

ARTICLE

# Dynamic Versus Static Modeling of Mortality-Related Benefits of PM<sub>2.5</sub> Reductions in the USA and Chile: 1990 to 2050

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## Abstract

Economic and health benefits assessments of air quality changes often quantify and report changes in deaths at a given point in time. The typical approach uses a method that attributes air pollution-related health impacts to a single year air quality change (or “pulse”). The perspective on benefits from these static pulse analyses can be enhanced by conducting a dynamic population assessment using life tables. Such analyses can provide a richer characterization of health risks across a population over a multiyear time horizon. In this article, we use the life table approach to quantify cumulative counts of reductions in PM-attributable deaths and life-years gained due to overlapping impacts of PM<sub>2.5</sub> changes over a multiyear period, using case studies of air quality improvements in the USA and Chile. Our comparison of health risk and economic valuation for the two approaches shows life table analysis can be a valuable adjunct analysis to the pulse approach though both come with their own set of uncertainties and limitations. If applied jointly, they provide a broader characterization of how air quality actions can change populations in terms of life-years lost, life expectancy, and age structure. The value of these metrics is illustrated using case studies with dramatically different air quality reduction trajectories.

## 1. Introduction

A substantial body of literature spanning decades supports the mortality-related benefits of reductions in fine particulate matter (PM<sub>2.5</sub>) in ambient air, including over 25 years of cohort-based epidemiological studies (U.S. EPA, 2019; Pope *et al.*, 2020). Health impact assessments of PM<sub>2.5</sub> air pollution rely on this literature to quantify either reduced or excess cases of PM-attributable death among a population at a given point in time. These calculations support health burden analyses of existing PM<sub>2.5</sub> concentrations or the retrospective or prospective evaluation of the benefits of policies to reduce fine particles. The U.S.

Environmental Protection Agency (U.S. EPA) and other analysts typically quantify these impacts in regulatory analyses using a “pulse” method that attributes reduced (or incurred) air pollution-related health impacts to an air quality change (or “pulse”) that is modeled or measured in a single year. This is an efficient strategy for complying with established government guidance for comparing costs and benefits, particularly since the computational requirements of analysis of air rules at fine spatial scales required (e.g., 12 km photochemical modeling simulations) generally constrain the number of years that can be evaluated. Pulse analyses inform the policymaking process by providing a contemporaneous comparison of annual costs and benefits expected at critical points in policy implementation.

While the pulse approach is useful for providing snapshots of the benefits resulting from PM<sub>2.5</sub> reductions, it likely underestimates cumulative impacts of these reductions on survival. First, by using static snapshots in time, analysts cannot describe the change in pollution-attributable risks in intervening years not selected for analysis. More importantly, however, these analyses assume that the impacts of each pulse are independent of one another. This approach ignores the long-term dynamic effect of death rates in one year altering the population-at-risk in future years. This assumption means the analysis cannot account fully for the influence of air pollution trends on the pool of individuals whose mortality risks may be affected by air pollution from one year to the next.

Alternatively, a dynamic approach that uses life-table risk assessment methods updates at-risk population estimates from year to year based on both mortality risk impacts from an individual year’s air quality changes and the delayed impacts of previous years’ changes that continue to ripple through the population. The consequent changes to the size and age distribution of the population-at-risk affect mortality estimates and hence air pollution impacts in future years of analysis, helping to estimate the cumulative impacts. This approach also provides advantages for calculating time streams of benefits or costs by estimating changes in life years and life expectancy – a more conceptually accurate depiction of the public health benefits of improved air quality. The stream of changes in life years can then be expressed as a net present value (NPV) of changes to the population survival curve. Further, the additional survival metrics generated by the life table approach enable analysts to undertake a richer comparison of the effects of different air pollution reduction scenarios.

In this article, we describe a variant on the dynamic approach using a life-table-based risk assessment tool called PopSim that quantifies PM<sub>2.5</sub>-related PM-attributable mortality and population effects using a dynamic population model. This tool contains information to support the analysis of policies in over 180 countries using readily available data and health impact functions. Our approach is not new – it is based on principles established in prior research, such as that of Miller and Hurley (2003) and Röösl *et al.* (2005), but it is an approach that is under-represented in the suite of air quality benefits tools available (Anenberg *et al.*, 2016), and its usefulness has yet to be thoroughly explored. The model was designed to track the effect of alternative assumptions about the mortality effects of PM<sub>2.5</sub> in the USA population over time. The tool incorporates detailed life table data for historical years, by age, gender, and cause of death as well as population projections for future years based on the U.S. Census Bureau’s data and models for the USA and countries worldwide. The U.S. EPA’s Science Advisory Board has supported the use of dynamic population modeling where practicable noting that, “it provides the most realistic available modeling of how, over time, changes in population risk lead to changes in the size and age distribution of the population, with consequent implications for estimated mortality impacts” (U.S. EPA, 2010).

We apply PopSim to two historical datasets: (i) a time series of observed and predicted annual mean PM<sub>2.5</sub> concentrations in the contiguous USA showing a steady decline, and

(ii) a time series of historical monitored PM<sub>2.5</sub> concentrations for a city (Santiago, Chile) with more rapid and dramatic improvements in air quality over a similar period. These case studies allow us to answer three questions:

- (i) What is the estimated long-term change in PM<sub>2.5</sub>-attributable counts of death, life expectancy and life years in each location due to these air quality changes?
- (ii) How can we best compare life table estimated values to a traditional “static” approach? What does each approach contribute to benefits analysis and under what circumstances might each be appropriate?
- (iii) What is the Present Value of the long-term stream of mortality-related benefits using this new approach?

## 2. Methods

This analysis applies U.S. EPA’s PopSim Model to estimate the cumulative impacts of historic PM<sub>2.5</sub> changes from 1990 to 2015 in the USA and in Santiago, Chile. The model is available in two versions: a domestic version that is included as part of U.S. EPA’s Benefits Mapping and Analysis Program (BenMAP-CE) tool, and an international version that is available for download separately. Additional detail on PopSim can be found in the BenMAP-CE user manual and online at <https://epa.gov/benmap>.

### 2.1 *PopSim model overview and structure*

The PopSim tool builds on the life table method described in Miller and Hurley (2003). In brief, the model allows users to:

- (i) Simulate population in the USA by single year of age and gender for years between 1990 and 2060 under alternative assumptions about the degree of hazard posed by air pollution relative to baseline historical and projected Census mortality rates;
- (ii) Estimate changes in life years relative to a fixed baseline of historical and projected Census mortality rates;
- (iii) Apply air pollution hazards specific to cause of death and age category; and
- (iv) Analyze the effect of alternative lag structures on the timing of mortality rate changes following changes in PM exposure (“cessation lag”).

The model includes a library of concentration-response functions for PM<sub>2.5</sub>-related mortality, or users can specify their own. Users specify a set of national population-weighted average PM changes at up to five specific points in the study period and can specify the trajectory of PM changes between these points as either a step function or through linear interpolation between target years. If desired, users can also incorporate a PM<sub>2.5</sub> threshold concentration and explore the impacts of varying susceptibility to air pollution by age.

By default, all calculations and results in the model are conducted at the national level, using population-weighted average changes in PM. However, if provided with appropriately scaled life-table and air quality inputs, the model can be applied at alternative spatial scales, as we do in this study for the city of Santiago, Chile. The USA model can be used to estimate changes in mortality risk for years between 1990 and 2060 (1990 and 2050 for the international model). The temporal range provides a “run-up” period using the more highly resolved by-cause mortality data available for historical years and allows for testing of hypotheses on a retrospective and prospective basis.

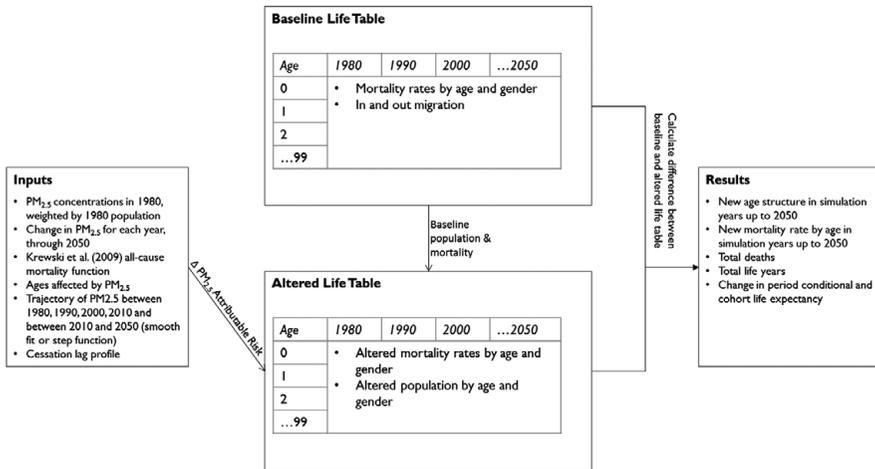


Figure 1. Conceptual framework for PopSim model.

The model consists of four linked components, as illustrated in Figure 1: Inputs (which includes the Hazard Estimation module); Baseline Life Table; Regulatory Life Table; and Results. The hazard estimation component of the model is integrated into the Inputs module. It applies user-specified parameters to calculate a mortality hazard adjustment factor (MHAF) that scales the baseline mortality hazard in a given year based on cumulative impacts across all previous years in the study period, according to the following equation:

$$MHAF_{j,t} = 1 + \left[ \sum_{i=BaseYear}^t [(e^{\beta \cdot \Delta PM_i} - 1) * LAF_{t-i+1} * TAF_i] * ASAF_j \right], \quad (1)$$

where  $MHAF_{j,t}$  is the Mortality Hazard Adjustment Factor for age group  $j$  in year  $t$ ;  $\beta$  is the coefficient from epidemiologic study or user-specified (% change in mortality per  $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub>);  $\Delta PM_i$  is the change in PM<sub>2.5</sub> concentration for year  $i$  ( $\mu\text{g}/\text{m}^3$ );  $LAF_{t-i+1}$  is a Lag Adjustment Factor in year  $t$  for total PM mortality impact from year  $i$ ;  $TAF_i$  is a Threshold Adjustment Factor for year  $i$ ; and  $ASAF_j$  is an Age-Specific Adjustment Factor for age group  $j$ .

To accommodate dynamic year-to-year variation in PM<sub>2.5</sub> concentrations, the MHAF for each calendar year is calculated as a weighted average of the mortality impact of PM changes associated in that year and all previously modeled years, with weights determined by the cessation lag structure chosen by the user. The weighted average effect estimate of the change in mortality, therefore, incorporates the effects of multiple, overlapping impacts for changes in annual average PM<sub>2.5</sub> prior to that year. The MHAF for each calendar year is calculated for each year of age in the cohort and is modified to reflect any additional adjustment factors specified by the user.

The lag between changes in PM<sub>2.5</sub> exposure and the full realization of the expected changes in mortality rates is modeled by applying a lag adjustment factor (LAF) to the relative risk (RR) calculated in each year. The LAF for a given year,  $t_n$ , represents the fraction of the total change in mortality associated with a PM<sub>2.5</sub> change in year  $t_1$  that is expected to be realized by year  $t_n$ . The threshold adjustment factor (TAF) enables the model to incorporate a threshold value for the mortality impacts of PM<sub>2.5</sub> concentration such that only changes

above the specified threshold would result in a change in mortality. The Age-Specific Adjustment Factor (ASAF) allows users to reflect on differences in susceptibility to  $PM_{2.5}$  across the population. The user can specify values for an ASAF between 0 and 2 for a specific user-defined age group, with a value of 1 meaning that the age group experiences the full mortality impact of the change in  $PM_{2.5}$ . Values greater than 1 indicate that an age group is more affected by changes in PM than the rest of the population and values less than 1 indicate that an age group is less affected by changes in PM.

The Baseline Life Table component represents historical population data and projection data for the USA and Santiago, Chile for the years 1990–2050. The data for Chile, as with the other countries in the International version of PopSim, is based on the Census International Database (IDB) historical population data and projection for the years 1990–2050 (U.S. Department of the Census. 2016). This database contains population data stratified by sex and year of age; mortality rates for each relevant cause, by age cohort; and natality (birth) rates by age.

For each country, the tool also includes estimates of net migration in and out of each country. For the USA, we developed net migration estimates by subtracting estimated deaths calculated using CDC mortality data from annual cohort population change reported in U.S. Census-based population estimates. For Chile, the tool includes estimates of net migration that we developed by subtracting estimated deaths calculated using historical mortality rates from the Global Burden of Disease study (2013), as estimated by Abubakar *et al.* (2015) and projected IDB mortality rates from the projected population. The data in this module are protected to preserve a static baseline from which gains and losses in attributable deaths, life years, and life expectancy are measured.

The Regulatory Life Table component begins with the same data contained in the Baseline component, and then applies adjustment factors (from the Hazards component) to the baseline mortality rates taken from the CDC data. As a result, the population simulation in the Regulatory component reflects the mortality rates implied by user's scenario specifications (entered in the Inputs component). The calculations in the Regulatory and Baseline components are equivalent, with the exception of the mortality rate, which is calculated as follows:

$$P_{regulatory}(death) = P_{baseline}(death) * MHAF. \quad (2)$$

## 2.2 Application of the PopSim model

We used PopSim to explore the effect of historical PM changes on the USA population aged 0–99 from 1990 to 2015 resulting from the application of air quality policies including but not limited to the U.S. Clean Air Act (U.S. Policy scenario), and the effect of air quality management actions taken by Chile over a similar period to reduce  $PM_{2.5}$  exposures to the population of Santiago, Chile aged 0–99 (Santiago Policy scenario). For the USA, we applied the domestic version of PopSim that accompanies the BenMAP-CE tool, version 1.5.0.4. For the Santiago analysis, we modified the International version of PopSim to estimate the Santiago city population from the country-level data in the PopSim tool. We scaled the Chilean population based on historic data on the proportion of the Chilean population residing in Santiago, which was relatively stable over the study period (Departamento de Demografía). Birth rates are held constant in both models. Specific model inputs for each PopSim run are presented in the Supplementary Material.

We applied the Krewski *et al.* (2009) PM concentration-response (C-R) function for all-cause mortality from BenMAP-CE, which estimates a roughly 6 % increase in mortality

associated with a  $10 \mu\text{g m}^{-3}$  increase in annual mean  $\text{PM}_{2.5}$ . We coupled this function with a 20-year distributed cessation lag with no threshold or age-specific hazard adjustments. We also compared the results from the PopSim model for the USA to results derived using BenMAP-CE (U.S. EPA, 2020), where the latter represent a standard static, pulse-type of analysis.

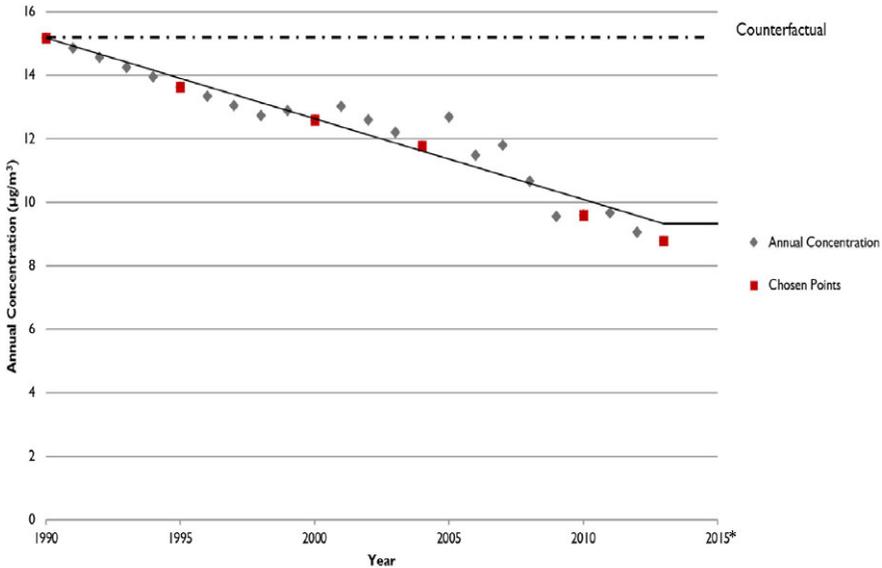
As noted above, the baseline scenario in the PopSim models is not modifiable by the user, and since our analysis is backward-looking, we assume that PopSim's Baseline population data already reflect the air quality changes we are analyzing. We thus designed a counterfactual scenario for both simulations in which  $\text{PM}_{2.5}$  concentrations and thus the fraction of  $\text{PM}_{2.5}$ -attributable mortality were assumed to be constant over the analysis period, with the difference between the two scenarios representing the impact of the USA or Chilean policy implementation.

For both countries, we estimated the historical average annual population-weighted  $\text{PM}_{2.5}$  concentrations for each year where data were available and used these data points to establish a  $\text{PM}_{2.5}$  concentration trend over time. PopSim currently only allows for five specified changes in  $\text{PM}_{2.5}$  over the study period; therefore, after developing these trend lines, we chose 6 years each for the USA and Santiago that best capture that location's trend in  $\text{PM}_{2.5}$  concentrations from 1990 forward. We assume that concentrations follow a linear trend from one chosen point to the next.

For the U.S. PopSim analysis, we used a combined dataset of modeled and monitored data to estimate average annual concentrations for each county in the USA. For the years 1990–2000, we used  $\text{PM}_{2.5}$  concentrations from research associated with the Multi-Ethnic Study of Atherosclerosis (MESA) study (Kim *et al.*, 2017) that used spatio-temporal modeling to estimate measurements of PM prior to the adoption of widespread  $\text{PM}_{2.5}$  monitoring in 1999. For the later period of the analysis after 2001, we relied on monitored PM concentration data from EPA's Air Quality System (AQS) that were pre-loaded in BenMAP-CE to calculate average annual concentration for each county in the USA. To generate a single average annual population-weighted concentration for the USA for each year, we merged this county-level  $\text{PM}_{2.5}$  concentration with county-level data for populations aged 30 and up. We obtained all population data from the U.S. Census, using Census data for 2010 and intercensal data for every other year (U.S. Department of the Census, 2018). Figure 2 demonstrates the roughly linear trend of average annual  $\text{PM}_{2.5}$  concentration in the USA from 1990 to 2013 ( $R^2 = 0.93$ ). The chosen data points for input to PopSim were for 1995, 2000, 2004, 2010, and 2013. Table 1 below summarizes the years chosen, the concentrations in each year and changes in  $\text{PM}_{2.5}$  concentration from the previous year in the table (labeled "Delta"). 1990 is included as the first year in the time period.

In the Santiago analysis, we obtained 5-year population estimates from the Chilean National Institute of Statistics (INE) and used these projections to generate Comuna-level population by age and sex from 1990 to 2015 in the Santiago Metropolitan Region (Departamento de Demografía, 2021).<sup>1</sup> Population-weighting was performed as in the USA analysis. For Santiago, we apply average annual  $\text{PM}_{2.5}$  concentrations for each of 11 monitors using data obtained from the Chilean Ministry of Environment. We population-weight these data using data for the given year in the Comuna where each monitor is located. In Figure 3 below, we plotted the average annual population-weighted concentration for the

<sup>1</sup> A comuna is a subdivision of Chile, similar to a U.S. county; Santiago consists of 32 separate comunas.



\* Difference in AQ in 2015 assumed to persist through the end of the study period (2050).

**Figure 2.** USA average annual PM<sub>2.5</sub> concentration, population weighted.

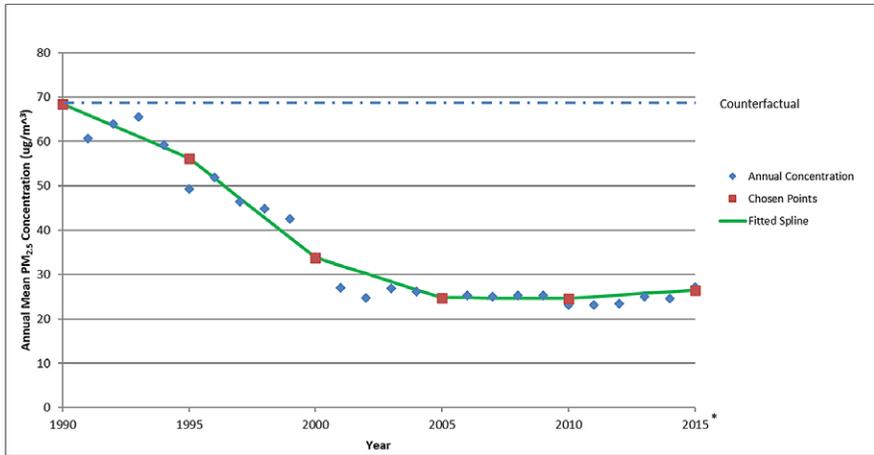
**Table 1.** PM<sub>2.5</sub> concentration inputs for U.S. PopSim model.

Year	PM <sub>2.5</sub> concentration (µg/m <sup>3</sup> )	Delta
1990	15.2	
1995	13.6	1.5
2000	12.6	1.1
2004	11.8	0.8
2010	9.6	2.2
2013	8.8	0.8

Santiago Metropolitan region across all monitors. We fit the concentration data over time using a spline model. The spline model has an  $R^2$  of 0.97. From this trend line, we chose 1995, 2000, 2005, 2010, and 2014 for our analysis as shown in Table 2. For both case studies, the air quality change in 2015 is assumed to persist unchanged for the remainder of the study period.

Because it calculates changes in mortality for each year and for each single year of age, PopSim can quantify and report multiple metrics to describe the survival curve shifts and corresponding lifespan changes resulting from changes in PM<sub>2.5</sub>. These metrics include the estimated changes in the numbers of deaths annually, the number of life years, and changes to life expectancy by age compared to baseline for the USA and Santiago case studies.

We estimate the economic value of projected mortality changes based on recommendations in federal guidance for economic analyses (U.S. EPA, 2014). Such economic analyses consider the marginal rate of substitution of wealth for mortality risk, or in simpler terms, the



\* Difference in AQ in 2015 assumed to persist through the end of the study period (2050).

Figure 3. Santiago average annual PM<sub>2.5</sub> concentration, population weighted.

Table 2. PM<sub>2.5</sub> concentration inputs for Santiago PopSim model.

Year	PM <sub>2.5</sub> concentration (µg/m <sup>3</sup> )	Delta
1990	68.4	
1995	56.2	12.2
2000	33.8	22.4
2005	24.7	9.1
2010	24.5	0.2
2014	26.4	-1.9

amounts that individuals are willing to pay for small reductions in mortality risk (“Value of Statistical Life [VSL]”) and the changes in VSL over time as real income grows (“income elasticity” of willingness to pay). The VSL represents a standardized unit, normalized to represent the amount that a population of size  $X$  is willing to trade to avoid a risk of  $1/X$  (for small changes in risk). To estimate VSL values for each year of the USA analysis, we follow the approach applied by Achakulwisut *et al.* (2019) to generate historical and projected VSL values in 2015 dollars from an assumed base VSL of \$10.0 million for 2015 and an income elasticity of 0.4 consistent with the BenMAP-CE default (U.S. EPA, 2020). For Santiago, we apply this method using a VSL based on a locally conducted stated preference survey that estimated a value of \$1.09 M (2015\$) (Greenlab UC, 2014). For changes in the risk of attributable deaths, we apply VSL estimates; for changes in life years, we derive a unit value per statistical life year (VSLY) by dividing the VSL by the discounted number of expected remaining life years among the exposed group, following the method used in Robinson *et al.* (2019). This method generates a constant VSLY for each year based on a year-specific VSL, and year-specific difference between the central estimate of the median age of the population and the average life expectancy for each country from the UN World Population Prospects

(2019) and World Bank World Development Indicators (2021), respectively. For the Santiago analysis, we use a combination of historical population and GDP growth data, OECD growth projections for 2021–2022, and a GDP growth rate of 3 % for 2023 and beyond based on a 10-year pre-pandemic average of the historical data (Organisation for Economic Co-operation and Development, 2020). We estimate the NPV of the reduction of PM-attributable mortality and life-years gained benefits by discounting the stream of annual monetized values to generate a metric suitable for comparing benefits and costs. We generate two NPV estimates: one using a 3 % discount rate and one using a 7 % rate.

We also conducted a sensitivity analysis of the PopSim results focused on the impact of alternative cessation lag models on results. We apply three models from work by Walton (n.d.) that vary the timing and speed of risk adjustments to the USA and Santiago runs. Model A presents a steep and rapid realization of the risk reduction over 8 years; Model C presents a more gradual realization over 15 years, and the last is a steady incremental accrual over 30 years.

### 3. Results

Tables 3–8 below provide the output from the PopSim model for the USA and Santiago runs, including changes in PM-attributable deaths per year, life-years gained, and life expectancy, as well as the value of these benefits (calculated separately). Tables 3 and 4 provide the estimated change in number of deaths per year by age cohort for the simulation period 1990–2050. Positive values represent fewer deaths for that age group in the given year; negative values represent an increased number of deaths. The estimates presented are for a single year, but as discussed reflect the summed impact of current and lagged mortality risk impacts from previous years' improvements.

For the USA case, we see substantial reductions in deaths overall and in most age categories, with the number of PM-attributable deaths reduced growing larger for most age categories until somewhere between 2020 and 2030. However, in some cases, the results are negative (increased deaths), which may appear counterintuitive. Not surprisingly, initially, all age groups experience fewer deaths in the cleaner U.S. Policy scenario, and the result of this change is that more individuals are alive to survive to older age bins that have higher baseline mortality rates. As a result, the oldest age category begins to quickly experience more deaths under the U.S. Control scenario, and the number of additional deaths grows in this group cohort over time. This phenomenon is only seen in the oldest cohort – in all other cohorts, there are fewer deaths in the U.S. Policy scenario, though eventually, the countervailing effect of population shifts begins to erode the total size of these benefits.

The NPV of these mortality risk reductions is also substantial, with individual-year “pulse” style benefits topping out between \$91 and \$201 billion in the decade between 2010 and 2020 due to the added impacts of discounting on the monetized values. The cumulative life-table estimated benefits are considerably larger, continuing to increase throughout the study period and reaching a total of between \$3 and \$8 trillion when improvements across all years are taken into account.

For Santiago, the results in Table 4 show consistent and substantial reductions in PM-attributable deaths in all except the oldest age groups, with smaller overall values reflecting the smaller geographic scale (and population) of the Santiago analysis. The tapering of mortality-related benefits over time is less pronounced in these results, with most categories peaking or nearing their peak in the last decade of the analysis. This delay in peak impact is

**Table 3.** *Estimated attributable deaths avoided and valuation (\$2015, millions) for U.S. PM<sub>2.5</sub> air quality improvements 1990–2013.*

Age categories	1990	2000	2010	2020	2030	2040	2050
<b>Deaths</b>							
30–39	0	638	1,282	1,666	1,514	1,162	894
40–49	0	1,340	2,929	3,062	2,878	2,543	1,970
50–59	0	2,088	6,201	7,449	5,981	5,506	4,972
60–69	0	3,241	8,606	13,413	12,522	10,204	9,963
70–79	0	5,613	10,344	17,155	21,485	20,597	18,469
80–89	0	5,024	9,350	10,955	13,384	16,312	18,504
90–99	0	779	–2,532	–8,067	–14,254	–21,979	–27,863
<b>Total avoided</b>	0	18,723	36,180	45,633	43,510	34,345	26,909
<b>Valuation</b>							
NPV, 3 %DR, single-year	0	\$140,000	\$201,000	\$201,000	\$150,000	\$95,000	\$59,000
NPV, 3 %DR, cumulative	0	\$760,000	\$2,400,000	\$4,600,000	\$6,200,000	\$7,400,000	\$8,100,000
NPV, 7 %DR, single-year		\$90,000	\$91,000	\$62,000	\$32,000	\$14,000	\$5,800
NPV, 7 %DR, cumulative		\$581,000	\$1,500,000	\$2,300,000	\$2,700,000	\$3,000,000	\$3,000,000

Abbreviations: DR, discount rate; NPV, net present value.

**Table 4.** *Estimated attributable deaths avoided and valuation (\$2015, millions) for Santiago, Chile PM<sub>2.5</sub> air quality improvements 1990–2015.*

Age	1990	2000	2010	2020	2030	2040	2050
<b>Deaths</b>							
30–39	0	167	248	322	396	413	496
40–49	0	245	493	532	722	909	907
50–59	0	401	814	1,240	1,390	1,907	2,291
60–69	0	618	1,228	1,937	2,996	3,344	4,316
70–79	0	853	1,428	2,076	3,264	4,679	4,557
80–89	0	696	816	554	368	101	–1,386
90–99	0	35	–295	–937	–1,620	–2,654	–4,136
Total avoided deaths	0	3,015	4,732	5,724	7,516	8,699	7,045
<b>Valuation</b>							
NPV, 3 %DR, single-year	0	\$2,100	\$2,700	\$2,600	\$2,700	\$2,500	\$1,600
NPV, 3 %DR, cumulative	0	\$9,400	\$36,000	\$62,000	\$88,000	\$110,000	\$140,000
NPV, 7 %DR, single-year	0	\$1,400	\$1,300	\$810	\$590	\$370	\$170
NPV, 7 %DR, cumulative	0	\$7,100	\$22,000	\$32,000	\$38,000	\$43,000	\$46,000

Abbreviations: DR, discount rate; NPV, net present value.

mainly due to the more dramatic initial decline in PM concentrations in Santiago versus the USA. The negative deaths (effectively more deaths) in the oldest cohort, and for the age 80–89 cohort in 2050, reflect that the pollution control scenario again allows the population to live longer lives, resulting in a shifting of mortality to older age groups. The net effect across all cohorts, though, remains positive throughout the simulation, even with a slight increase in PM concentrations in 2014.

Valuation of the Santiago benefits also shows some evidence of an early peak in “pulse” style valuations, particularly when applying the 7 % discount rate, peaking in 2000 at \$1.4 billion; this peak is delayed to 2030 when using the lower discount rate, but the pulse values quickly approach the peak and remain steady within 10–15 % of \$2.7 billion for much of the study period. This pattern most likely reflects the dramatic early decline in PM concentrations followed by declines in later years that are a combination of the lag from the large initial benefits and benefits of smaller reductions in later years. The cumulative benefit estimates again are an order of magnitude or two greater, continuing to increase over the course of the study period to a total of \$46–\$140 billion, depending on the discount rate.

Tables 5 and 6 illustrate a second output from the PopSim model, estimated life-years gained by age group and year of the simulation. These estimates effectively compare the number of individuals in each age group in the two simulations; in other words, each additional individual in a group represents an additional life-year lived for that group. In the USA case, life-years gained from air quality management efforts are positive for all groups in all years; that is, populations in each age group are larger in the USA policy scenario than in the Baseline scenario. The same is true for Santiago, with the number of life years increasing substantially as the population shifts toward an older age structure, all else equal. In the USA, air quality management results in 1.5 million additional life years in 2050 and over 5 million additional life years lived in the USA population summed across the 6 years shown in

**Table 5.** *Estimated life-years gained and valuation (\$2015, millions) for U.S. PM<sub>2.5</sub> air quality improvements 1990–2013.*

Age categories	1990	2000	2010	2020	2030	2040	2050
<b>Life years gained</b>							
30–39	0	1,764	4,018	6,959	6,758	5,199	3,966
40–49	0	4,556	16,076	24,646	27,828	25,415	19,507
50–59	0	6,896	34,833	66,729	67,548	67,513	60,641
60–69	0	11,253	51,004	132,409	161,932	143,971	139,500
70–79	0	21,409	69,982	180,797	302,504	322,286	280,526
80–89	0	24,793	98,533	199,575	358,675	522,430	544,483
90–99	0	11,554	49,182	133,713	193,128	329,351	481,587
Total years gained	0	82,225	323,628	744,828	1,118,373	1,416,165	1,530,210
<b>Valuation</b>							
NPV, 3 %DR, single-year	0	\$26,000	\$77,000	\$140,000	\$170,000	\$170,000	\$140,000
NPV, 3 %DR, cumulative	0	\$92,000	\$620,000	\$1,700,000	\$3,200,000	\$4,900,000	\$6,400,000
NPV, 7 %DR, single-year	0	\$30,000	\$62,000	\$77,000	\$62,000	\$43,000	\$25,000
NPV, 7 %DR, cumulative	0	\$110,000	\$580,000	\$1,300,000	\$1,900,000	\$2,400,000	\$2,700,000

Abbreviations: DR, discount rate; NPV, net present value.

**Table 6.** Estimated life-years gained and valuation (\$2015, millions) for Santiago, Chile PM<sub>2.5</sub> air quality improvements 1990–2015.

Age categories	1990	2000	2010	2020	2030	2040	2050
Life years gained							
30–39	0	378	978	1,242	1,604	1,617	1,988
40–49	0	702	3,442	4,481	5,945	7,501	7,650
50–59	0	1,134	5,546	11,149	13,094	17,659	22,094
60–69	0	1,807	8,853	18,149	30,897	35,290	47,243
70–79	0	2,670	12,391	25,211	44,292	70,601	77,485
80–89	0	2,864	14,232	26,156	43,766	70,851	101,357
90–99	0	566	4,743	11,582	17,813	27,308	42,109
Total life years	0	10,121	50,185	97,970	157,411	230,827	299,926
Valuation							
NPV, 3 %DR, single-year	0	\$270	\$1,200	\$1,800	\$2,400	\$3,000	\$3,300
NPV, 3 %DR, cumulative	0	\$870	\$8,200	\$23,000	\$44,000	\$71,000	\$102,000
NPV, 7 %DR, single-year	0	\$340	\$980	\$1,000	\$920	\$760	\$550
NPV, 7 %DR, cumulative	0	\$1,100	\$8,100	\$18,000	\$27,000	\$35,000	\$41,000

Abbreviations: DR, discount rate; NPV, net present value.

**Table 5.** In the case of Santiago, air quality management results in 300,000 additional life years in 2050 and 850,000 additional life years in the Santiago population summed across the 6 years shown in [Table 6](#).

[Tables 7](#) and [8](#) provide estimates of the increase in life expectancy from the PopSim model, specifically period conditional life expectancy (PCLE). The PCLE metric is constructed using age-specific mortality rates for a single year, with no allowance for projected changes in mortality. PopSim also provides estimates of changes in cohort life expectancy, which represent the probability of a person in an age cohort (same year of birth) dying at each age throughout his or her lifetime, factoring in changes in age-specific mortality rates over time (Office for National Statistics, 2017). While all individuals will experience a future, unknown risk of mortality that unfolds through their lifetime, we focus on PCLE because it is the methodology generally reported by the CDC and, for past years, in particular, PCLE provides a measure that can be compared to CDC's estimates over time (Office for National Statistics, 2017). In the USA, effects on life expectancy are immediately experienced across all cohorts and grow rapidly to a gain of approximately 0.3 year for women as early as 2010 in the youngest cohorts and eventually reaching similar magnitude for the age 60 cohort by 2050. The gains for men start more slowly, at about 0.2 years additional in 2010, but grow to a slightly higher value than that for women by 2050. For Chile, in [Table 8](#), the gains are higher due to a much greater decline in average annual PM<sub>2.5</sub> concentration from 1990 to 2015 compared to the USA. The life expectancy gains for those aged 30 or 40 of both genders are as high as 2 years in 2050.

**Table 7.** Increase in period conditional life expectancy for USA population due to PM<sub>2.5</sub> air quality improvements 1990–2015.

Females							
Age categories	1990	2000	2010	2020	2030	2040	2050
30–39	0.00	0.11	0.31	0.33	0.34	0.33	0.31
40–49	0.00	0.10	0.29	0.32	0.33	0.32	0.30
50–59	0.00	0.10	0.27	0.30	0.30	0.30	0.29
60–69	0.00	0.08	0.23	0.26	0.28	0.27	0.27
70–79	0.00	0.07	0.18	0.22	0.23	0.23	0.23
80–89	0.00	0.05	0.12	0.17	0.17	0.17	0.18
90–99	0.00	0.02	0.03	0.06	0.06	0.06	0.05
Males							
Age categories	1990	2000	2010	2020	2030	2040	2050
30–39	0.00	0.12	0.21	0.36	0.36	0.35	0.33
40–49	0.00	0.11	0.20	0.34	0.35	0.34	0.32
50–59	0.00	0.10	0.18	0.31	0.32	0.31	0.31
60–69	0.00	0.08	0.16	0.27	0.28	0.28	0.27
70–79	0.00	0.07	0.12	0.21	0.22	0.22	0.23
80–89	0.00	0.05	0.08	0.14	0.15	0.15	0.16
90–99	0.00	0.02	0.03	0.06	0.07	0.07	0.07

**Table 8.** Increase in period conditional life expectancy for Santiago population due to Santiago, Chile PM<sub>2.5</sub> air quality improvements 1990–2015.

Females							
Age categories	1990	2000	2010	2020	2030	2040	2050
30–39	0.00	1.24	2.05	2.13	2.09	2.03	1.99
40–49	0.00	1.20	1.98	2.06	2.00	1.93	1.88
50–59	0.00	1.13	1.87	1.92	1.86	1.77	1.69
60–69	0.00	1.00	1.68	1.72	1.63	1.51	1.41
70–79	0.00	0.85	1.43	1.45	1.35	1.20	1.08
80–89	0.00	0.62	1.05	1.09	1.05	0.97	0.89
90–99	0.00	0.19	0.32	0.36	0.38	0.41	0.42
Males							
Age categories	1990	2000	2010	2020	2030	2040	2050
30–39	0.00	1.36	2.25	2.32	2.27	2.22	2.19
40–49	0.00	1.28	2.12	2.18	2.11	2.03	1.98
50–59	0.00	1.17	1.94	1.99	1.90	1.79	1.72
60–69	0.00	1.01	1.69	1.71	1.59	1.46	1.36
70–79	0.00	0.82	1.38	1.38	1.25	1.09	0.97
80–89	0.00	0.61	1.03	1.06	0.99	0.89	0.80
90–99	0.00	0.21	0.35	0.38	0.40	0.42	0.42

It is challenging to compare these PopSim results against BenMAP estimates using the pulse approach, because as Table 9 shows, the tools have numerous differences that render the conclusions of any comparison partial at best. Figure 4 compares the PopSim results for changes in PM-attributable mortality in the USA example against a small set of BenMAP runs for individual years, where we have corrected for some of these differences by applying the standard U.S. EPA 20-year cessation lag model to BenMAP and then stacking the resulting time series of benefits across the three runs. Compared to any individual year's pulse results, the PopSim model's cumulative benefits appear substantially greater – nearly a factor of two compared to the first year's impacts from PM changes in 2013. However, when summed with lagged pulse results from previous years, the figure suggests that summing the results of multiple years of BenMAP-CE analyses should begin to approximate the PopSim estimate.

These differences also need to be understood in the context of the projected demographic changes in the population during the study period. Figure 5 shows the predicted growth in key relevant population segments for PM-attributable mortality and in the mortality rate for those subpopulations. From 1990 to 2050, the USA population aged 65 and older is predicted to increase dramatically, expanding 1.5 times by 2030 and nearly doubling by 2050, while mortality rates exhibit more modest declines. The population trends in BenMAP are likely similar but may not match exactly, since BenMAP bases its population growth estimates on projection data from Woods and Poole that supports finer scale estimations of population growth than the national-scale Census estimates used in PopSim.

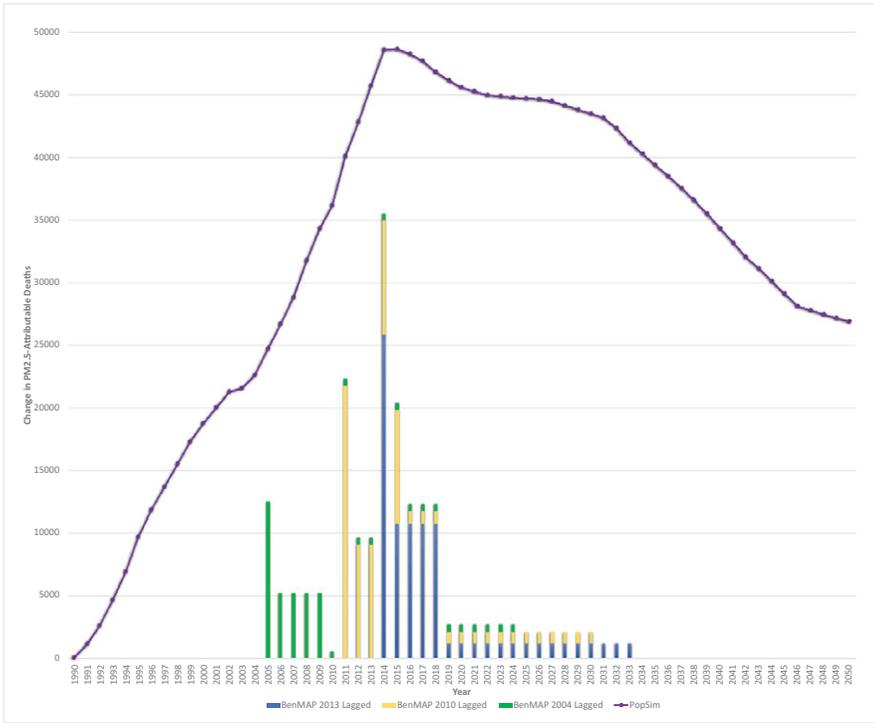
Figure 4 also illustrates that the number of reduced PM-attributable deaths are not monotonically increasing over time, but peak and begin to decline in the USA starting

**Table 9.** A comparison of BenMAP and PopSim tools.

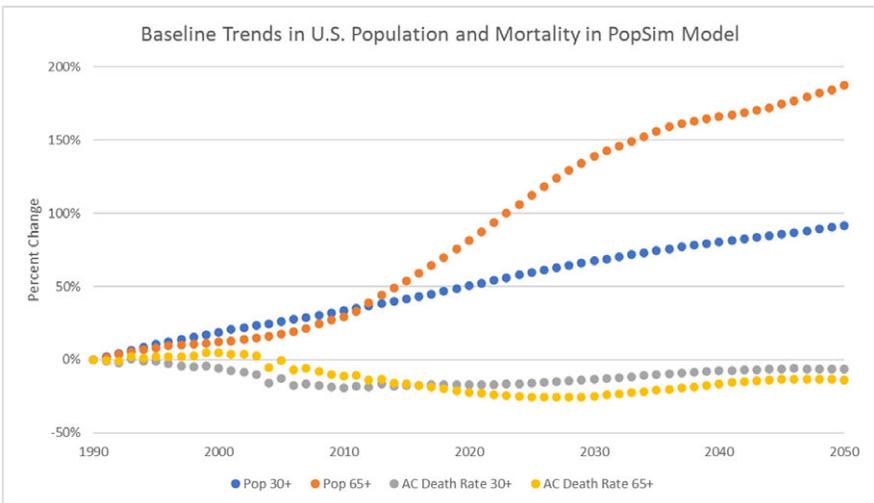
	BenMAP-CE	PopSim
Risk assessment method	Pulse	Life-Table
Number of AQ changes assessed per run	One	Up to five
Time span per run	Single year	Multiple years, up to 1990–2050
Geographic resolution	Accommodates air quality and other spatial data at various scales (e.g., 12 km cells)	Single spatial scale; uses national population-weighted AQ by default, other scales require user supplied data.
Includes cessation lag?	Not built-in	Yes
Allows alternative cessation lag models?	No	Yes
Accounts for population projections	Yes	Yes
Accounts for mortality projection	Yes	Yes
Updates population estimates for past AQ changes?	No	Yes
Results for a given year reflect...	Total impact of air quality changes in that year	Impacts of current and past AQ changes in study period

about 2015, reflecting the eventually increasing numbers of deaths observed in the oldest age groups in Tables 3 and 4. Figure 6 shows that the reduction in PM-attributable mortalities in the Santiago case study peak much later, reaching a high of 8,700 PM-attributable deaths reduced in 2040. Figure 7 compares the trajectory for the two cases for life years gained; the peak of the life years metric lags that of PM-attributable mortalities, with the USA reaching its peak in the last decade of the analysis and Santiago life years continuing to increase through the end of the study period. Figure 7 also shows the effect of these additional life years on the per cent of individuals aged 65 and older, with a 1.6 % point increase in this fraction of the population in the Santiago case.

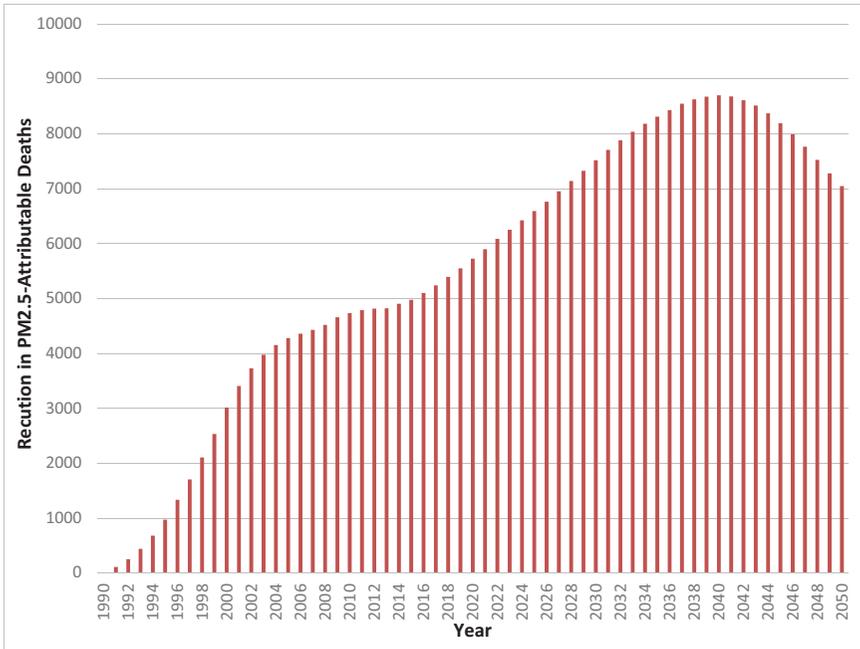
Figure 8 illustrates the sensitivity of the PopSim results to alternative cessation lag models on results. Not surprisingly, the steeper models result in more jagged, earlier peaks in attributable death reduction coupled with corresponding earlier and steeper declines as deaths in benefits begin to increase earlier in the older populations, as compared with the standard U.S. EPA lag, shown in gray. The resulting NPV estimates of models A and C are modestly different from the main model. However, the more gradual and lengthier models provide more interesting results. They smooth out the effect seen in Model A and shift the peak later, as expected, but they also eventually result in peaks as large or in the case of Santiago, larger than those of the other models. This effect reflects the interaction of the lag



**Figure 4.** Conceptual comparison of change in attributable deaths using dynamic (*PopSim*) and static (*BenMAP*) approaches for USA case study, 1990–2050.



**Figure 5.** Baseline projections for population and mortality in *PopSim* model.



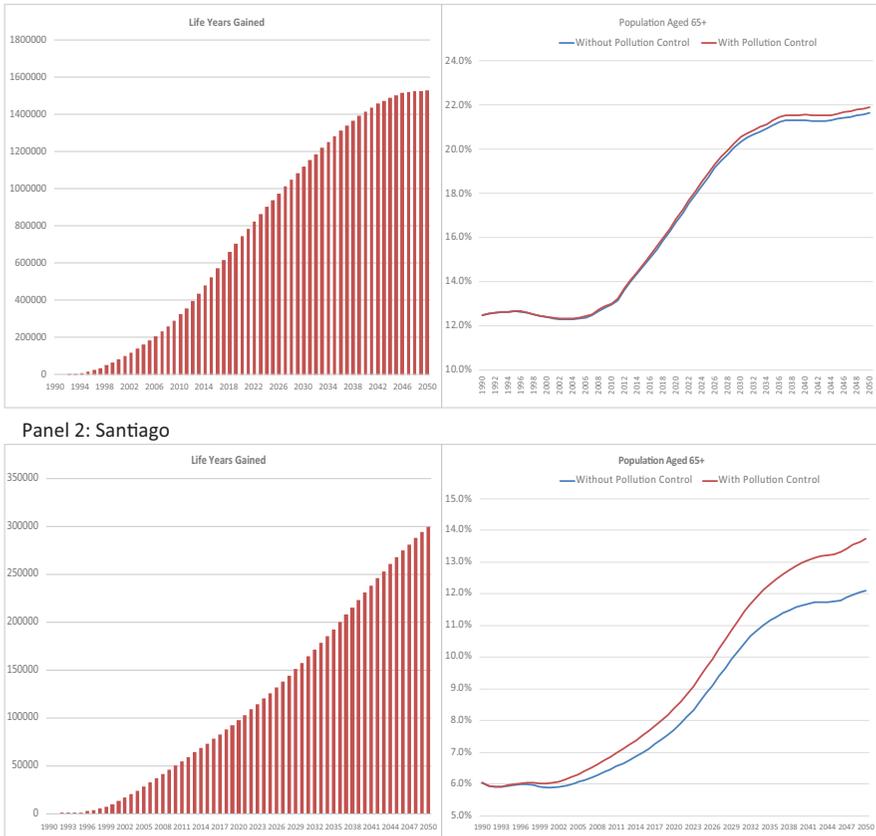
**Figure 6.** Reduction in PM<sub>2.5</sub>-attributable deaths using dynamic (PopSim) approach for Santiago, Chile, 1990–2050.

with changes in the population structure; the 30-year model delays the period of maximum risk reduction until the point where we see the maximum increase in the 65+ age category in each population. As observed in the NPV estimates for this model, this interaction mitigates the effect of discounting on the results for each model.

#### 4. Discussion

The application of life-table-based models such as PopSim is a useful complement to the traditional method of air quality benefits assessment, augmenting it in several key ways: it provides an efficient way to estimate on a national or regional scale the cumulative effects of reductions in mortality risks on the size and structure of an exposed population over time; produces alternative characterizations of the risk-reduction benefits of air quality management that can supplement the traditional attributable deaths metric; and enables the encapsulation of decades of public health benefits into a single estimate of NPV. The results of applying PopSim to these two case studies, in particular, demonstrate in each case a substantial cumulative effect of USA and Chilean air quality management on their respective populations and add insights into the life expectancy gains attributable to cleaner air.

The number of PM-attributable deaths estimated each year from PopSim is fundamentally and conceptually different from that estimated by BenMAP-CE. While BenMAP-CE estimates the number of PM-attributable deaths that will eventually be reduced as a result of a single pulse reduction in air pollutant exposure for a given year, the PopSim estimate for that same year estimates these as the sum of a series of processes capturing the overlapping

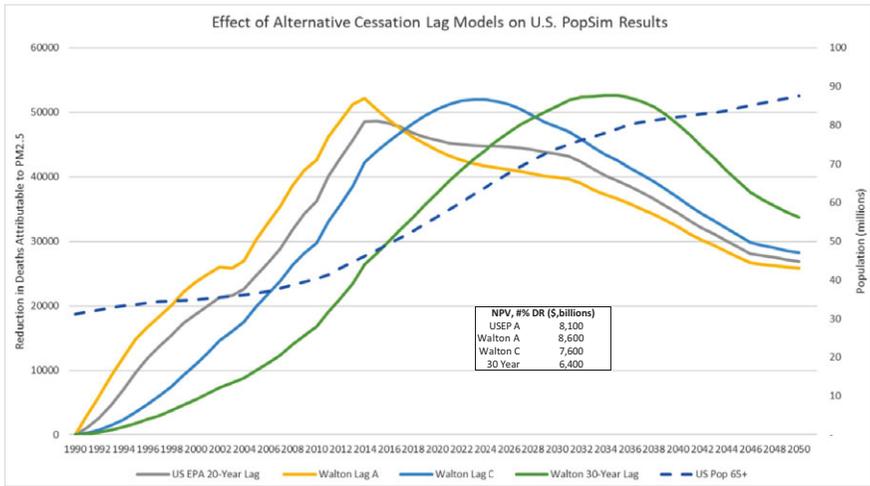


**Figure 7.** Comparison of life-years gained and population 65+ for USA and Santiago (Chile) applications.

lagged benefits of past exposure changes plus a portion of the current year's benefits applied to a population cohort whose age distribution, size, and mortality rates reflect the impacts of previous years. We can see from this approach that in the absence of other perturbations, a series of reductions in mortality rates from improved air quality will yield additional cumulative benefits resulting from changes to the underlying at-risk population over time that the pulse approach does not capture.

While the comparison of PopSim and BenMAP-CE results in Figure 4 suggests that one could roughly approximate PopSim by running BenMAP-CE for every year of analysis and summing those results, in reality, such an approach would not only be unwieldy, but would not capture the compounding effects of population changes that are automatically endogenized into the PopSim model. In the case of both the USA and Santiago analyses, this additional effect is likely to be substantial. The potential magnitude is easiest to explore for the USA case, where the PM concentrations decrease roughly linearly over the study period and thus could be approximately by a series of equally sized PM reductions of the average decrease ( $3.2 \mu\text{g}/\text{m}^3$ ) from 1990 to 2015. A back-of-the-envelope calculation using those changes, the Krewski *et al.* RR, and a static estimate of 2.5 million deaths per year suggests a change of about 1.2 million cumulative attributable deaths, while PopSim is reporting a

U.S.



Santiago

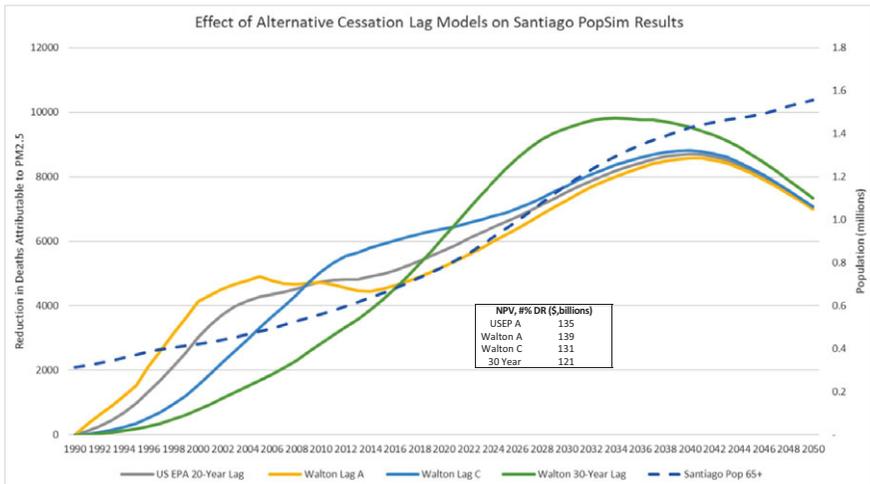


Figure 8. Sensitivity analysis for alternative cessation lag models.

change of about 2 million attributable deaths. This substantial gap is most likely due to the substantial projected increases in the population aged 65 and older, which far outweighs the projected baseline reduction in mortality rates for that group. This effect is also likely enhanced because the cessation lag model we applied focuses much of the mortality risk changes in the period of steepest population growth. Theoretically, we could account for this population change by conducting many BenMAP runs that incorporate that tool’s projection estimates and that will narrow the gap; however, those runs will not capture the compounding effect of these changes because it does not update its population estimates from year to year for past changes.

PopSim also illustrates how benefits from improved air quality will at some point peak and begin to decline as the age structure and mortality risk profile of the population changes, as shown in [Figure 4](#). To be clear, air quality regulations are not the cause of these additional deaths – it is merely that the life-extending benefits of breathing less polluted air yield higher numbers of individuals surviving to cohorts with high non-pollution-related mortality rates. As the population age structure shifts toward larger elderly populations, the presence of greater populations in age bins with higher mortality rates begins to outweigh the impact of the reduced mortality rates at lower ages. Of note, the number of reduced PM-attributable mortalities appears to peak sooner than the number of life years gained.

In [Figure 6](#) for Santiago, the number of annual reduced PM-attributable deaths does not peak until the penultimate decade of the analysis; the peak occurs sooner in the USA, with positive, though steadily declining values in much of the USA analysis. There are two primary reasons for this difference. First, the historical USA air quality changes exhibit more gradual, steady declines over the study period that follow a largely linear trajectory, while Santiago begins the study period with a dramatically sharp and large decline in  $PM_{2.5}$  that continues to reverberate through the population for much of the study period. Second, the age profiles of the two populations differ. Santiago's population is skewed toward younger ages at the start as shown in [Figure 7](#) so it takes longer to shift the population enough to overcome the substantial risk reduction benefits from those initial reductions.

Considerations of life-years gained estimates from the model provide additional context for understanding the effects of air quality on health, because it illustrates the magnitude of life extension that can be expected as mortality risk reductions play out against expected future demographic and health changes in the population. It also illustrates the effect of the trajectory of air quality changes on benefits. For example, examination of [Table 5](#) shows that the dynamic approach estimates more than 1.5 million life-years saved in the USA in 2050 due to the current air quality management regime compared to 300,000 additional life years expected in the Santiago case. Chile and Santiago's rapid and dramatic actions on air quality yielded 20 % of the USA scenario benefits, even though its population of adults 30–99 was less than 2 % of the corresponding USA population at the start of the analysis. This is due both to the larger magnitude of  $PM_{2.5}$  reductions, and to the fact that they were achieved quickly in the early years. As a result, much if not all of the lagged impact is captured in the study period and the resulting changes in the size and structure of the at-risk population early in the analysis contribute to larger benefits of later  $PM_{2.5}$  reductions. It also has a strong enough effect on the population to effectively carry through a strong lives-saved benefit to the end of the simulation, with life-years gained continuing to increase through 2050 and likely beyond. The change in population age structure is also both substantial and dramatically different in each case: the air quality actions are expected to lead to significant increases in the percentage of the population aged 65 and older – on the order of 0.3 % in the USA (1.4 million people) and 1.6 % in Santiago (about 260,000 people) above and beyond projected demographic trends in aging.

The estimation of changes in life expectancy by the PopSim tool provides yet another perspective on the public health benefits of air quality. Our USA results find that adults between 30 and 49 would experience an approximately 0.2–0.3 year gain in life expectancy from the changes in  $PM_{2.5}$  from 1990 to 2015 starting in 2020 and persisting through 2050. These results are generally consistent with or more conservative than estimates reported by other researchers (Correia *et al.*, 2013; Fann *et al.*, 2017; Schwartz *et al.*, 2018). Of note,

Schwartz *et al.* found an increase in life expectancy of 0.9 years comparing areas with annual mean  $\text{PM}_{2.5}$  of 12 and  $7.5 \mu\text{g}/\text{m}^3$  using causal modeling methods instead of life-tables.

Our estimates for the change in life expectancy in Santiago are in the vicinity of 2 years for adults 30–49, from 2010 to 2050. We have fewer points of comparison for estimates of this magnitude, but a study of China's Huai River policy by Chen *et al.* (2013) suggested air pollution concentrations in China may be reducing life expectancy in some areas by as much as 5 years. We note, however, that our analysis uses the Krewski *et al.* (2009) concentration-response function, which assumes an essentially linear response to the absolute amount of PM and may potentially bias our estimates upwards for Santiago, given recent work by Cohen *et al.* (2017) and Burnett *et al.* (2018) on nonlinear functions showing flatter slopes at higher PM concentrations. Interestingly, while it is typically stated that older cohorts are the main recipients of the benefits of cleaner air, we note risk changes across all categories result in younger adults seeing a larger life expectancy gain in absolute terms, although older adults would see a larger gain in terms of the percentage of their remaining life expectancy.

The comparison of monetized benefits using VSL and a constant VSLY illustrates that the monetized benefits of the life-years gained are modestly but consistently smaller than those generated using VSL, though still quite substantial (a cumulative \$2.7 to \$6.4 trillion in the USA in 2050 and \$47 to \$120 billion in Santiago for a 3 or 7 % discount rate, respectively). These values are about 80–95 % of the VSL-based valuation in the USA and Santiago. This reflects in part the fact that the PM-attributable deaths reduced skew toward older individuals who have fewer remaining life years than the general population. Additionally, we derive an estimate of VSLY from VSL – a standard approach (Robinson *et al.*, 2019), but necessary due to the lack of reliable directly elicited estimates of VSLY in the peer-reviewed literature. The VSLY method adopted here reflects the current standard but strong assumption valuation that a life-year is proportional to discounted remaining life years, though this pattern is not consistently observed in revealed (Aldy & Viscusi, 2007) and stated preference studies of VSL by age (see also Hammitt, 2013). Recently, Yin *et al.* (2021) has applied an age-adjusted measure of VSLY that reflects not only remaining life expectancy but quality of life in older age. That work acknowledges that the complex interplay of age-dependent health, longevity, and wealth, as well as the choice of a discount rate, complicates the estimation of age-specific VSLY; however, we note that no consensus has yet been reached on applying age-adjusted VSLY. The method demonstrated in Yin *et al.* (2021), could be combined with the dynamic life-expectancy calculation in PopSim (rather than the static life expectancy in Yin *et al.*), as a potentially interesting extension of our work that could provide some insights into potential uncertainties in VSLY estimates used with PopSim results.

The eventual peak and then gradual decline of benefits seen in the lives and life-years metrics is also reflected in the monetized benefit values using VSL and VSLY, but the peak is shifted earlier due to the effects of discounting the value of the health benefits, particularly when using a 7 % discount rate. Regardless, we still see in the Santiago example that it could take several decades, all else equal, for the benefits of substantial improvements in  $\text{PM}_{2.5}$  to peak if aggressive action is taken. This illustrates that life-table-based benefit estimates using PopSim or other tools represent an important supplement to the traditional VSL based monetized benefits found in many air pollution assessments, including U.S. EPA (2011).

There are several limitations to acknowledge related to the life-table approach generally and the PopSim tool specifically. The life-table approach enables us to estimate the change in PM-attributable deaths between two different air quality scenarios using standard damage assessment methods, survival curve data, and concentration-response relationships that are

proportional to baseline death rates. We also use these data to generate the best estimate of impacts on life-years gained in the population assuming that changes in attributable deaths are distributed proportionally to baseline death rates, a reasonable assumption consistent with the epidemiological literature. However, as explained in Hammitt *et al.* (2020), we cannot know with certainty from these data alone the specific distribution of attributable death differences across and within the various age categories and as a result, the effect on life-years gained may be larger or smaller than estimated by PopSim. With regard to PopSim, the estimates generated by PopSim are specific to the particular population projection used in that tool. As noted above, additional sensitivity analysis to understand the impacts of model uncertainty in these projections would be beneficial. Results are also sensitive to the specific cessation lag model applied, though as our results show, these impacts on the timing of risk reduction interact with the specific population projection and age-distribution assumptions, which can have either a mitigating or exacerbating effect on the NPV of benefits.

Finally, use of the life-table approach highlights the need to better elucidate variations, if any, in the effect of PM<sub>2.5</sub> on mortality by age. Mortality health impact functions used historically by EPA, such as the Krewski *et al.* (2009) function used in our analyses, employ a single impact estimate for a large age range (e.g., 30 and older). In recent years cohort studies have shifted toward using larger data sets of the 65 and older Medicare population (e.g., Di *et al.*, 2017), which can help shed some light on whether the mortality effect might vary with age. If age does modify this effect, then our results would be sensitive to these differences, because of the significant shifts in age distribution over time in the populations we studied. Future life-table analyses could be improved by exploring the use of age-specific mortality functions to account for effects of projected demographic changes on the health impacts of changes in PM<sub>2.5</sub> concentrations over time.

## 5. Further research

The dynamic life-table method described above is not meant to replace static estimates from BenMAP-CE, but rather provides a richer representation of the effects over time at the cohort level. We note that this additional information comes at the expense of geographic resolution, due to the increased computational complexity of the life table approach. BenMAP-CE allows changes in PM and the resulting mortality effects to be expressed at a finer spatial scale than currently allowed by PopSim. The spatial scale of BenMAP-CE is particularly important in benefits analysis where the location of an air quality improvement and whether the improvement occurs in a highly populated area can have a large effect. A key future improvement to PopSim would be to allow for finer spatial resolution within the simulation – for example, multistate regional estimation for the USA. A future version of the PopSim model could be configured to support Environmental Justice analyses by (i) incorporating race or ethnicity-specific baseline death rates; and (ii) using Hazard Ratios that account for effect modification of air pollution risk by race (Di *et al.*, 2017; Fann *et al.*, 2017; Rosofsky *et al.*, 2018).

In addition, our estimates for Santiago are limited by the lack of detailed population projections for the metropolitan area; our proof-of-concept analysis proves the PopSim model is capable of conducting analyses at this scale, but further evaluation of effects on the Santiago population should be informed by geographically specific, age-stratified projections of population reflecting local health and demographic trends.

The PopSim tool is currently programmed using widely available software to ensure it is accessible to most users. Improvements to the computational speed will be necessary to facilitate analyses at a finer geographic scale and accommodate treatment of uncertainty either at the concentration-response function level or at the scenario-level in terms of alternative population and health forecasts. Other potential improvements include accommodating the use of recent nonlinear concentration-response functions, such as the Integrated Exposure Response function (Cohen *et al.*, 2017) and the Global Exposure Mortality Model (Burnett *et al.*, 2018), which may be more appropriate for high concentration scenarios such as Santiago. Additional research on this topic includes investigating applications using cause-modified life tables (Brand, 2005; Stieb *et al.*, 2015) that employ closed-form mathematical relationships and simplifying assumptions to expand applicability of the life-table approach while reducing the computational burden.

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**Supplementary Materials.** To view supplementary material for this article, please visit <http://doi.org/10.1017/bca.2022.5>.

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**Competing Interests.** The authors declare no competing interests.

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