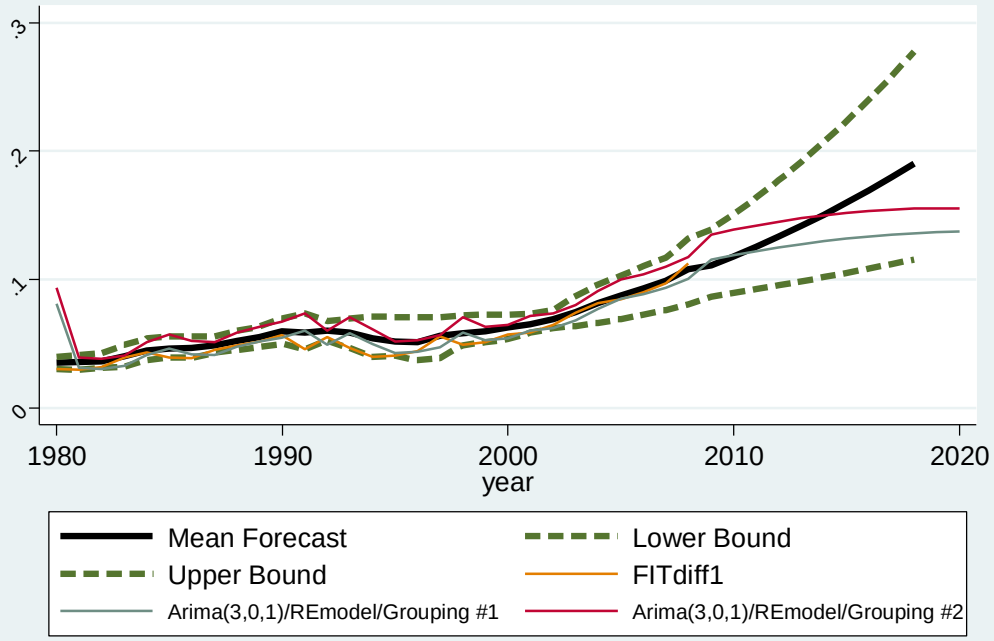
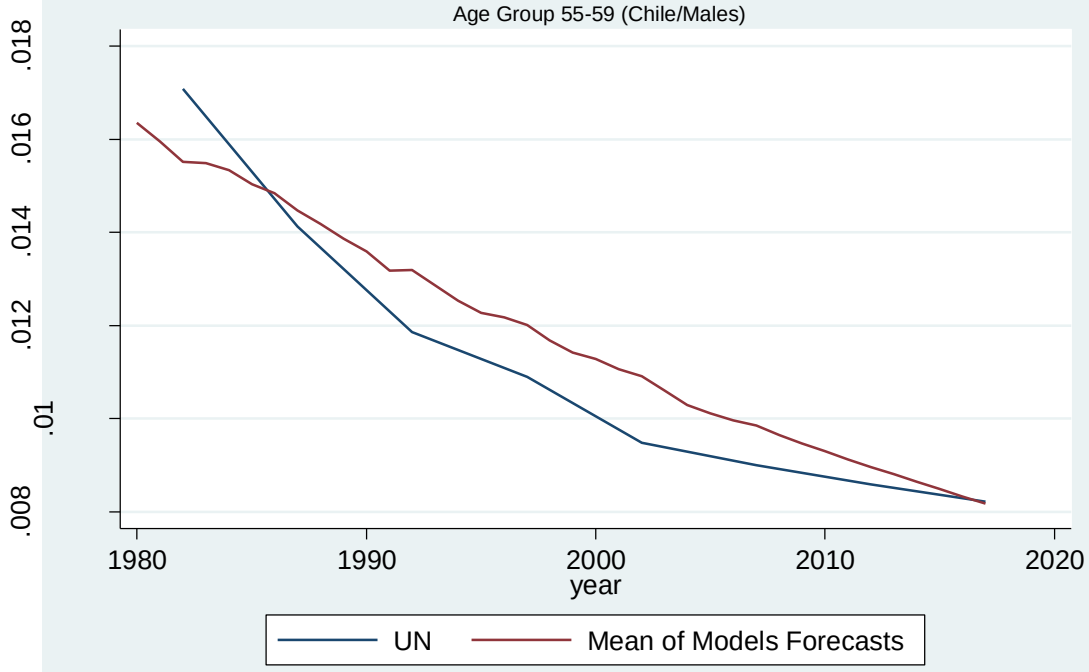


Figure 9a: Comparison Age-Specific Mortality Rates: UN and Model



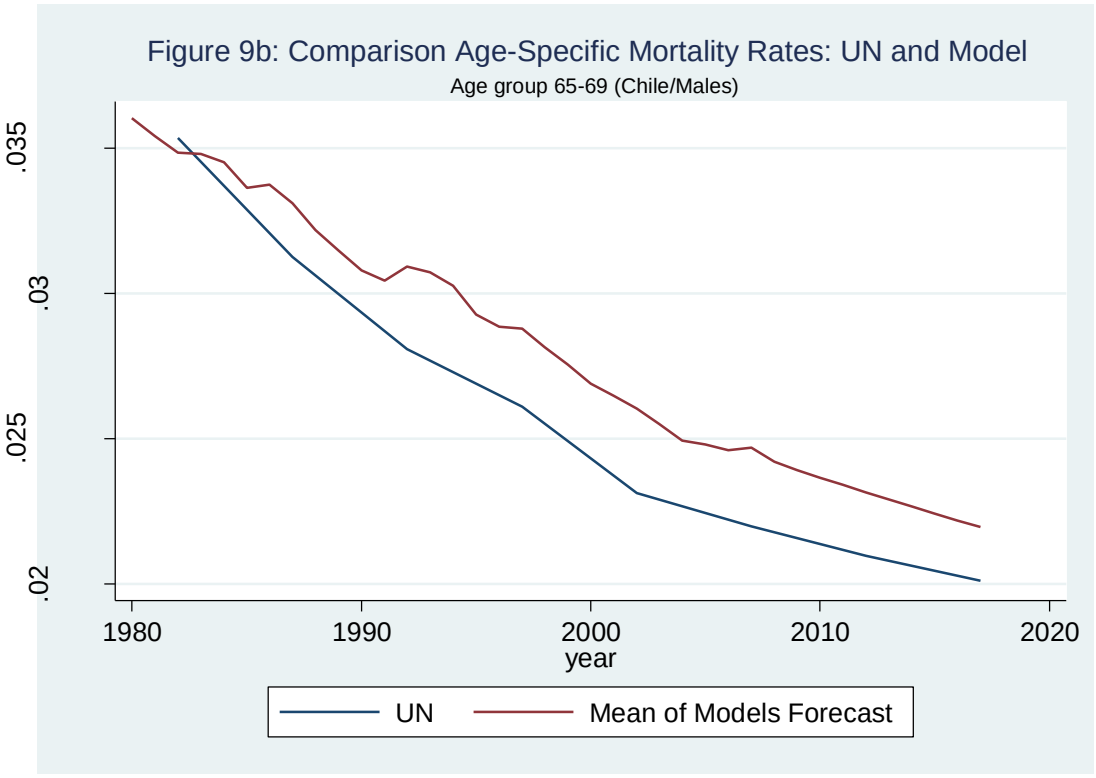


Figure 9b: Comparison Age-Specific Mortality Rates: UN and Model
Age group 65-69 (Chile/Males)
.035
.03
.025
.02
1980
1990
2000
2010
2020
year
UN
Mean of Models Forecast

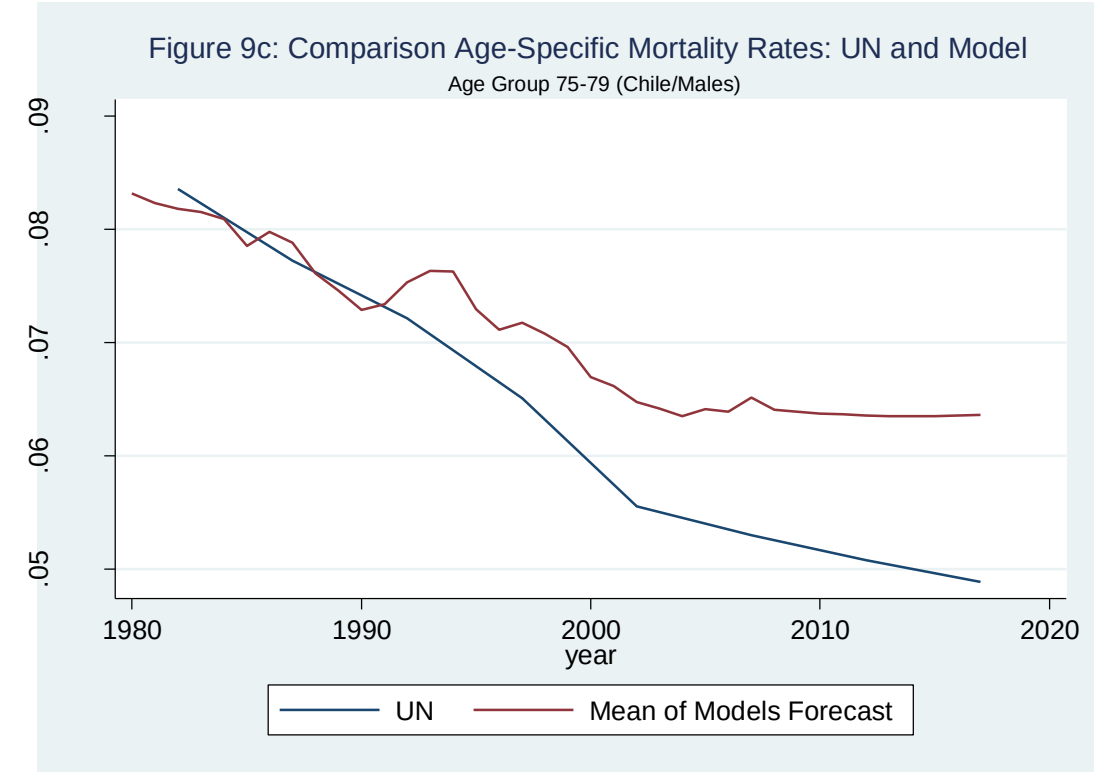


Figure 9c: Comparison Age-Specific Mortality Rates: UN and Model
Age Group 75-79 (Chile/Males)
.09
.08
.07
.06
.05
1980
1990
2000
2010
2020
year
UN
Mean of Models Forecast

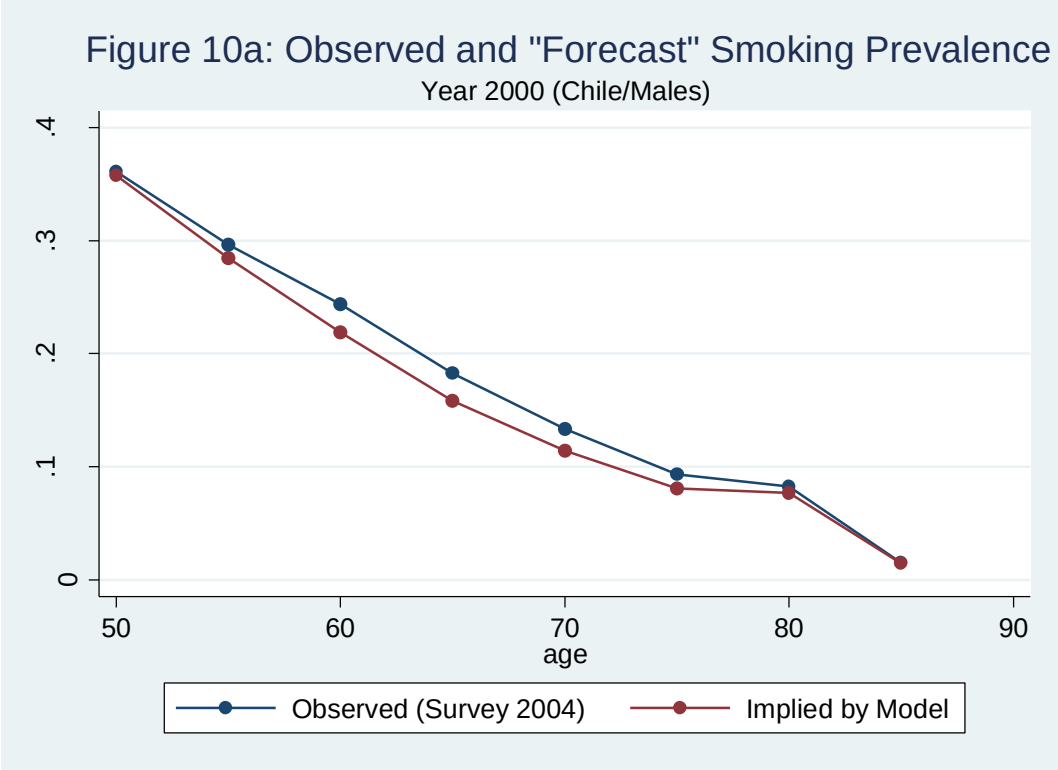


Figure 10a: Observed and "Forecast" Smoking Prevalence
Year 2000 (Chile/Males)
0
.1
.2
.3
.4
50
60
70
80
90
age
Observed (Survey 2004)
Implied by Model

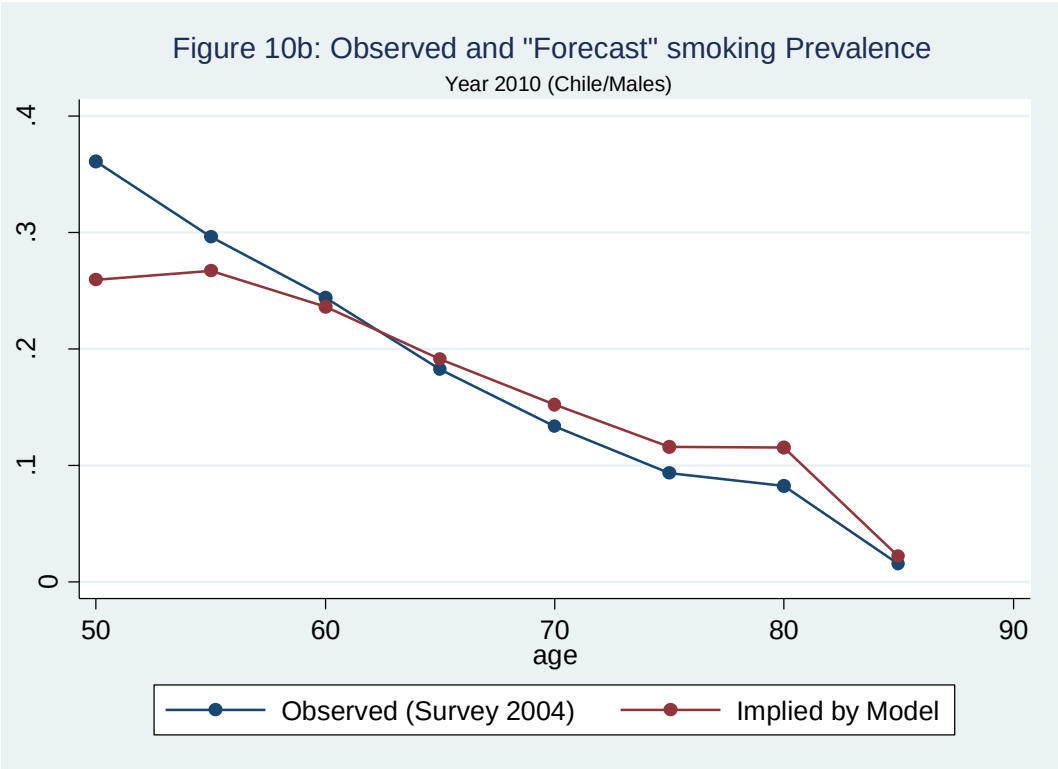
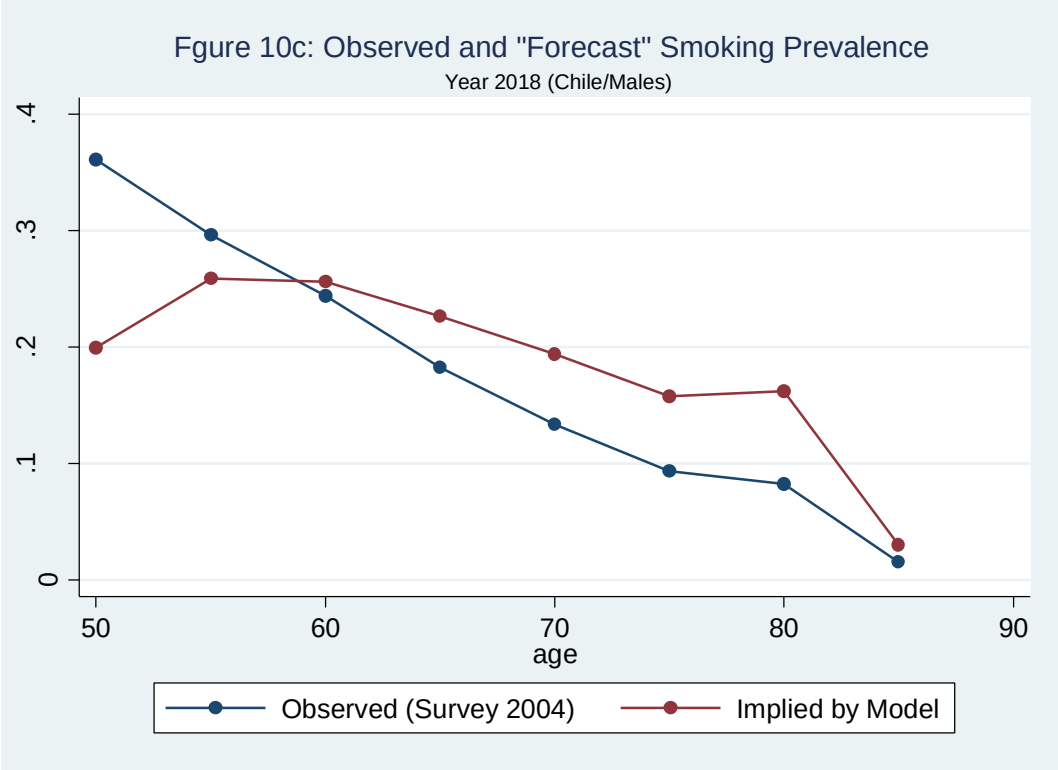


Figure 10b: Observed and "Forecast" smoking Prevalence
Year 2010 (Chile/Males)
0
.1
.2
.3
.4
50
60
70
80
90
age
Observed (Survey 2004)
Implied by Model


Fgure 10c: Observed and "Forecast" Smoking Prevalence
Year 2018 (Chile/Males)
0
.1
.2
.3
.4
50
60
70
80
90
age
Observed (Survey 2004)
Implied by Model

Appendix 1

Classification of Causes of Death according to ICD-10

- Lung, trachea, bronchus cancer (C33, C34)
- Bladder cancer (C67)
- Prostate cancer (C61)
- Breast cancer (C50)
- Uterus/cervix cancer (C53, C54, C55)
- Stomach, intestines, rectum, colon cancer (C16, C17, C18, C19, C20, C21)
- Liver, pancreas cancer (C22, C25)
- Kidney cancer (C64)
- All remaining cancers (total cancers minus causes shown above)
- Total cancers (C00-D48)
- Emphysema, bronchitis, asthma (J40, J41, J42, J43, J45)
- Circulatory diseases (I00-I99)
- Infectious diseases, including pneumonia and influenza (A00-B99, J12, J13, J14, J15, J16, J18, J10, J11)
- Ill-defined causes (R00-R99)
- Remaining well defined causes (Total minus causes shown above)
- Total (AAA)

Appendix 2

Countries and Years in the Data Base for Regression Analysis

Countries	Data Availability (years)	Source	ICD
Argentina	1980-2008	WHO	9,10
Brazil	1980-2008	WHO	9,10
Chile	1980-2007	WHO	9,10
Colombia	1984-2007	WHO	9,10
Costa Rica	1980-2009	WHO	9,10
Cuba	1980-2008	WHO	9,10
Dominican Republic	1980-2005	WHO	9,10
Ecuador	1980-2009	WHO	9,10
El Salvador	1981-2008	WHO	9,10
Guatemala	1980-2008	WHO	9,10
Mexico	1980-2008	WHO	9,10
Nicaragua	1988-2006	WHO	9,10
Panama	1980-2008	WHO	9,10
Paraguay	1980-2008	WHO	9,10
Peru	1980-2007	WHO	9,10
Uruguay	1980-2004	WHO	9,10
Venezuela	1980-2007	WHO	9,10
US	1980-2008	WHO	9,10

Appendix 3

Differences between the Lee-Carter and Brass Logit models for lung cancer

The Brass logit model can be rewritten as follows:

$$H(x,t) = \alpha(t) + (\beta(t)-1)* \phi(M_L^s(x))$$

$$= \alpha(t) + \varphi(t) * \phi(M_L^s(x)) \qquad \text{(A3.1)}$$

where H(x,t) is the **difference** between the (logit-scaled) observed lung cancer mortality rates at age x and time t, $M_L(x,t)$, and the (logit-scaled) standard mortality rates, $\phi(M_L^s(x))$

The Lee-carter model can be rewritten in a similar way:

$$R(x,t) = K(t)* b(x) \qquad \text{(A3.2)}$$

where R(x,t) is the difference between (log-scaled) observed lung cancer mortality rate at age x and time t and the average (log-scaled) lung cancer death rates observed during the period, A(x). Because the values of $M_L(x,t)$ are small, logit and log scaling yield virtually identical quantities. Thus, expressions (A3.1) and (A3.2) refer to approximately the same functions, particularly when in both cases we use the average observed rates to express the standard (either A(x) or $M_L^s(x)$.

Because the functional form on the right hand side of expressions (A3.1) and (A3.2) are analogous, the first and higher derivatives with respect to age and time will have the same form in either case. In each model the partial derivatives with respect to age depend on a product of an age- (but not time) dependent term and a time- (but not age) dependent term. The same holds for the partial derivatives with respect to time. The models are mathematically equivalent: both require an *ex-ante* chosen standard; both include a term to capture shifts over time and across ages and in both cases the effects of time and age are multiplicative and separable. The key difference is that (A3.1) is estimated separately by period whereas (A3.2.) is estimated simultaneously over all periods. The inclusion of additional observations after the last, say T, will change estimates of parameters when (A3.2) but leave all those before T invariant if (A3.1) is used. (A3.1) requires extra parameters to model changes over time and across ages whereas (A3.2) is constrained to use the same parameters to accommodate extra variation whereas (A3.1.) use extra parameters that help separate the impact of changes in the total mortality level from impacts that are age-time specific. Such flexibility is absent in (A3.2) and this will explain is worse performance

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