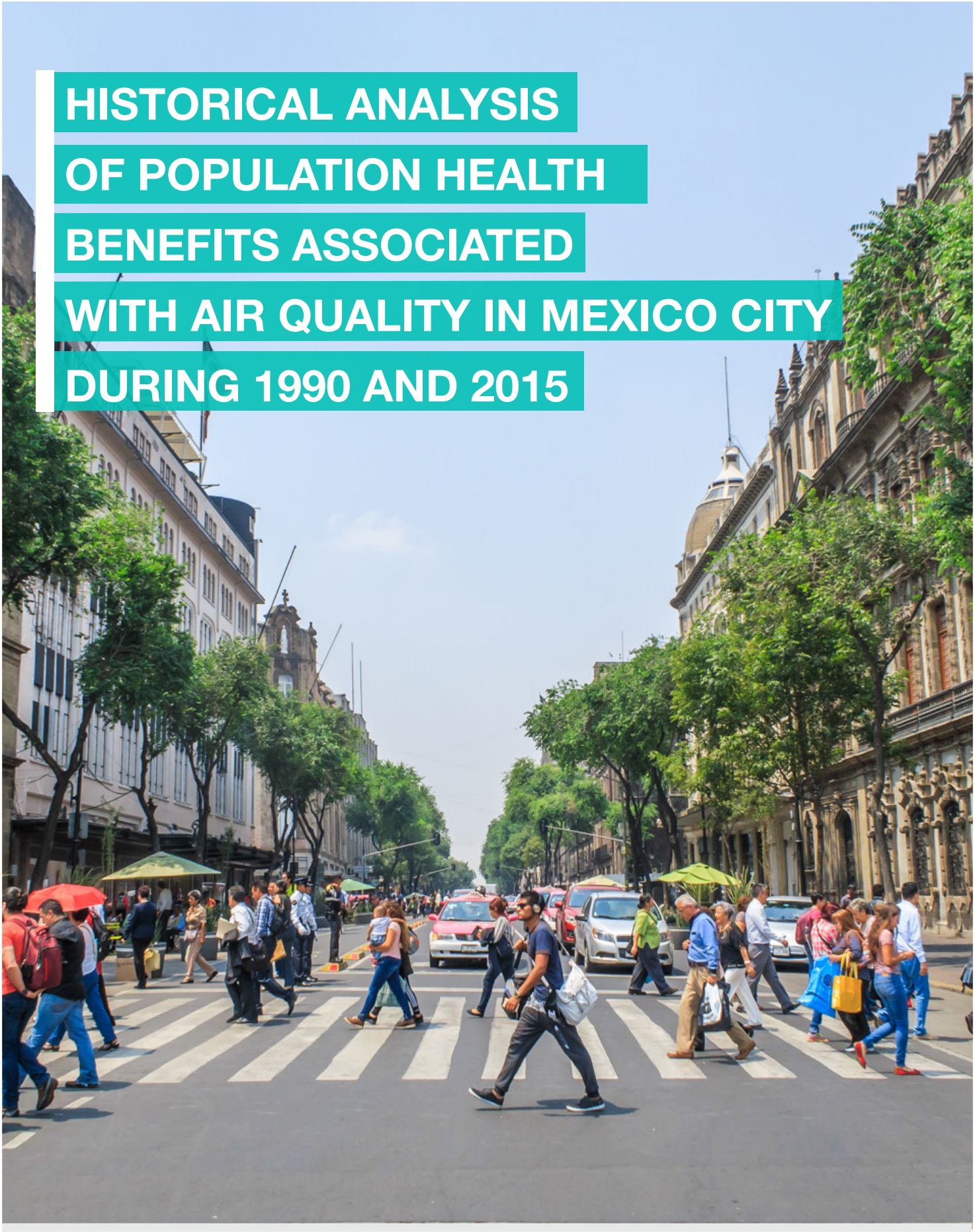
HISTORICAL ANALYSIS OF POPULATION HEALTH BENEFITS ASSOCIATED WITH AIR QUALITY IN MEXICO CITY DURING 1990 AND 2015



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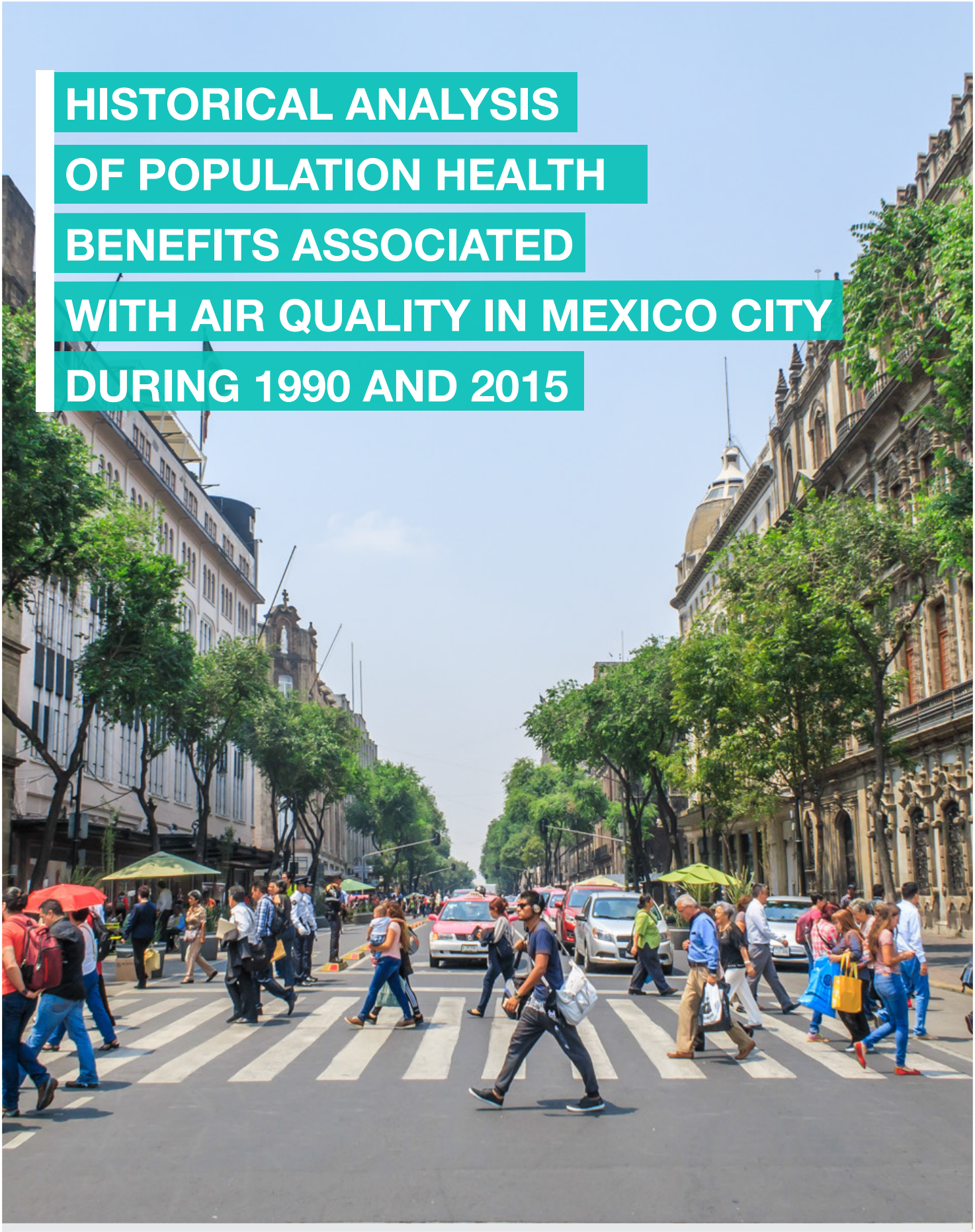

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Air quality is a major environmental risk to health, and one of the major challenges cities worldwide are facing in the 21st Century. According to the World Health Organization (2017), 9 out of 10 people in the world breathe poor air quality; moreover air pollution is a silent killer responsible of 7 million deaths in the world every year.

Mexico City was once the most polluted city worldwide (UNEP, 1992). This condition compromised quality of life and health for millions of inhabitants in the city. For over 25 years, the Government of Mexico City has implemented bold policies which have led to a substantial improvement on air quality. Mexico City is no longer the most polluted city in the world, not even in the country, ranking now in the position 88 of the World Health Organization (2016), together with 42 cities that have $PM_{2.5}$ concentrations of 22 ug/m^3.

The implementation of public policies to improve air quality have achieved that pollution levels maintain a downward trend, despite the steady growth of the City and the vehicle fleet. What is the impact in public health of the implementation of these policies? Have we achieved an improvement in health as we have achieved in air quality? How can we continue improving air quality?

Convinced that scientific evidence is key for responsible environmental policies, in 2014 the Government of Mexico City initiated this collaboration with the Harvard T.H. Chan School of Public Health, with the Secretarìa del Medio Ambiente (SEDEMA), Secretarìa de Salud (SEDESA), the Harvard David Rockefeller Center for Latin American Studies (DRCLAS), and with the participation of the Mario Molina Center for Strategic Studies on Energy, and Environment, and the National Institute for Public Health (INSP).

The Historical Analysis of Air Quality in Mexico City from 1990 to 2015 evidence the health benefits in the population related to air quality. The Study also provides information about cost-benefit analysis of measures to continue improving air quality and policy recommendations for further improvements that are necessary for major health benefits.

This administration has prioritized air quality policies for better quality of life; with scientific evidence-based decision making and the participation of academia, recognized international researchers, as well as national research institutes.

Policies to improve air quality must be effective and inclusive, privileging the common good over particular interests; and with a metropolitan and long term vision, as air pollution does not recognize administrative or political frontiers. The challenge is big and of course there is still a lot to be done, but we are decisively advancing in the right direction, committed to guarantee sustainability and quality of life for all.

Tanya Müller García
Secretary of Environment

Air pollution is increasingly being recognized as a global, but preventable threat to public health. In a recent analysis(1) it was estimated that there were 4.2 million excess deaths worldwide in 2015 attributable to fine particulate air pollution and another 254,000 attributable to ozone. For Mexico, it was estimated that there were 29,000 excess deaths due to fine particles ($PM_{2.5}$) and 18,100 attributable to ozone (O_3).

Nevertheless, there are remarkable examples of significant achievements in reducing air pollution exposures to the population. Mexico City, once labelled as the most polluted mega-city in the world, has taken the challenge seriously, and has substantially reduced air pollution exposures in the Valley. In this report, we examine whether these policies and the sacrifices that the Mexico City population and economy have had to bear to achieve better air quality have been matched by improvements in health.

The health effects of air pollution have been studied extensively in Mexico and specifically in Mexico City. Mexican investigators have been leaders in understanding the chemistry and transport of air pollution, the advantages and disadvantages of various control strategies, and the associated health effects of air pollution. There is a larger body of evidence on the health effects of air pollution, particularly from developed countries in North America and Europe. These results along with estimates of average air pollution from models and remote sensing have allowed estimates of the burden of disease from air pollution in countries around the world, even those without air pollution monitoring(1).

Our challenge was to apply these approaches at the local level in Mexico City. Our overall goals were to develop tools to support cost-effectiveness analyses, to estimate and validate public health benefits from policies over the past 25 years and provide a basis for estimating benefits of proposed policies. Building on the groundbreaking 2002 analyses, Air Quality in the Mexico Megacity: An Integrated Assessment led by Luisa Molina and Mario Molina(2), we have undertaken a multi-disciplinary, cross-institutional assessment of changes in air pollution, population health, and public policy in Mexico City over the past twenty five years (1990 to 2015).

The project was conducted in collaboration with the *Secretaría del Medio Ambiente of the Government of Mexico City (SEDEMA CDMX)* with resources from the Public Environmental Fund. This project has drawn on expertise and assistance from institutions in Mexico and Harvard including the *Secretaría del Medio Ambiente (SEDEMA CDMX)*, the *Secretaría de Salud (SEDESA CDMX)*, the *Instituto Nacional de Salud Pública (INSP)*, the *Centro Mario Molina para Estudios Estratégicos sobre Energía y Medio Ambiente*, the *Harvard David Rockefeller Center for Latin American Studies (DRCLAS)*, and the *Harvard T.H. Chan School of Public Health.*

We show that the policies to control air pollution in Mexico City over the last twenty-five years have substantial benefits in terms of live saved and increased life expectancies. These health benefits can be monetized for cost-benefit analyses to inform public policy decisions. While the improvements in air quality and population health should be lauded, there is also evidence that further improvements in air quality would lead to additional public health benefits. This project provides the tools to better inform these public policy decisions.

The experience in Mexico City in dramatically improving air quality and population health provides unique evidence for the benefits of clearing the air and will serve as a model for mega-cities around the world.

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BACKGROUND AND OVERVIEW

Air quality in Mexico City (CDMX) in the late 1980s and early 1990s was characterized as the worst of all mega-cities in the world(3). Most criteria pollutants (lead, sulfur dioxide, carbon monoxide, nitrogen dioxide, ozone, and particulate matter) frequently exceeded national ambient air quality standards. Since then, municipal, state and federal governments have used laws and regulations to control and reduce air pollutant emissions, to improve air quality and protect public health[4]. Federal public policy actions have been implemented, such as standard setting to regulate emissions of mobile and point sources, improvements in fuel quality, and establishing air quality maximum permissible levels for criteria pollutants (Figure 1.1).

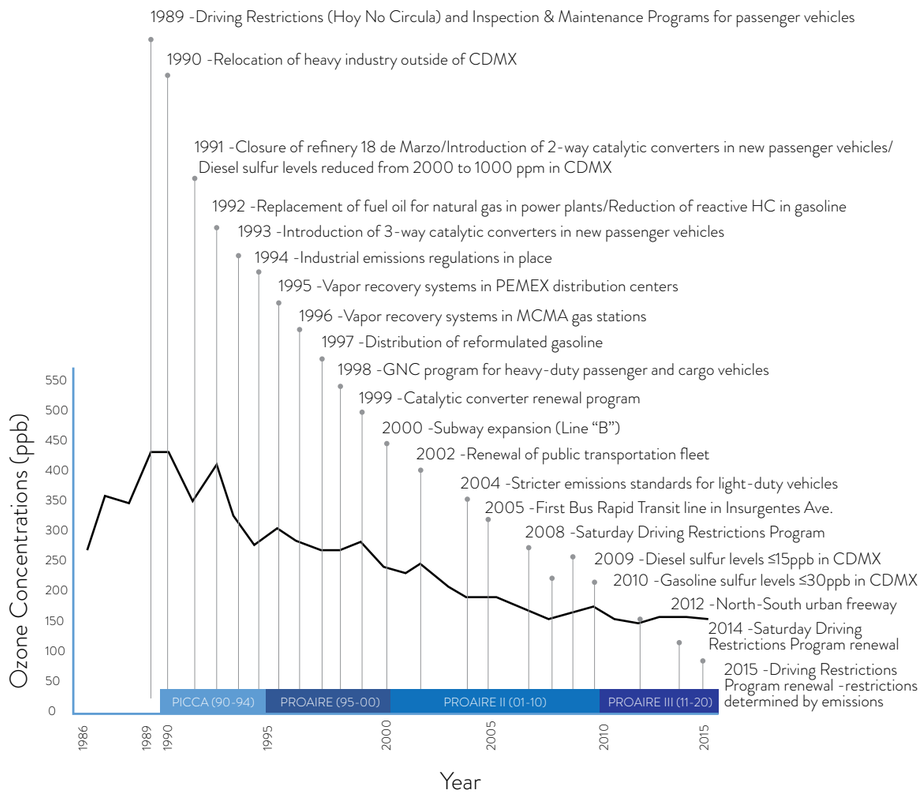

Figure 1.1. Actions implemented aimed at improving air quality in Mexico City since the early 1990

The Government of Mexico City has implemented a series of comprehensive air quality management programs, known as ProAire. These have been developed in coordination with federal authorities, representatives from academia, and the private sector. In addition, several important public policy specific strategies have been launched, including the Environmental Contingencies Program (PCAA), the Hoy No Circula, and the Inspection and Maintenance Programs. These programs have been evaluated and modified in numerous occasions. The now intertwined Hoy No Circula, and Inspection and Maintenance programs have promoted the renewal of the vehicle fleet, accelerated the entry of advanced pollution control technologies, and mandated the continued maintenance of vehicles that circulate in Mexico City and urbanized areas from neighboring states. Such strategies have encouraged the switch to new vehicles and vehicles which comply with in-use emissions standards. In turn, the PCAA seeks to trigger smog alerts for corrective actions to reduce pollutant emissions such as banning circulation of certain vehicles when air pollution exceeds thresholds deemed harmful to sensitive population sub-groups. First stage smog alerts have decreased from 33 per year in 1992 when the program was first launched to zero between 2006 and 2014.

This ensemble of air quality management actions has been successful in reducing emissions and air pollution concentrations in Mexico City (Figure 1.1).

OBJECTIVES

The Secretaries of Environment (SEDEMA) and Health (SEDESA) of the government of Mexico City initiated a program of collaboration with the Harvard T.H. Chan School of Public Health in 2014 (Figure 1.2).

Figure 1.2. Signing ceremony for the Memorandum of Understanding, 14 August 2014.

The overarching theme for this collaborative project is examining the link between the sizeable improvements in air pollution observed in Mexico City over the past 25 years and the public health benefits. We quantified such benefits by means of life expectancy improvements, life of years lost, and attributable deaths avoided. In the following sections we describe the foundation to such analyses, that is the air quality changes, specifically for $PM_{2.5}$ and for ozone, from 1990 to 2015.

We first describe air quality changes as reported using official sources that focus in measurements from fixed-site monitoring sites. Later, in the report, we will present air quality trends at alcaldía level, which is the spatial resolution for our health-related analyses.

AIR POLLUTION LEVELS

Data from SEDEMA's air quality network was integrated and analyzed. We computed fine particles ($PM_{2.5}$) and ozone exposure metrics from 1990 to 2015 and used such estimates for our analyses. $PM_{2.5}$ concentrations were predicted via a Generalized Additive Model (GAM)for years prior to official and continuous air quality fixed-site monitoring efforts (1990 to 2003). For ozone, official data from the monitoring network is available for the entire study period. The indicators of exposure for each pollutant are those used in cohort epidemiological studies and have been associated with adverse health impacts. For $PM_{2.5}$ we estimated annual average concentrations, and for ozone seasonal (6-month) 1-hour maximum daily concentrations.

OZONE AIR POLLUTION

Ozone air pollution concentrations have decreased substantially since 1990 (Figure 1.3). Hourly peak seasonal concentrations in 1990 ranged between 85 and 185 ppb among monitoring stations. The steady decline in ozone concentrations across Mexico City led to 2015 levels ranging between 57 and 92 ppb. Historically, highest ozone levels have been recorded in the southwestern areas of the city *(Pedregal)*.

In the 1990s the 1-hour standard (110 ppb) was exceeded on over 300 days (with a maximum of 344 days in 1994). Since 2003, the standard exceedances have decreased, with a minimum of 118 days reached in 2012. With the new and more stringent standard, lowered to 95 ppb in 2014, the MCMA has seen more days above the limit (over 200 days in 2015).

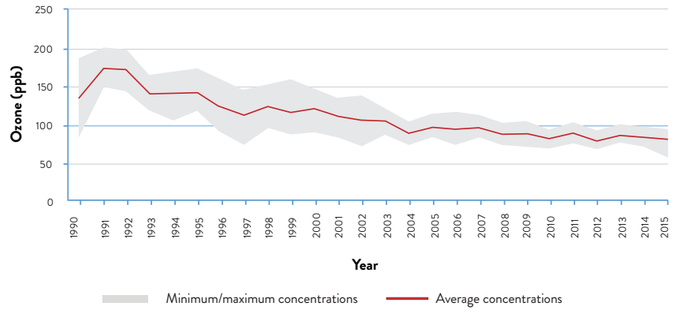

Figure 1.3. Average seasonal (6-month) 1-hour maximum Ozone concentrations in the Mexico City Metropolitan Area, 1990-2015

Source: Elaborated by the authors with official data from fixed-site monitoring network (Red Automática de Monitoreo Atmosférico, SEDEMA, CDMX).

PARTICLES

Particulate air pollution, measured as PM_{10} (particulate matter less than 10 µm aerodynamic diameter) decreased approximately 60% between 1990 and 2015, from over 110 µg/m^3 to less than 45 µg/m^3 (Figure 1.4). In 2014, the MCMA complied with the previous 24-hour standard (120 µg/m^3). However, if the stricter standard in force since the end of 2014 had been applied, the MCMA would have been out of compliance with both the 24-hour and the annual standards (75 µg/m^3 and 40 µg/m^3, respectively) (INECC, 2016).

Fine particles (particulate matter with an aerodynamic diameter ≤ 2.5 µm, $PM_{2.5}$) have decreased slightly since 2004, when fixed-site measurements started in the MCMA. The maximum annual average concentrations decreased from 34 µg/m^3 in 2004 to 24 µg/m^3 in 2015 (Figure I.4). Our $PM_{2.5}$ estimates (via our GAM model) show that in the early 1990 annual average concentrations ranged from close to 30 to almost 70 µg/m^3. The annual $PM_{2.5}$ air quality standard was 15 µg/m^3 until 2014 and was tightened to 12 µg/m^3 by the end of that year. This $PM_{2.5}$ air quality standard has been exceeded every year and at every single monitoring station (5, 6). Historically, the highest annual concentrations have been reported in the northern part of the MCMA at the monitoring stations of Xalostoc and, more recently, Camarones. The lowest levels are reported in the southern areas of Mexico City, such as *Pedregal*.

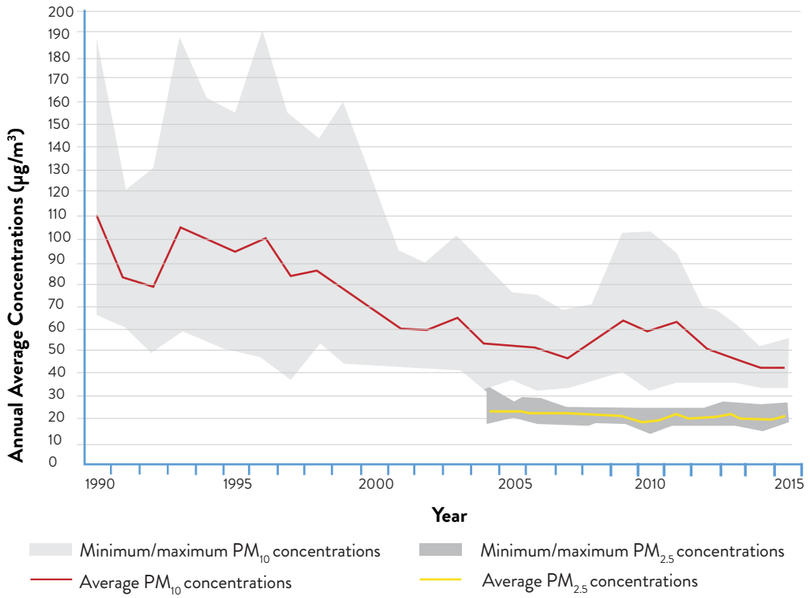

Figure 1.4. Annual PM_{10} and $PM_{2.5}$ concentrations in the Mexico City Metropolitan Area, 1990-2015
Source: Elaborated by the authors with official data from fixed-site monitoring network (Red Manual de Monitoreo Atmosférico, SEDEMA, CDMX). Data available for $PM_{2.5}$ from 2004-2015, and for PM_{10} from 1990-2015 from official monitoring sites. Values estimated from five fixed site monitoring stations that use manual sampling equipment.

OVERVIEW OF ANALYSES

This report presents our findings related with the public health benefits in the population of Mexico City that are attributable to improvements in $PM_{2.5}$ and ozone concentrations since 1990. Epidemiological methods and risk assessment approaches were applied to estimate health benefits that include public health indicators, such as life expectancy, temporary life expectancy, life lost years attributable to death causes that have been determined to be causally associated with air pollution in the GBD analyses and avoided attributable mortality. Finally, this report also includes results of the cost-effectiveness analysis of a public policy strategy to reduce primary particle emissions, improve air quality and protect public health. This policy refers to controlling emissions of in-use heavy-duty diesel vehicles. These analyses are founded on the international state of knowledge relevant to Mexico City.

The study was conducted in four phases. Phase I examined the state of knowledge of the health effects of air pollution in Mexico and the relevance of international studies. Phase II was a risk assessment of the benefits of changes in air pollution in Mexico City over the past twenty-five years. Phase III examined the surveillance data on life expectancy over this same period to validate the risk assessment with observational data. Phase IV presents a tool for cost-effectiveness analyses to improve air quality applied to alternative emission controls of diesel-fueled heavy-duty vehicles that circulate in Mexico City.

PHASE I. STATE OF KNOWLEDGE AND RELVANCE TO MEXICO CITY

Review the state of knowledge and describe the scientific evidence from the most solid epidemiological studies to date, which are to be relied on when interpreting the relationship between air pollutants exposures and adverse health outcomes.

Today, there is robust evidence regarding the adverse health impacts of ambient air pollution. This evidence stems primarily from epidemiological studies, mainly time-series studies of short-term exposures and cohort studies of long-term exposures. These designs complement each other since together the adverse health effects are evaluated for different outcomes and times scales. In Mexico City time-series studies, conducted since the early 1990s, have been the dominant epidemiological design to evaluate the relationship between air pollutant exposures and adverse health impacts (7-10). These studies reported associations between particle and ozone exposures and total mortality, and between ozone and cardiovascular mortality. Sensitive populations sub-groups were identified, people over 65 years old being more sensitive to ozone exposures, and infants to particle exposures.

The most recent times-series analysis, ESCALA (Study of Air Pollution and Health Effects in Latin America) was conducted as a multicity project, that included Mexico City, estimated all-natural cause, cause-specific and age-specific daily mortality associated with daily exposures to PM_{10} and to ozone(11). This study found positive associations between daily levels of PM_{10} and all-cause mortality. The highest risk was reported for chronic obstructive pulmonary disease mortality. Ozone was less strongly associated with increased all-cause mortality than were particles.

Even considering the quantitative and regional variations in the association between air pollution and mortality that have been found among time-series studies conducted in cities around the world, the scientific consensus is that daily fluctuations in particulate matter and ozone have an adverse impact on daily mortality(12).

In Mexico no epidemiological studies to evaluate the long-term health effects associated with chronic air pollution exposures have been conducted. However, there is relevant evidence from cohort studies conducted elsewhere.

The first cohort study to examine the mortality impacts of air pollution exposure was the Harvard Six Cities Study(13). This study showed that individuals living in cities with higher levels of $PM_{2.5}$ air pollution experienced higher rates mortality rates. Figure 2.1. shows that survival rates were much lower (mortality rates higher) in the dirtiest city (Steubenville) than in the cleanest city (Portage). For every 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations, mortality rates increased by approximately 1.5%. Alternatively, Figure 2.1. shows that those living in Steubenville were dying several years earlier than those in Portage, that is higher $PM_{2.5}$ was associated with shorter life expectancy.

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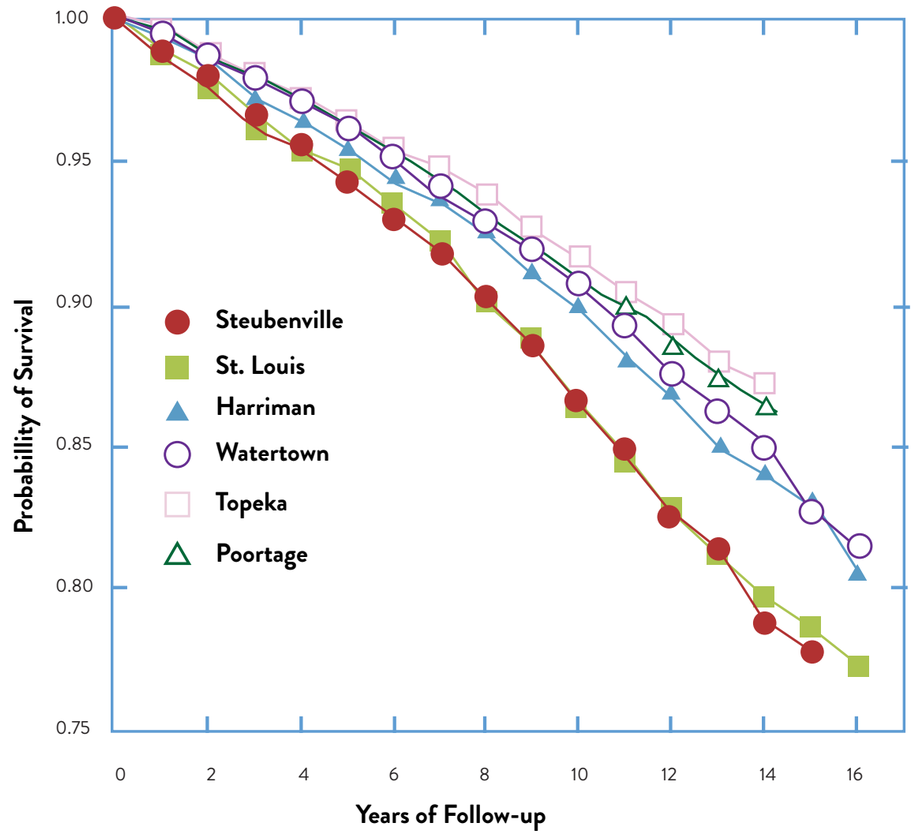


Figure 2.1. Six Cities Study: Crude probability of survival vs. years of follow-up

This study was soon followed by a larger cohort study, the American Cancer Society (ACS) study (14). Consistent with the Six City study, the ACS study found an association between $PM_{2.5}$ concentrations and mortality. However, the size of the effect was about one third smaller, that is for each 1 $\mu g/m^3$ increase in ambient levels of $PM_{2.5}$ mortality rates increased by about 0.4%. The ACS cohort is more than 50 times larger, triples the number of deaths that occurred during the study period; includes white, black and Hispanic subjects (not only white participants); and, improves the statistical analysis and design to control for individual risk factors.

The Six Cities and the ACS study have been vetted thoroughly and have been extended to include prolonged periods of follow-up, that have increased the number of deaths that occurred during the periods under study and the statistical power of the analysis (15, 16). During the extended follow-up periods, air quality improved in the cities included in these cohorts, and the authors found that mortality was reduced, and life expectancy was extended. This is relevant for our project in Mexico City, given the improved air quality today compared to pollution levels in the 90s.

The qualitative consistency of results from these studies is noteworthy. Both found that cardiovascular mortality (a broad category that includes ischemic heart disease and cerebrovascular stroke) and lung cancer mortality were associated with long-term $PM_{2.5}$ exposures. Also, the concentration-response function was found to be nearly linear within the range of concentrations observed in the cities included in each study -from 5.8 to ~30 $\mu g/m^3$ in the ACS, and from 8 to ~30 $\mu g/m^3$ in the Six Cities (15-17).

For ozone, only the ACS found a significant association with mortality, possibly because of the broader range of ozone exposures in the cities that were included in this cohort. The association between seasonal (six month) 1-hour maximum concentrations and mortality was preserved when controlling for $PM_{2.5}$, and the primary effect was on respiratory causes of death (18).

Several new cohort studies have been conducted in the United States, Europe, and Asia. Results have been qualitatively consistent, although there is heterogeneity among their estimated risk coefficients (Figure 2.2). This quantitative variability arises because each study yields a concentration-response relationship for a different population sample (for instance, sub-groups with pre-existing medical conditions or specific occupations). In addition, there are differences between studies in analytical methods and in the elements that comprise the causal chain of the exposure-response relationship. A meta-analysis that evaluated over a dozen cohort studies summary coefficients showed that a 1 $\mu g/m^3$ increase in annual average $PM_{2.5}$ concentrations is associated with a 0.6% increment in all-cause mortality, and an 1.1% increment in cardiovascular mortality (figure 2.2) (19).

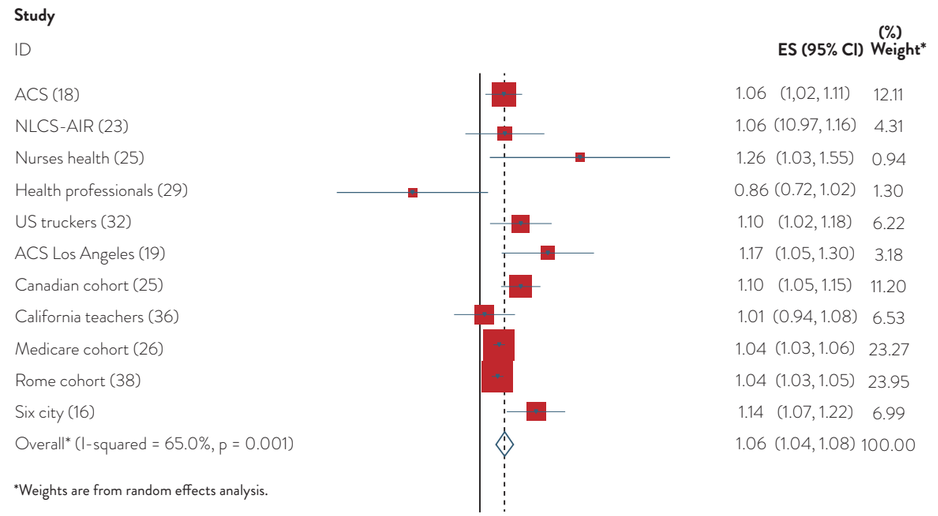
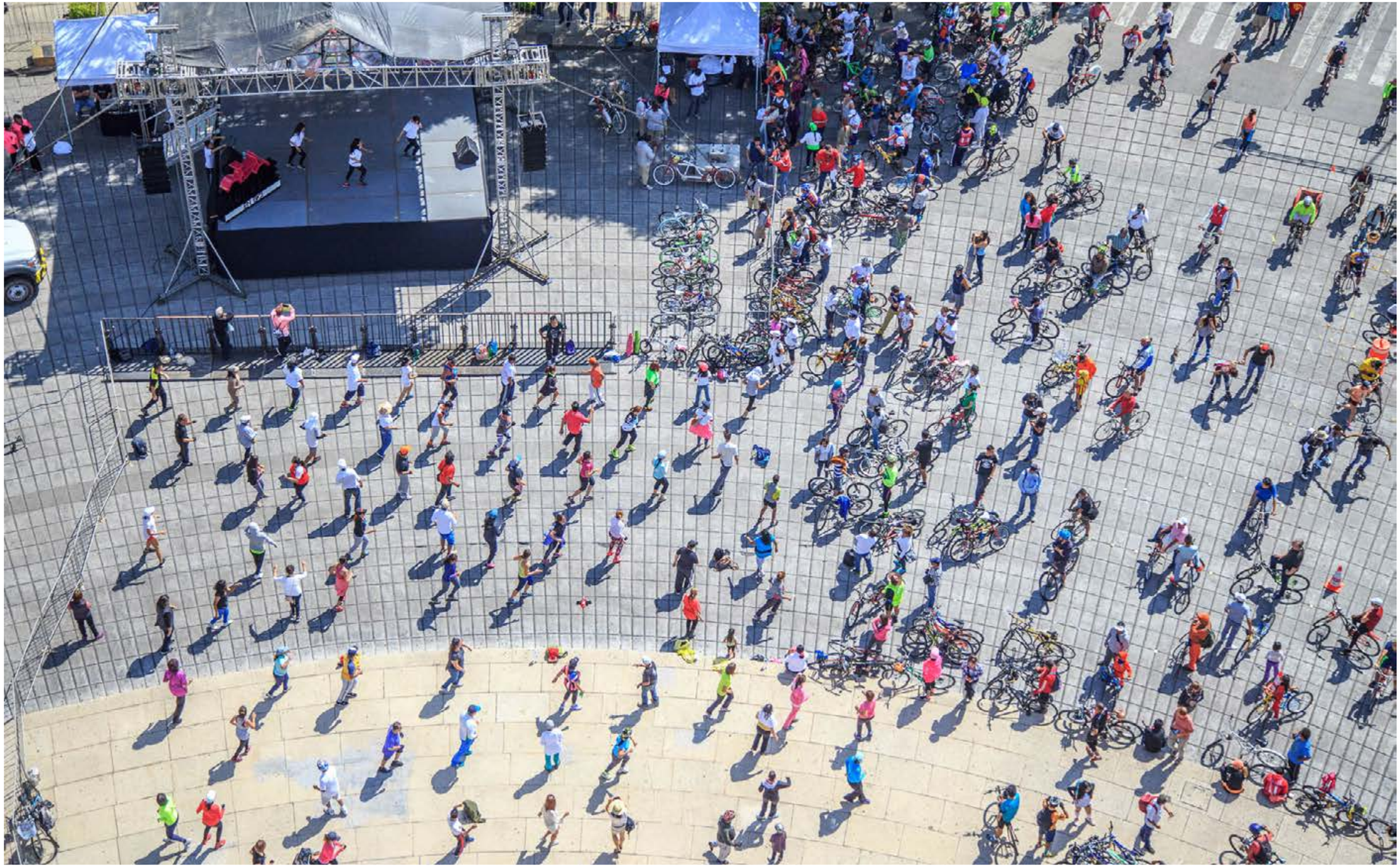


Figure 2.2. Analysis of multiple cohort studies risk estimates for the association between chronic $PM_{2.5}$ exposure and all-cause mortality (Relative Risk per 10 $\mu g/m^3$) (19).

A worldwide effort known as the Global Burden of Disease (GBD), found that ambient $PM_{2.5}$ and ozone air pollution are ranked in the 10th and 21st positions among the nearly 70 risk factors analyzed for 2010 and 2013. A recent analysis(1) estimated that there were 4.2 million excess deaths worldwide in 2015 attributable to fine particulate air pollution and another 254,000 attributable to ozone. For Mexico, it was estimated that there were 29,000 excess deaths due to $PM_{2.5}$ and 18,100 attributable to ozone.

The GBD assessments found that exposure to $PM_{2.5}$ was causally associated with premature deaths from ischemic heart disease, cerebrovascular stroke, and lung cancer, whereas exposure to ozone was causally associated with deaths from chronic obstructive pulmonary disease. The GBD approach was used as the basis for the analyses the effects of air quality improvements on health in Mexico City between 1990 and 2015.



PHASE II. ESTIMATION OF THE HEALTH BENEFITS OF AIR POLLUTION IMPROVEMENTS IN CDMX, 1990-2014

Risk assessment of the health benefits attributable to the reductions in fine particulate matter and ozone concentrations that have been achieved, as a result of public policy strategy implementation from 1990 to 2014 in Mexico City.

The impact of air pollution exposures on public health can be measured as "premature deaths" when the assessment refers to the adverse health impacts of air pollution or as "premature deaths avoided" when the assessment refers to the health benefits of air quality improvements. Risk assessment and burden of disease methods have been applied for this purpose globally and locally.

Recently, a worldwide effort known as the Global Burden of Disease (GBD), found that ambient $PM_{2.5}$ and ozone air pollution are ranked in the 10th and 21st positions among the nearly 70 risk factors analyzed for 2010 and 2013 [20-22]. $PM_{2.5}$ exposures cause around 3 million premature deaths (GBD 95% uncertainty intervals: 2.6 million to 3.6 million premature deaths). For ozone, the GBD estimated approximately 220 thousand premature deaths (95%UI: 160 thousand to 272 thousand premature deaths) (20-22). The GBD assessments show that exposure to $PM_{2.5}$ causes predominantly premature deaths from ischemic heart disease, cerebrovascular stroke, and lung cancer, whereas exposure to ozone is related with chronic obstructive pulmonary disease.

A few risk assessments have been conducted for Mexico or for Mexico City to assess health impacts of air pollution. $PM_{2.5}$ chronic exposures have been reported to be responsible for 7,600 annual premature deaths per year in Mexico (23). For the Mexico City Metropolitan Area (MCMA) roughly 3,000 premature deaths were attributed to chronic exposures to $PM_{2.5}$, and for Mexico City, 6,100 premature deaths were attributed to PM_{10} chronic exposures (23, 24).

The GBD 2010 and 2013 studies analyzed the per-country and per-state burden of disease, including Mexico and Mexico City. For the country, over 13,000 premature deaths attributed to $PM_{2.5}$ chronic exposures, and close to 2,000 to chronic ozone exposures were estimated [25]. For Mexico City, estimated attributable deaths per year for $PM_{2.5}$ and ozone were approximately 2,100 and 220, respectively (25) .In Mexico City ambient exposures to $PM_{2.5}$ and ozone were among the first 20, out of 70, risk factors that were evaluated (Figure 3.1).

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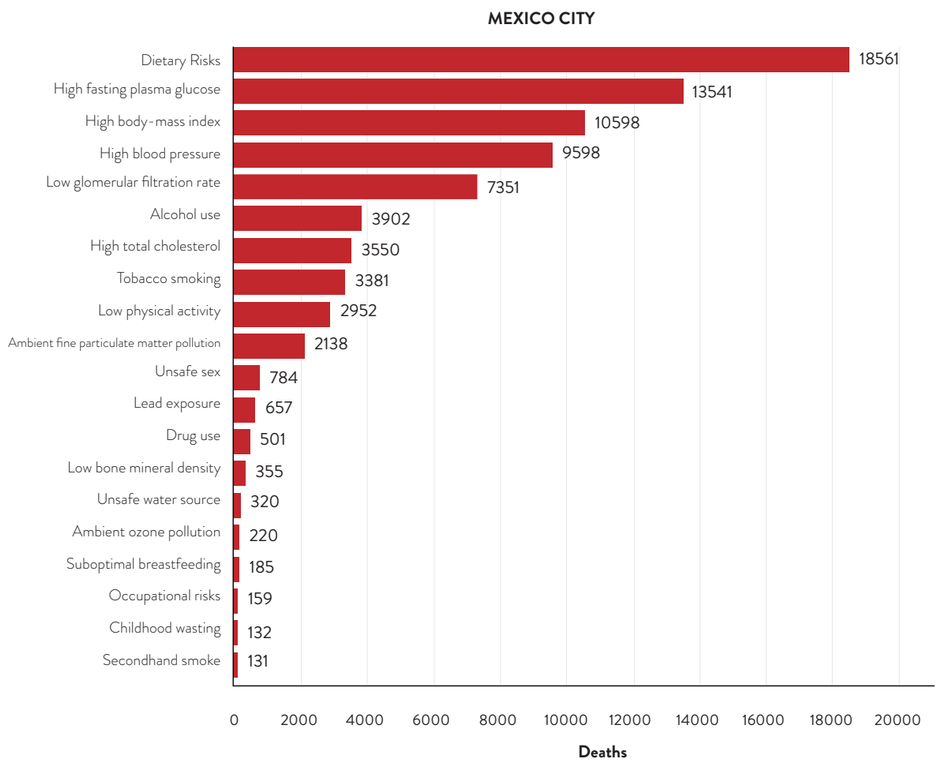


Figure 3.1. Main risk factors and associated premature deaths for Mexico City 2013
Source: Prepared by the authors with information from IHME, 2016.

APPROACH

We applied this indirect method of risk assessment to estimate the benefits associated with air quality improvements in Mexico City in the past 25 years (1990 to 2014). To do so, we characterized the exposure-response relationship to estimate the health benefits accrued due to the improvements in air pollution that occurred in Mexico City since 1990. That is, how much mortality risk decreases for every unit decrease in $PM_{2.5}$ ($\mu g/m^3$) or ozone (ppb).

We relied on a novel approach, known as the "integrated exposure response (IER) function", developed and used to support the GBD analyses for 2010 and 2013 (20, 21, 26). Meta-analysis was used to pool estimates of risk from eight cohort studies of ambient air pollution with results from studies of mortality risk among people exposed to fine particles through active smoking, passive smoking, and use of dirty fuels (coal, dung, wood) indoors for cooking and heating. The GBD analysis of the IER coefficients for fine particle exposures was conducted separately for five classes of disease: for adults, ischemic heart disease, cerebrovascular stroke (haemorrhagic and ischemic), chronic obstructive pulmonary disease, and lung cancer, and for young children, lower respiratory infections.

For ozone, we also followed the approach used by the 2010 and 2013 GBD analyses for estimating mortality risks which relies on analysis of ozone-related mortality in the ACS study (18). Ozone exposure was assessed as the seasonal average (from 1 April through 30 September) of daily 1-hour maximum ozone values, and the health outcome was chronic obstructive pulmonary disease mortality in adults.

In our analysis the shape of the integrated exposure-response functions for $PM_{2.5}$ and for ozone was constrained by the risk observed in high-exposure settings like active and passive smoking. By constraining the concentration-response functions we were able to better model the risk for $PM_{2.5}$ and ozone elevated concentrations that were observed Mexico City in the 1990s, which were higher than those observed in the cohort studies. In the United States and Europe annual average $PM_{2.5}$ concentrations were lower than 30 $\mu g/m^3$ and ozone concentrations did not exceed 104 ppb. In contrast, in Mexico City average $PM_{2.5}$ concentrations from 1990 to 1996 were often on the order of 35 $\mu g/m^3$ and were frequently as high as 50 to 60 $\mu g/m^3$. In the early 1990s ozone seasonal averages of daily 1-hour maxima were frequently in the range of 120 to 180 ppb, and even reached 200 ppb.

RESULTS

By reducing citywide alcaldía-average annual ambient $PM_{2.5}$ concentrations from close to 40 $\mu g/m^3$ in 1990 to close to 20 $\mu g/m^3$ in 2014 and simultaneously reducing citywide alcaldía-average ambient seasonal hourly peak ozone concentrations from over 160 ppb in 1990 to 85 ppb in 2015, Mexico City has been able to reduce the number of deaths attributable to air pollution over this 25-year period by an estimated 22.5 thousand, with a 95% Confidence Interval of 17.9 to 28.0 thousand (Table 3.1.). Roughly 80% of the benefits are due to improvements in $PM_{2.5}$.

Air Pollutant Exposure	Attributable Deaths Avoided (thousands)	CI 95%
Ozone	4.1	(2.7-5.6)
$PM_{2.5}$	18.2	(14.0-23.5)
$PM_{2.5}$ & Ozone	22.5	(17.9-28.0)

Table 3.1. Attributable deaths avoided due to reduction of $PM_{2.5}$ and O_3 exposures in Mexico City, 1990 – 2014

The largest part of the impact is due to reduction of mortality from ischemic heart disease and chronic obstructive pulmonary disease (Figure 3.2, upper panel). Cerebrovascular stroke and lung cancer among adults, and lower respiratory infections among young children also contribute, but together they account for only about one fourth of the mortality benefits of air pollution improvements.

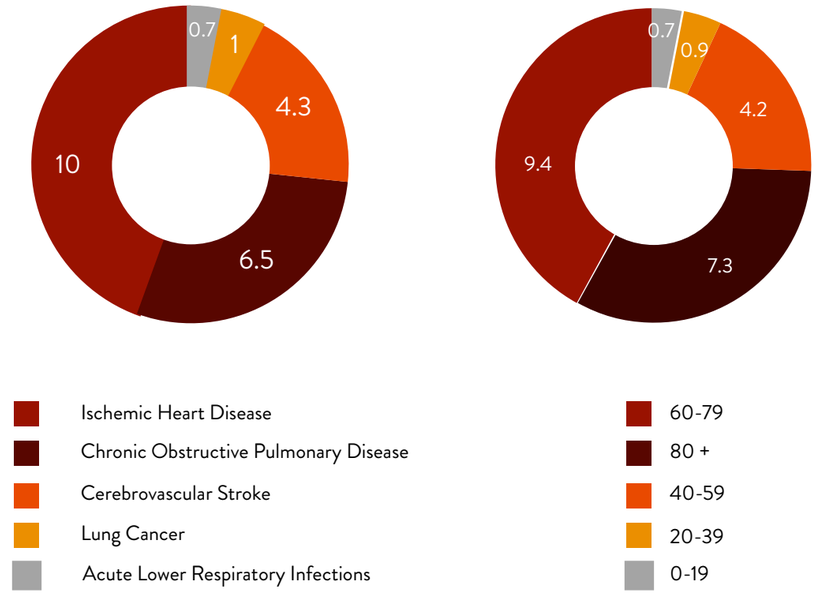


Figure 3.2. Contributions of specific causes of death (upper pane) and age-groups (lower pane) to the expected number of premature deaths (1000's) avoided by reduction of $PM_{2.5}$ and O_3 exposures in Mexico City, 1990 – 2014 (Attributable deaths avoided in thousands).

We also find that the impact of air pollution on mortality is concentrated among the elderly because air pollution primarily affects chronic diseases (Figure 3.2, lower panel). However, when viewed from the perspective of their impact on longevity, deaths among young children from acute lower respiratory infections become much more important. Each of these deaths among children involves many decades of lost life expectancy. In contrast, deaths among adults due to ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, or lung cancer typically involve loss of life expectancy of perhaps one or two decades.

The choices made among our analytical assumptions have impacts on our estimates of health benefits. For instance, if we had not constrained the ozone concentration response function, our central effect estimate would have been ~20% larger (27.0 attributable deaths, 95% CI 21.1 to 33.4 thousand). Likewise, if we had used 1993 instead of 1990 as the reference year, our central estimate would have been ~20% lower (17.8 attributable deaths, 95% CI 13.9 to 22.4 thousand).

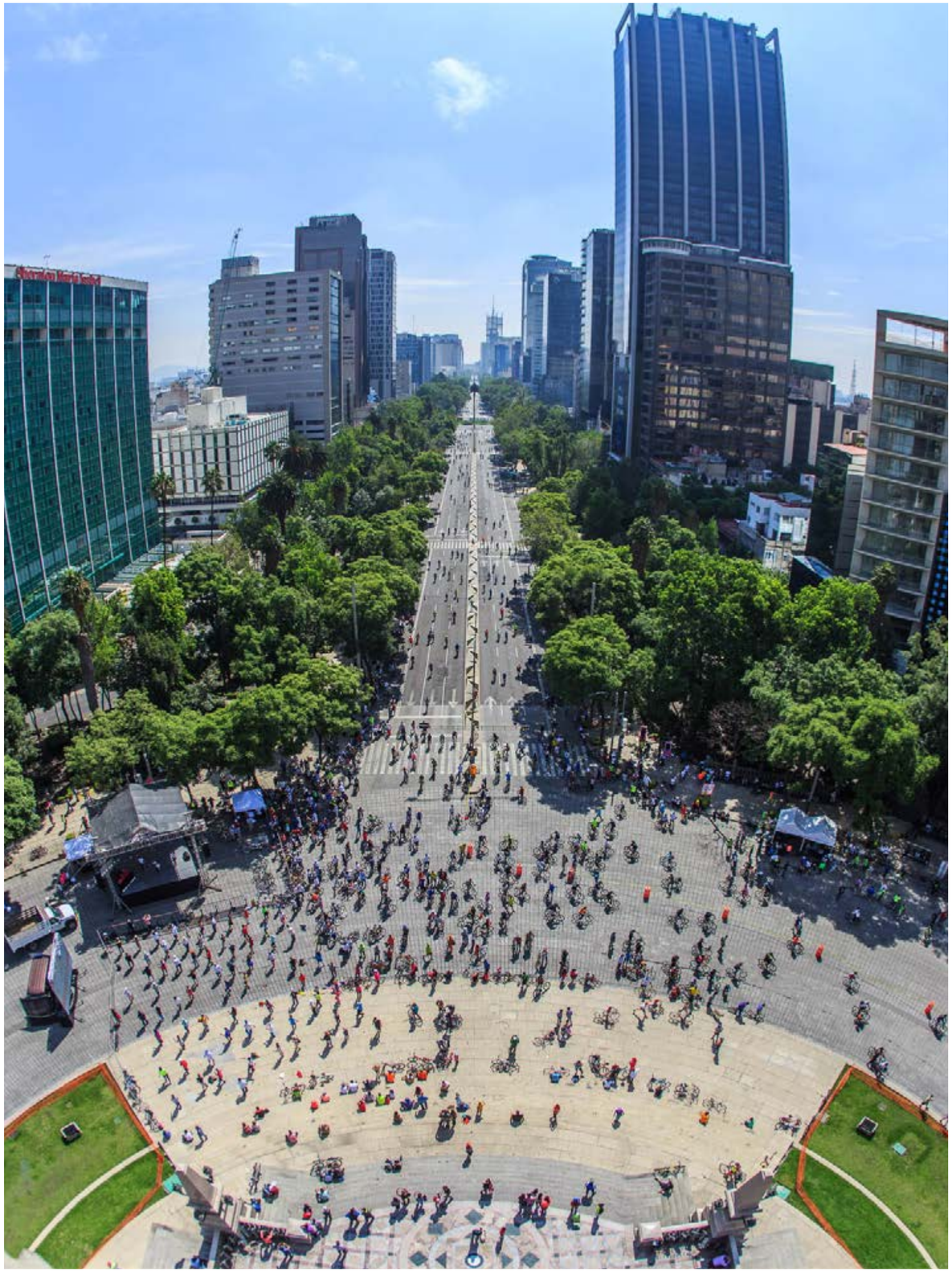
Our estimates have imprecision, with a 95% confidence interval that ranges from almost 18 to 28 thousand deaths attributable to air pollution. This uncertainty arises from the fundamental scientific uncertainty about the true concentration-response functions for $PM_{2.5}$ and ozone, and from uncertainties in the estimates of population exposures. Exposure uncertainties include $PM_{2.5}$ and ozone measurement at monitoring sites, limited monitoring sites in the earlier years of the study period, the spatial interpolation of concentrations from these sites to the alcaldías, and the need to estimate $PM_{2.5}$ concentrations before 2004 when $PM_{2.5}$ was not measured.

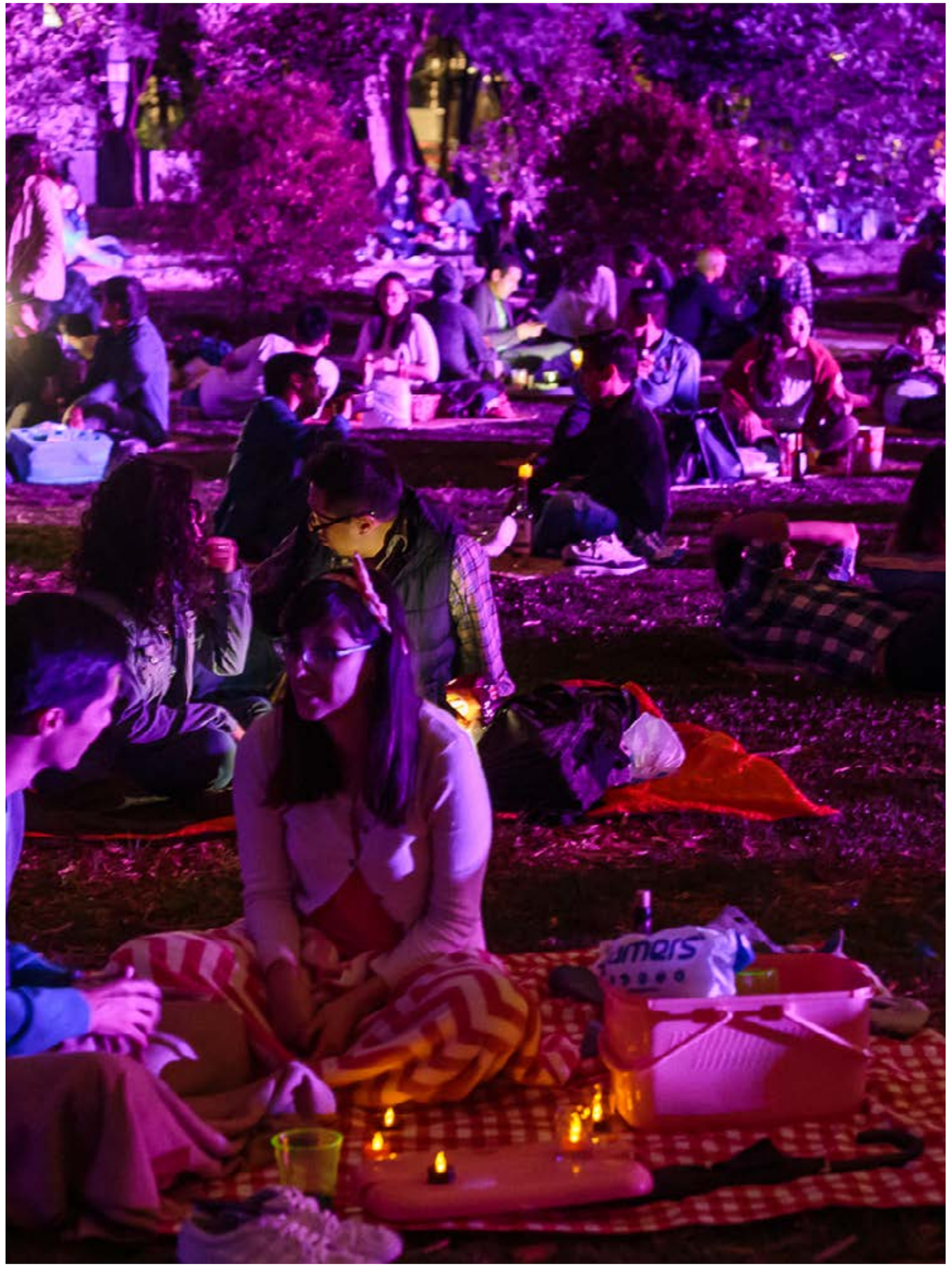
INTERPRETATION

The essential finding is that reductions in $PM_{2.5}$ and ozone over the past twenty-five years have led to substantial improvements in health and reductions in mortality, saving ~ 20 thousand lives over the period.

It is important to recognize that, lives cannot be saved by air pollution controls or any other public policy intervention, but rather are extended. This report uses the measure of 'premature deaths avoided' as a proxy for the increases in life expectancy achieved by improvements of air pollution. Reducing air pollution levels leads to increases in life expectancy. The analyses of life expectancy increases associated with air pollution improvements were conducted in Phase III of this project.

Finally, this analysis assumes that, without the rigorous air pollution controls put in place since 1990, air quality would have remained as it was through the study period. In reality with the growth of the population, the size increase of the pool of middle aged and elderly segments of the population who are most susceptible to mortality due to chronic exposure to air pollution, plus growth in economic activity in Mexico City and the surrounding urbanized area, it is virtually certain that without substantial regulation, air pollution levels would have increased. Thus, our estimates of the mortality benefits of these controls almost certainly underestimate the true benefits of government regulations and programs.





PHASE III. VERIFICATION OF HEALTH BENEFITS DUE TO IMPROVED AIR QUALITY IN MEXICO CITY

Quantification of the public health benefits of air quality improvements in terms of life expectancy using epidemiological analyses of alcaldía-specific health outcomes, air pollution, and additional risk factors in Mexico City from 1990 to 2015.

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The objective of these analyses is to validate that the postulated health benefits of improved air pollution in Mexico City can be observed in surveillance data. That is, can the posited air pollution associated health effects assumed in the risk assessment can be observed in the Mexico City population over the last 25 years. The risk assessment is based on the approach of the Global Burden of Disease (GBD) Comparative Risk Assessment (20-22). The GBD air pollution associations were based on extensive review of the worldwide literature and meta-analyses to develop exposure response functions which can be applied to populations around the world, including populations lacking direct observational studies of the effects of local air pollution.

Air pollution has been associated with a wide range of health effects ranging from premature death, to clinical conditions such as hospital emergency visits and admissions, or diagnosis of chronic disease, to functional changes such as changes in lung function, blood pressure, or cognitive function, to sub-clinical indicators such as lost school days or increased respiratory symptoms. The GBD and this risk analysis have focused on premature mortality as offering the strongest evidence of association, and the most compelling evidence in terms of economic effects. Moreover, the most compelling evidence came from prospective follow-up studies of time to death in populations exposed to varying levels of fine particle ($PM_{2.5}$) and ozone (O_3) air pollution. The GBD found community annual average $PM_{2.5}$ was causally associated with increased total mortality rates and increased mortality among adults (>25 years) from ischemic health disease, cerebrovascular stroke, chronic obstructive pulmonary disease, and lung cancer. Among children (0-4 yrs) $PM_{2.5}$ was causally associated increased mortality from acute lower respiratory infections. Increased seasonal average peak O_3 was independently associated with increased mortality from chronic obstructive pulmonary disease among adults.

This should not be interpreted to mean that there are no effects on other health outcomes for other air pollution components, or for other exposure time periods. Rather these $PM_{2.5}$ and O_3 associations with mortality are well documented and representative of the net effects of the larger range of air pollution exposures.

The objective of these analyses is therefore to evaluate whether the observed changes in health of the Mexico City population over the past 25 years are consistent with expected changes given the improvements in air quality over that time period.

As summarized in Phase I, there is observational evidence of health effects associated with short term air pollution exposures in the Mexico City population. However, there are not as yet longitudinal follow-up studies of mortality in the Mexico City population, consistent with the larger body of evidence for chronic mortality air pollution studies which are the basis for the GBD Comparative Risk Analyses.

We have taken an alternative approach to examine routinely collected alcaldía level surveillance data from death records, census, and environmental monitoring.

This approach examines the substantial differences in life expectancy and air pollution between alcaldías in each of the census years, and the substantial changes in life expectancy and air pollution between census years between 1990 and 2015.

AIR QUALITY

$PM_{2.5}$ exposure metrics were built with direct routine monitoring records and for the earlier years via extrapolation of the $PM_{2.5}$ 2004-2015 records through a Generalized Additive Model. O_3 exposure metrics for each year from 1990 to 2015 were estimated based on routine monitoring. For both pollutants spatial interpolation methods were applied to assign concentrations from monitoring site to alcaldía level.

To illustrate, Figure 4.1. shows the alcaldía-specific mean $PM_{2.5}$ concentrations in three representative years - 1990, 2000 and 2015. In 1990 annual average $PM_{2.5}$ concentrations were estimated to exceed 40 $\mu g/m^3$ in alcaldías in the north. Average $PM_{2.5}$ concentrations in Mexico City were equal to 36 $\mu g/m^3$. By 2015 levels in all alcaldías were below 23 $\mu g/m^3$ and citywide annual average equaled 21 $\mu g/m^3$.

O_3 also has shown very significant improvements (Figure 4.1). City-wide average levels, estimated as seasonal (6 month) 1-hour daily maximum concentrations, in 1990 ranged between 117 and 185 ppb among Mexico City alcaldías, and were above 160 ppb in the southwest. The steady decline in O_3 concentrations through the City led to 2015 mean levels of 84 ppb, and values below 91 ppb in all alcaldías.

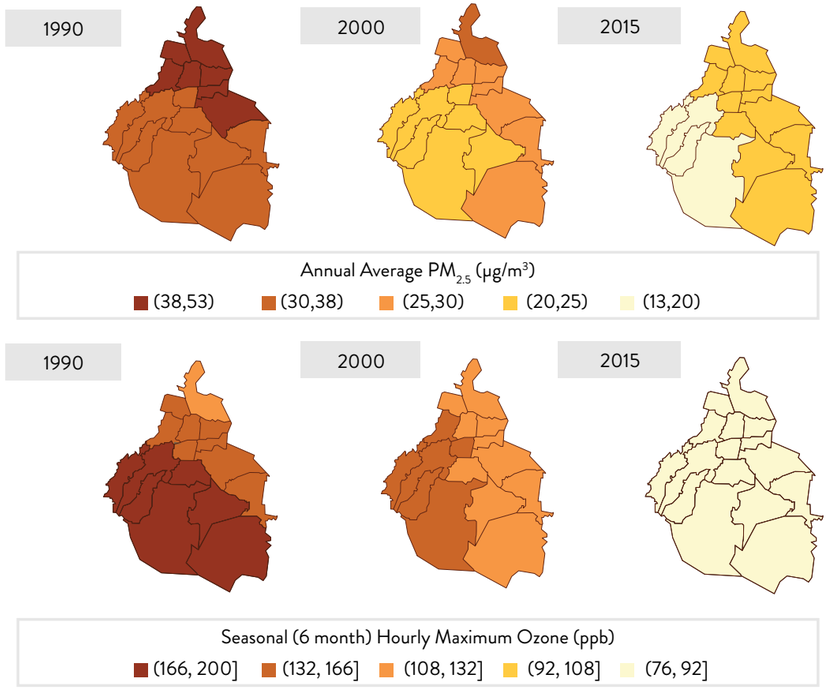

Figure 4.1. Alcaldía-specific mean annual $PM_{2.5}$ and seasonal (6 month) 1-hour maximum O_3 concentrations (ppb) for representative years during study period.

LIFE EXPECTANCY AND YEAR LIFE LOST

Life expectancies for each alcaldía and census year were computed from death counts. Life expectancy at birth for total Mexico City population increased by almost 8% from close to 72 years in 1990 to almost 78 years in 2015 (Figure 4.2).

We calculated temporary life expectancies within specific age ranges. For adults aged 25 to 74 years, the temporary life expectancy in 1990 was close to 42 years, compared to a total possible of 50 years, rising to almost 44 years by 2015. For children 0-4 years, temporary life expectancy increased from 4.87 in 1990 to 4.95 years in 2015.

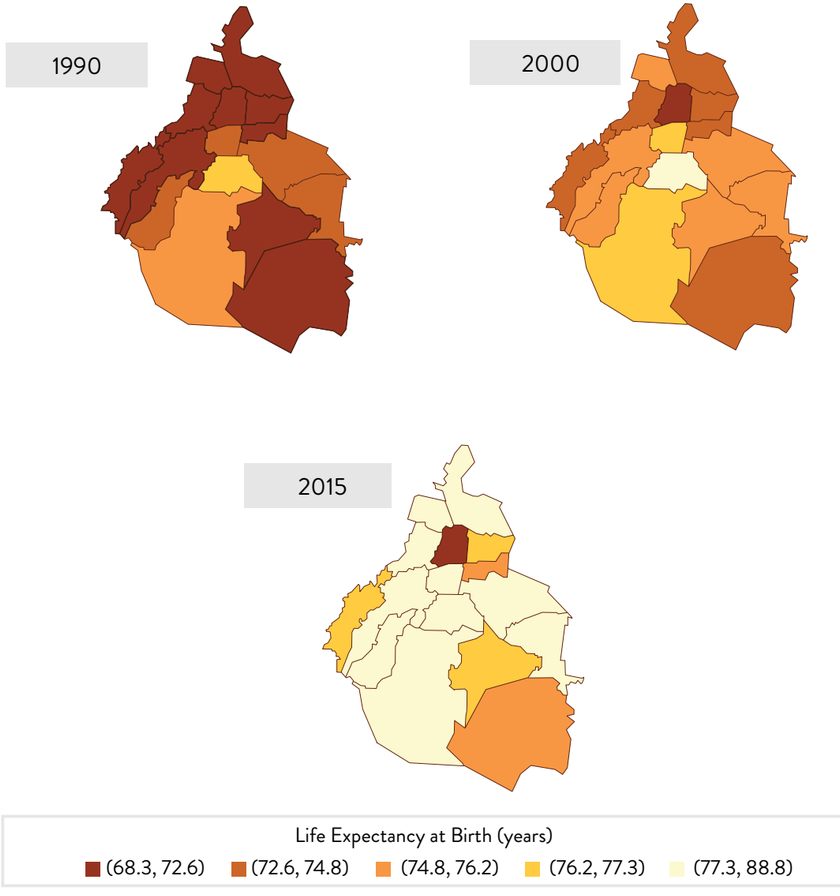

Figure 4.2. Time trends of alcaldía-specific life expectancy at birth (years)

Years life lost is the difference between possible years of life and observed temporary life expectancy. In addition to total years life lost, we examined years of life lost from the five selected causes of death associated in the GBD with exposures to $PM_{2.5}$ or O_3 – ischemic heart disease, cerebrovascular stroke, lung cancer, chronic obstructive pulmonary disease among adults (25 to 74 years), and acute lower respiratory infections among children (0 to 4 years). Table 4.1. shows the trends in years of life lost for adults and children for these causes for the entire Mexico City population.

	25 TO 74 YEARS					0 TO 4 YEARS	
CENSUS YEAR	Total	Ischemic Heart Disease	Cerebrovascular Stroke	Lung Cancer	Chronic Obstructive Pulmonary Disease	Total	Acute Lower Respiratory Infections
1990	8.22	0.89	0.20	0.14	0.24	0.125	0.015
1995	7.68	0.86	0.18	0.12	0.18	0.097	0.011
2000	6.69	0.74	0.21	0.10	0.16	0.078	0.006
2005	6.24	0.74	0.17	0.09	0.14	0.071	0.007
2010	6.07	0.70	0.16	0.09	0.14	0.062	0.004
2015	6.17	0.68	0.13	0.08	0.11	0.047	0.004

Table 4.1. Years of life lost between 25 and 74 years and 0 to 4 years of age, total and by causes related to air pollution for Mexico City

SOCIO-ECONOMIC POSITION INDICATORS

Life expectancy also is affected by socio-economic position. This study relies on CONAPO's socioeconomic position indicators constructed from census data. Table 4.2. shows the fraction (%) of the population of Mexico City reporting each of CONAPO's socioeconomic position indicators for 1990 to 2015.

YEAR	Low Income	Overcrowding	Low Education	Illiterate	No Sewer / Toilet	No Electricity	No Running Water	Soil Floor	Small Villages
1990	62	48	17.5	4.4	1.1	1.1	4.3	3.5	1.3
1995	47	47	12.0	3.1	0.1	0.1	2.9	1.1	1.3
2000	42	36	12.2	3.0	0.2	0.2	1.8	1.7	1.1
2005	33	30	9.6	2.6	0.2	0.2	2.0	1.4	1.5
2010	28	26	8.5	2.1	0.1	0.1	2.4	1.2	1.8
2015	28	20	6.5	1.5	0.1	0.1	1.7	0.6	1.8

Table 4.2. Behavior of Socioeconomic Position Indicators for Mexico City (%), 1990-2015

There have been substantial improvements in these socioeconomic position indicators over this 25-year period in Mexico City. To illustrate, the fraction of houses with some degree of overcrowding dropped from 48% to 20% between 1990 and 2015 (Table 4.2). However, as shown in Figure 4.3. there were equally large differences between alcaldías in 1990 which had been substantially reduced by 2015.

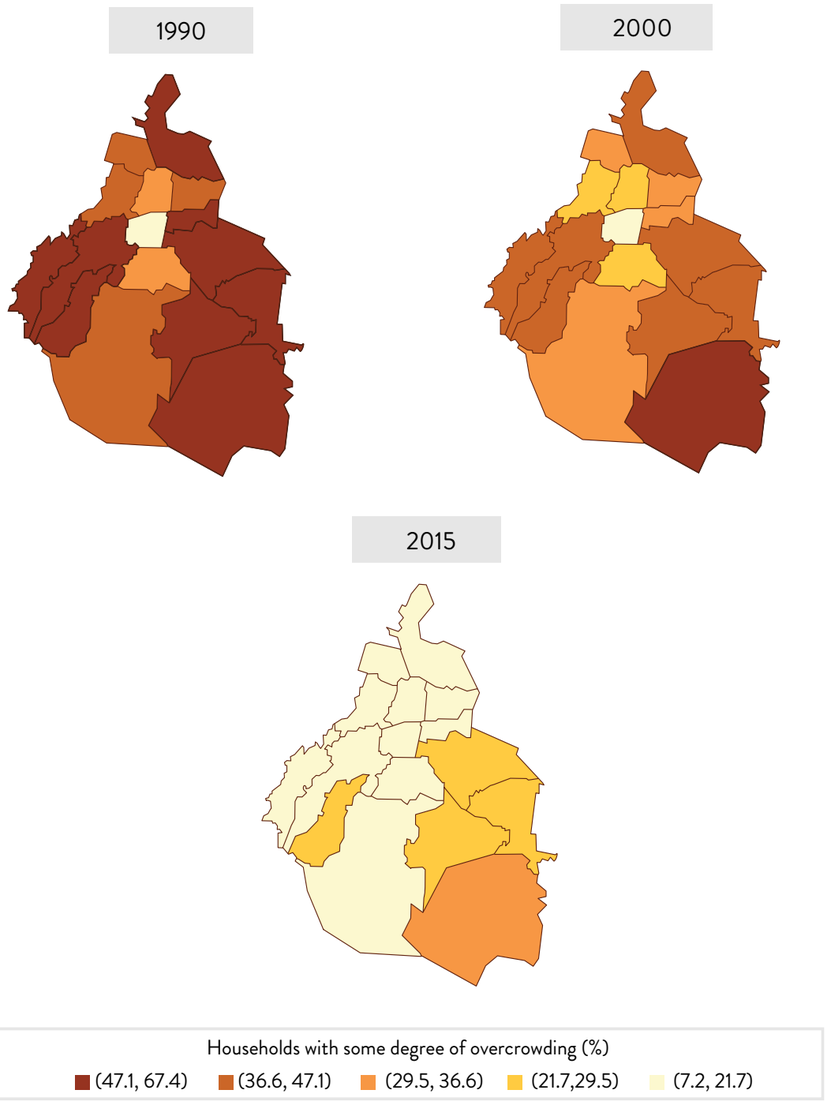

Figure 4.3. Spatial distribution of alcaldía-specific households with some degree of overcrowding (%) by year

REFERENCE CAUSES OF DEATH

Changes in life expectancy or in years of life lost are likely to be associated with factors not captured by the socioeconomic position indicators described above. Such risk factors may include individual behavioral factors like nutrition, or institutional factors such as access to health care. To provide insight into these unmeasured risk factors, we include "reference" causes of death, which are common causes of death not expected to be associated with air pollution. Reference causes of death in these analyses are diabetes, hypertension, colon cancer, stomach cancer, and external causes (including assault).

Diabetes mortality rates increased from 117 per 100,000 in 1990 to 172 in 2015 (Figure 4.4). Hypertension mortality rates also show an increase of close to 30%, with rates of 25 to 33 per 100,000 in 1990 and 2015, respectively. Colon cancer mortality increased as well, with an 80% surge (6.6 in 1990 to 12 per 100,000 in 2015). In contrast, mortality rates of stomach cancer in Mexico City have shown little change from 1990 to 2015, remaining close to 11 per 100,000 throughout the period.

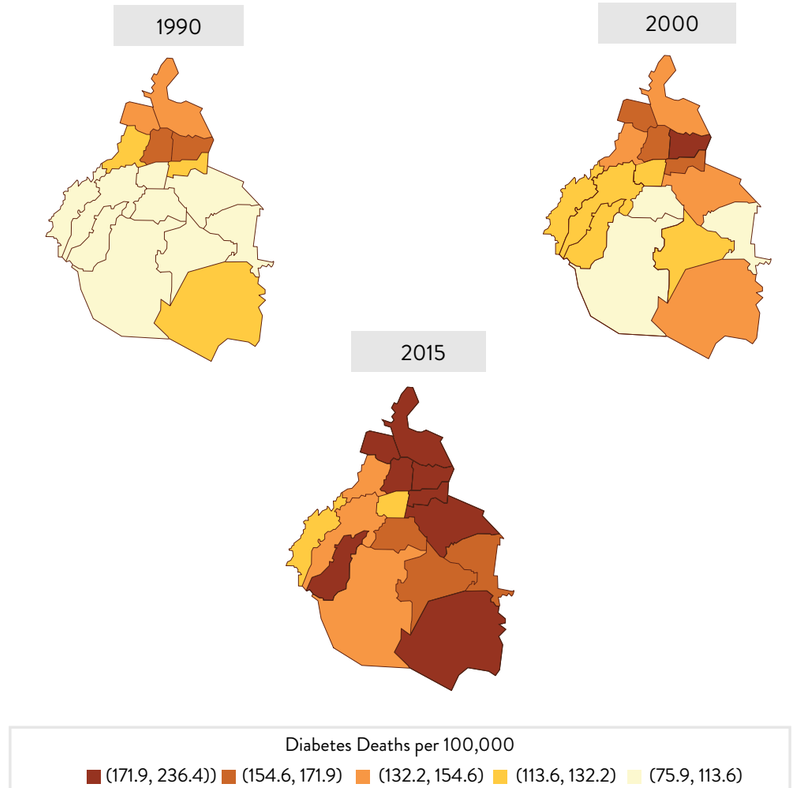

Figure 4.4. Time trends of alcaldía-specific diabetes mortality rates (deaths per 100,000)

External causes include deaths by assault (homicide and injuries inflicted by another person with intent to injure or kill by any means). There is some heterogeneity in the alcaldía-specific rates of mortality due to external causes; it's noteworthy that Cuauhtémoc shows high rates thorough the period and higher rates than the rest of the alcaldías in 2015 (Figure 4.5).

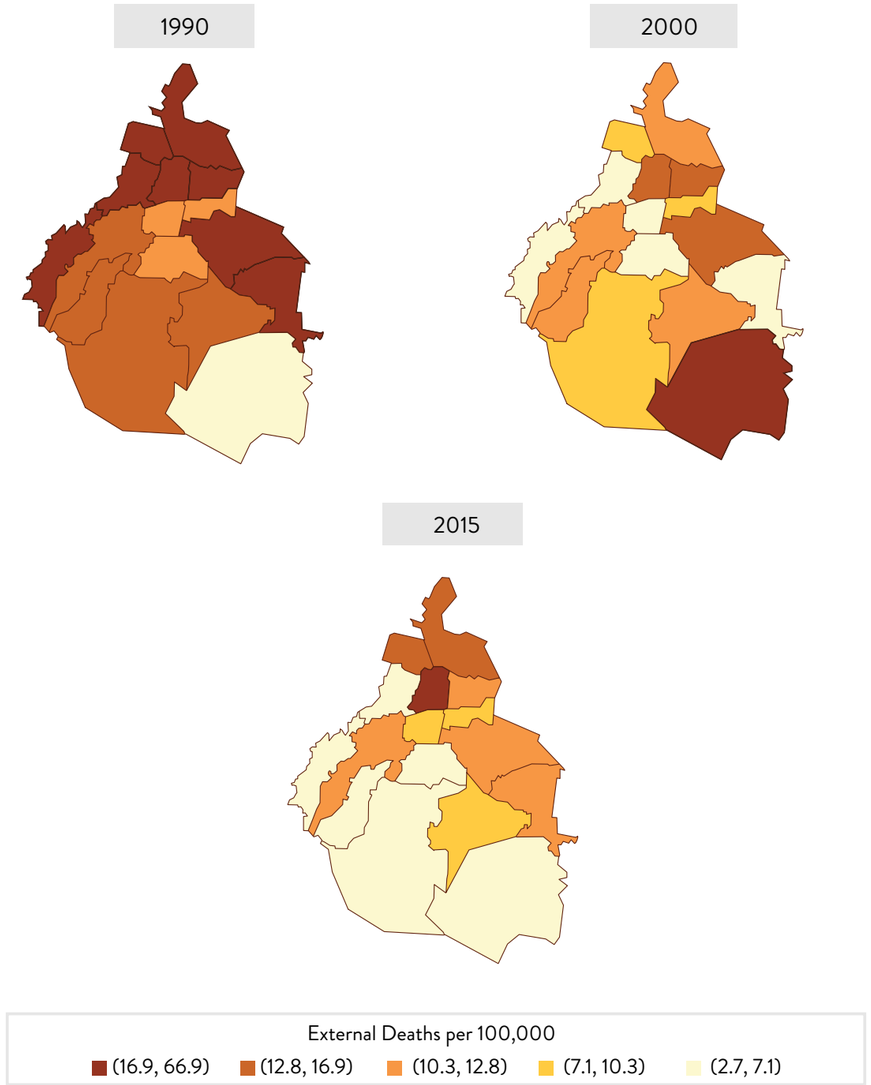

Figure 4.5. Time trends of alcaldía-specific external causes' mortality rates (deaths per 100,000)

SMOKING RELATED DISEASES

Due to limited smoking prevalence data, we used death rates for COPD and lung cancer as proxy indicators of exposure to smoking. The COPD mortality rate in Mexico City dropped during the study period, from 44 to 36 deaths per 100,000 in 1990 and 2015, respectively. There was limited between-alcaldía variability through the period (Figure 4.6).

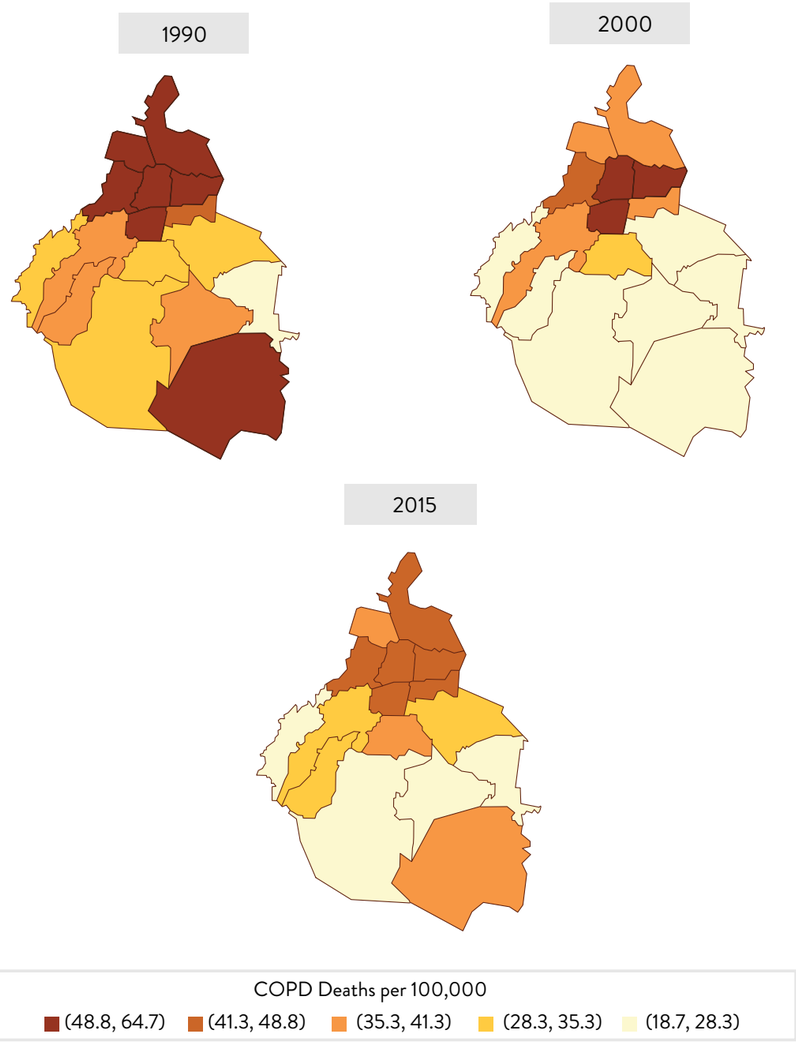

Figure 4.6. Time trends of alcaldía-specific COPD mortality rates (deaths per 100,000)

Lung cancer mortality rates in Mexico City have changed little over the 25-year study period, from 13 to 12 deaths per 100,000 between 1990 and 2015 (Figure 4.7).

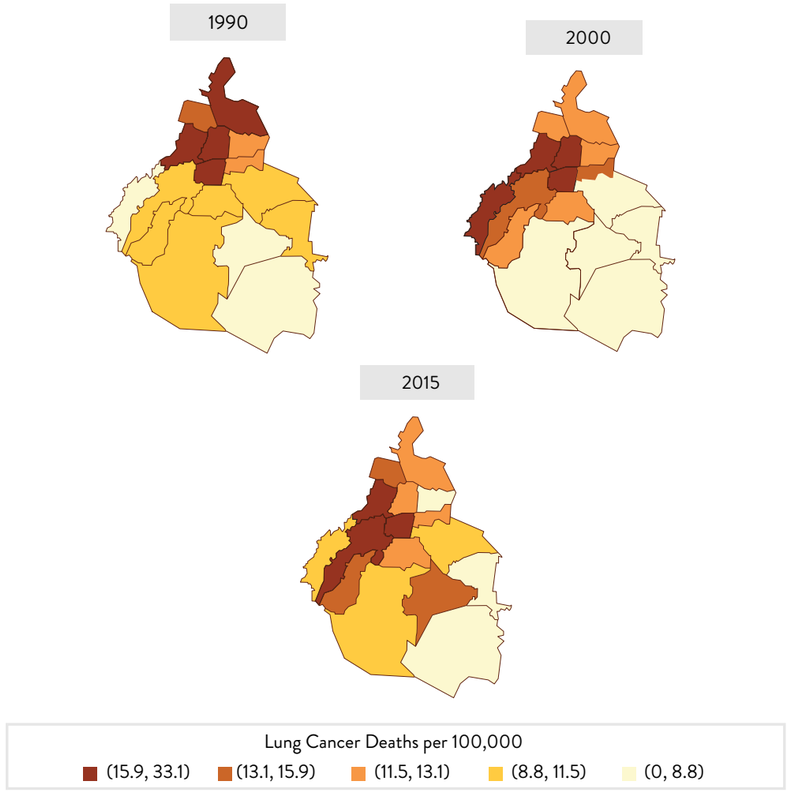

Figure 4.7. Alcaldía-specific lung cancer mortality rates (deaths per thousand)

APPROACH: AIR POLLUTION ASSOCIATIONS

Our analyses seek to assess how life expectancy and years of life gained are affected by alcaldía-specific air pollution controlling for possible confounding by individual, population, and community risk factors.

Plots of life expectancies versus air pollution suggest negative associations between alcaldía- and year-specific life expectancy and both $PM_{2.5}$ (Figure 4.8) and O_3 (figure not shown).

The following figure presents the same data showing the longitudinal relations by alcaldía between life expectancy and PM2.5 (Figure 4.9).

Negative longitudinal (within each alcaldía) associations between life expectancy and $PM_{2.5}$ are observed. A similar negative pattern was noted for the ozone longitudinal relation with life expectancy (figure not shown).

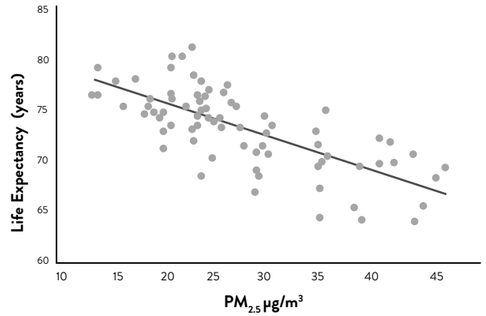

Figure 4.8. Scatterplot and fitted line of alcaldía- and year-specific life expectancy versus corresponding measures of annual average $PM_{2.5}$ concentrations

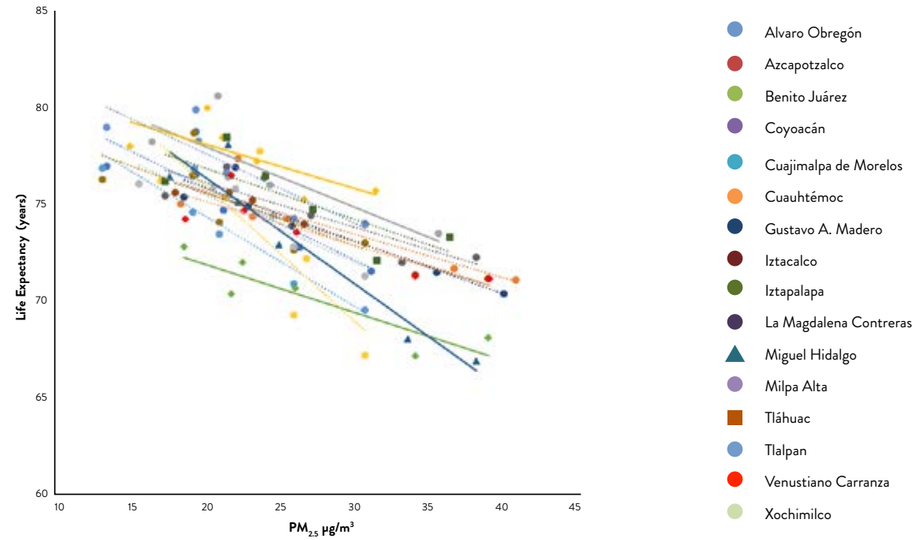

Figure 4.9. Scatterplot and fitted lines of year-specific life expectancy versus annual average $PM_{2.5}$ concentrations by alcaldía

We built mixed models for the associations of life expectancy with $PM_{2.5}$ and O_3 in a stepwise fashion. We first assumed a linear association of $PM_{2.5}$ and O_3 with life expectancy, and that each alcaldía and that each census year had a separate, random level of life expectancy. We gave more weight to the points with larger populations, weighting by the square root of the population. We added all nine-socio-economic position (SEP) indicators, the death rates for the five reference causes of death and the two proxy indicators of smoking (lung cancer and COPD death rates) for each alcaldía and census year. In this full model, there were only significant associations with ozone, diabetes and COPD death rates, and the random indicator for alcaldía --and marginally with overcrowding (%) and colon cancer death rates.

Therefore, we built a parsimonious model that eliminated in backward stepwise regression all nonsignificant predictors, other than those we defined a priori as critical. This final, parsimonious life expectancy model included the air pollution effects ($PM_{2.5}$ and O_3), the random effects of alcaldía and census year, fixed effects for one SEP indicator (% overcrowding), fixed effects of three reference death rates (diabetes, colon cancer, and external causes), and the two proxy indicators of smoking (lung cancer and COPD death rates).

RESULTS

We applied one Parsimonious Model to each of our health outcome indicators: total and sex specific life expectancy at birth; years of life lost for children and adults; and years of life lost due to specific mortality causes expected to be associated with air pollution (Table 4.3).

	$PM_{2.5}$ (10 µg/m³)			O_3 (10 ppb)		
	Years Life Lost	(95% CI)		Years Life Lost	(95% CI)	
Lifetime Ages 25-74 year	0.89	(0.14, 1.65)	*	0.24	(0.08, 0.40)	**
Ischemic Heart Disease	0.56	(0.28, 0.83)	***	0.103	(0.03, 0.17)	**
Cerebrovascular Stroke	0.09	(0.03, 0.16)	*	0.003	(-0.01, 0.02)	
Lung Cancer	0.023	(-.001, 0.047)+		0.001	(-.005, 0.007)	
Chronic Obstructive	0.013	(-.002, 0.028)+		0.003	(-.001, 0.008)	
Pulmonary Disease	0.037	(0.007, 0.067)	*	0.0052	(-.002, 0.012)	
Ages 0-4 year						
Acute Lower	0.0070	(-.006, 0.020)		0.0037	(0.000, 0.007)	*
Respiratory Infections	-0.0033	(-.006, -.001)*		0.0004	(0.000, 0.001)	

+ p,0.10, *p<0.05, ** p<0.01, *** p<0.001

Table 4.3. Effects of $PM_{2.5}$ and O_3 in Parsimonious Models of total years of life lost, for two-age groups (25 to 74 years and 0 to 4 years), and for causes of death associated with air pollution, with one model per health outcome.

For total life expectancy, the Parsimonious Model showed that improvements in O_3 (10 ppb mean seasonal peak) were significantly associated with 0.24 years of increased life expectancy (95% CI 0.08 to 0.40 years). There was an independent, significant association of improved $PM_{2.5}$ (10 µg/m³ annual mean) with an increase of 0.89 years life expectancy (95% CI .14 to 1.65 years). We found no difference between men and women in their association with $PM_{2.5.}$ The association with O_3 was stronger among men than women, although the confidence intervals of these sex-specific associations were overlapping.

Our results further indicate a statistically significant association of life of years lost between ages 25 and 74 years with $PM_{2.5}$ (0.56 years) and with O_3 (0.103 years). For this age-group $PM_{2.5}$ was associated with significant increases in years of life lost attributable to ischemic heart disease (IHD) and chronic obstructive pulmonary disease (COPD); also, there were positive, marginally statistically significant ($p<0.10$) associations for cerebrovascular stroke and lung cancer with $PM_{2.5}$. We found a positive, but statistically nonsignificant association of O_3 with COPD and lung cancer deaths. Recall that the Global Burden of Disease Comparative Risk Analyses found causal associations of $PM_{2.5}$ with these four causes of death among adults >25 years of age The GBD also found causal associations of O_3 with deaths only from chronic obstructive pulmonary disease.

For children between ages 0 and 4 years, we found modest, statistically non-significant increases in years of life lost associated with $PM_{2.5}$ and O_3, but no association with acute lower respiratory infections (ALRI). In comparison, the GBD analyses postulated a causal association between ALRI and $PM_{2.5}$ in this age group.

INTERPRETATION

Life expectancy in Mexico City inhabitants is affected by exposures to air pollution. We found that over the past 25 years air quality improvements in Mexico City have been associated with increased life expectancy.

For $PM_{2.5}$ we found that for each 10 µg/m³ improvement in annual mean, there was an increase of 0.89 years life expectancy (95% CI .14 to 1.65 years). The evidence for longer life expectancy in Mexico City associated with reduced $PM_{2.5}$ is very consistent with, although larger than, similar studies of county-specific life expectancy changes in the United States. Pope et al. (27) and Correia et al. (28) reported, respectively, that life expectancy increased by 0.61 years (95% CI 0.22 to 1.00) and 0.35 years (95% CI 0.04 to 0.66) associated with each 10 µg/m³ improvement in annual average $PM_{2.5}$.

There is limited evidence that living in communities with higher O_3 is associated with increased mortality and shorter life expectancy independent of $PM_{2.5}$. It is likely that the differential spatial variability pattern of O_3 and $PM_{2.5}$ concentrations in Mexico City, with high O_3 levels in the southwest vs. high $PM_{2.5}$ levels in the north and northeast, allowed the identification of an independent effect for O_3. The finding in Mexico City that improvements in life expectancy are associated significantly with reductions in O_3 may have been also possible due to the wide range of concentrations seen across the study period, spanning 80 to 160 ppb, which provides the statistical power to detect an association. This is an important contribution to the scientific evidence of population health benefits that result from air quality improvements.

What is the overall effect of the improvements in air quality in Mexico City over the last 25 years? If we apply the Parsimonious Model results to the observed changes in $PM_{2.5}$ and O_3, we can estimate the net benefits. Annual average $PM_{2.5}$ concentrations have decreased by almost 15 µg/m³ from 1990 to 2015, which would imply an increase of 1.3 years in life expectancy. Improvements for ozone, with decreased seasonal 1-hour maximum daily concentrations of almost 80 ppb, would imply an increase in life expectancy of close to 1.9 years. Thus, the joint net benefit associated with improvements in both pollutants represents an increase in life expectancy of 3.2 years. As seen in Figure 4.10, net benefits present a different spatial pattern for $PM_{2.5}$ and O_3.
Greater improved $PM_{2.5}$ air quality in the north has led to larger gains in life expectancy (up to 1.7 years) in those alcaldías attributable to $PM_{2.5}$. Greater improvements in O_3 air quality in the south have led to larger gains in life expectancy (up to 2.6 years) attributable to O_3. Together, the joint effects of the improvements in $PM_{2.5}$ and O_3 have led to substantial improvements in life expectancy (2.6 to 3.4 years) in all alcaldías (Figure 4.10).

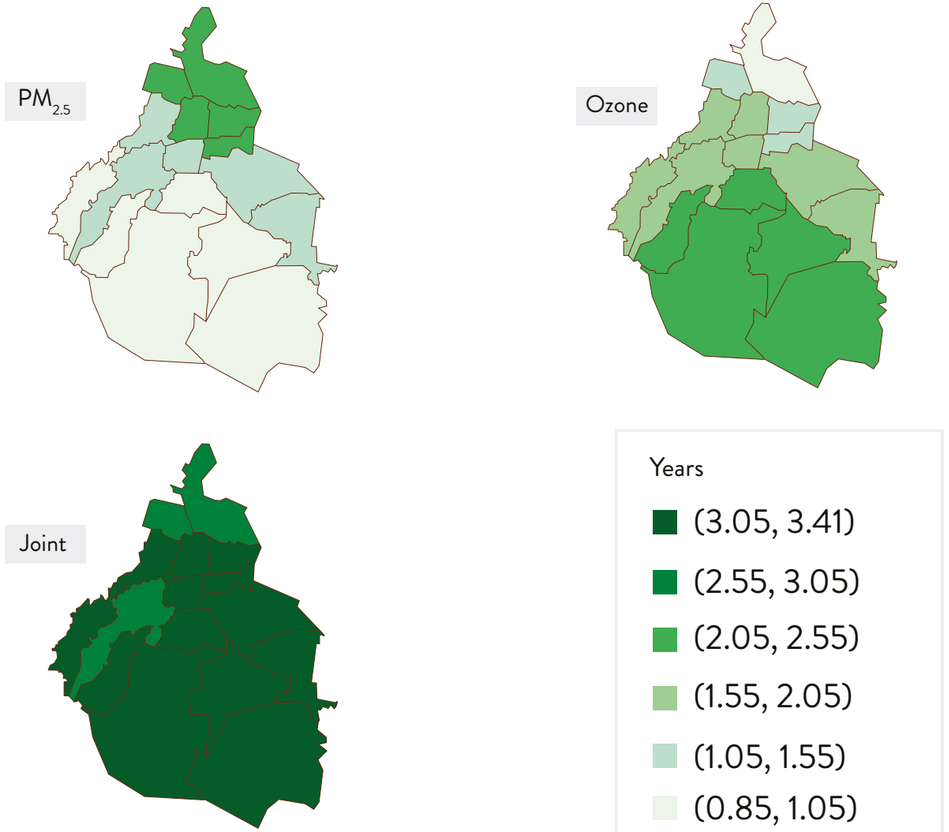

Figure 4.10. Independent and joint net benefits measured as life expectancy gains (years) from improved annual average $PM_{2.5}$ concentrations and seasonal maximum 1-hour daily ozone concentrations in Mexico City, 1990 – 2015

A recent estimation of the net effect of air pollution on life expectancy using the GBD approach estimated that current $PM_{2.5}$ exposures reduce life expectancy globally by 1.03 years, and O_3 exposures by 0.05 years (29). They suggest that if all countries met the World Health Organization Air Quality Guideline for $PM_{2.5}$ of 10 $\mu g/m^3$, median life expectancy could increase by 0.6 years (interquartile range of 0.2–1.0 year), a benefit of a magnitude that is similar to that of eradicating lung and breast cancer together. They report an average $PM_{2.5}$ for Mexico (i.e. the whole country) of 18.3 $\mu g/m^3$ which implies an average of 0.48 years life lost due to $PM_{2.5}$. Our results, based on analyses using direct alcaldía-specific information on air quality and health-related outcomes, adjusting for socioeconomic position indicators and proxy indicators of accumulated exposure to smoking, are consistent with world-wide most recent findings that indicate that air quality improvements have beneficial public health effects, measured as increased life expectancy and reduced life years lost. Therefore, public policies aimed at further improving air quality should be encouraged as they will continue to benefit public health.

PHASE IV. SPECIFIC CONTROL POLICY: CLEANING IN-USE HEAVY DUTY VEHICLES

Cost-effectiveness analyses of the technologies to control emissions of primary fine particles from heavy-duty diesel vehicles in Mexico City

We performed a cost-effectiveness analysis focused on heavy-duty diesel vehicle emission controls in Mexico City because of the potential adverse health impacts of diesel emissions to the atmosphere and the fact that the Air Quality Management Plan PROAIRE 2011-2020, specifically Strategy 3 Quality and energy efficiency in all sources, Measure 21, refers to the renewal of diesel vehicles by adopting emissions controls.

Diesel vehicles are a major source of air pollutant emissions, most importantly fine particles. The most recent inventory for Mexico City and the MCMA, Emissions Inventory, 2014 (30) (30), indicates that mobile sources account for 33% of total primary $PM_{2.5}$ emissions for Mexico City. Heavy duty diesel vehicles, despite their small share of the vehicle fleet (less than 6%), are responsible for 24% of primary fine particle total emissions.

We conducted cost-effectiveness analyses of control technologies for in-use heavy-duty diesel vehicles in Mexico City. The elements involved in our analysis can be summarized as follows (Figure 5.1.):

(I) Control efficiency for reducing baseline diesel particulate matter emissions;

(II) Impacts of emissions reductions on primary fine particle ambient concentrations and resulting reduction in population exposures;

(III) Health benefits from emissions controls and ambient air quality improvements, estimated as reductions in attributable mortality;

(IV) Costs of potential control technologies, i.e., cost of the equipment, installation, associated reduced fuel economy, periodic inspection and maintenance of the equipment; and

(V) Societal values in monetary units of health benefits with the estimated net benefit --comparison of benefits and costs.

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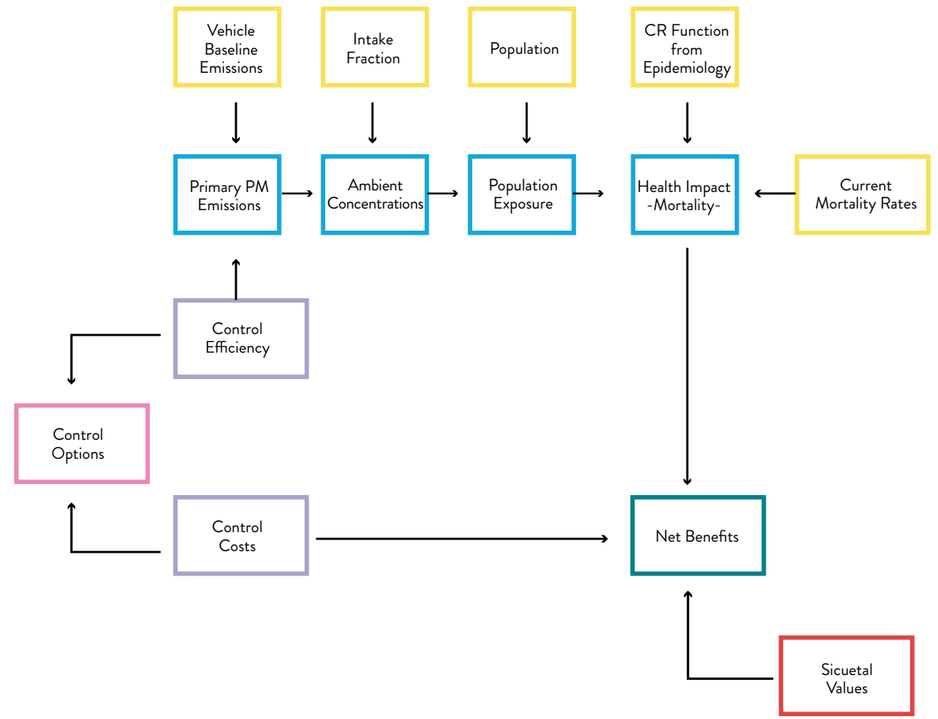

Figure 5.1. Conceptual model for the cost-effectiveness analysis to retrofit heavy-duty vehicles in Mexico City, 2014

In Mexico City there are over 100,000 in-use heavy-duty diesel-fueled vehicles, grouped in three main categories which are further divided in 10 classes:

- **Buses:** M1 public transportation, school and personnel, concession, and Metrobús, with local plates; and tourism and passenger with federal plates.

- **Trucks ≥ 3.8 tons:** Medium-sized delivery trucks with local plates (4.6 to 27.2 tons), or federal plates (11.8 to 14.9 tons).

- **Long-Haul Trailers:** Large vehicles, such as tractor trailers, and food supply vehicles weighing over 27.2 tons, with either local or federal plates.

License plates for heavy-duty vehicles may be local or federal, depending on whether they circulate only within the City or on highways that are under federal jurisdiction, regardless of the use of the vehicle, i.e. passengers, tourism or goods.

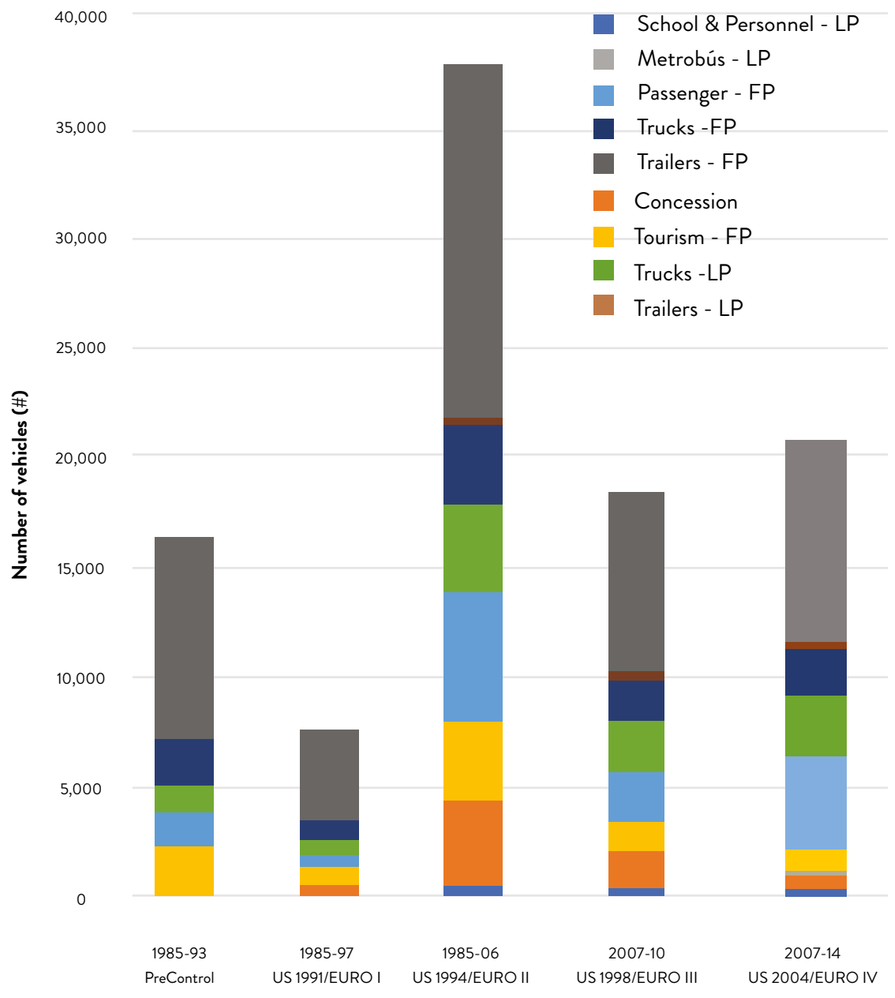

Figure 5.2. Composition of heavy-duty vehicles by class and model-year group

Our unit of analysis is a single vehicle. We evaluate representative vehicles from each vehicle class and model-year group --to span the range of vehicle types, uses and model years in the heavy-duty fleet operating in Mexico City. We include vehicles from model years 1985 to 2014 in the following model-year groups: 1985-1993 (pre-control); 1994-1997 (US 1991/Euro I); 1998-2006 (US 1994/Euro II); 2007-2010 (US 1889/Euro III); and 2011-2014 (US 2004/Euro IV). We exclude those older than 1984 because the 2014 Emissions Inventory, pools them in one category aggregating a wide range of technologies, and those that were retrofitted under the Autorregulación Program (n=45).

APPROACH

The analysis begins by characterizing each vehicle in terms of its nature (bus, truck, tractor trailer) and age (model-year group), its activity level (vehicle km travelled each year), its baseline emissions rates (g/km travelled) and fuel economy (km/L), and its remaining useful lifetime (yr). Data on age, activity and baseline emissions rates come from the official emissions inventory for 2014 (30), and data on fuel economy from U.S. Department of Energy (31).

Long-haul tractor trailers make up almost half of the fleet, most of them having federal plates. Buses account for about one third of the fleet, with two thirds of these having federal plates serving as tourism or passenger buses. Trucks, split equally between those with local plates and federal plates, account for the remaining 20% of the fleet. The heavy-duty diesel fleet is relatively old (Fig. 5.2.), with roughly 60% of the vehicles of more than 10 years old, 20% of more than 20 years old, and only 20% of vehicles belonging to the most recent model-year group.

Annual kilometers traveled within Mexico City vary considerably between within classes. Concession buses-local plate and long-haul trailers-federal plate account for the most vehicle kilometers travelled (VKT) with averages exceeding 16 million VKT. RTP buses-local followed in activity levels, with an average of almost 9 million VKT. In sharp contrast, the average activity levels for school & personnel buses-local plate and for long-haul trailers-local plate were only 800,000 and 440,000 VKT, respectively.

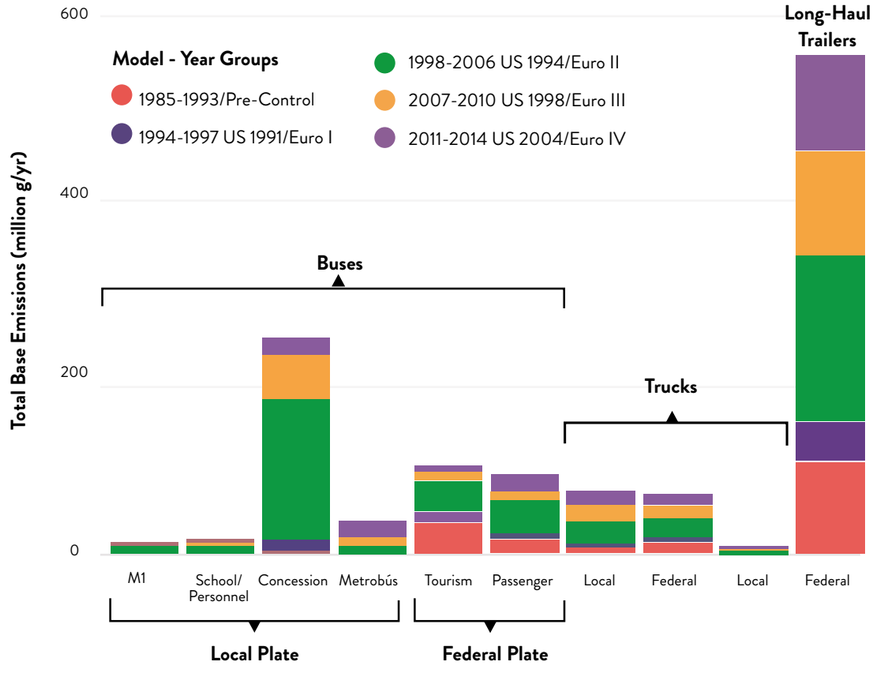

Figure 5.3. Annual Emissions of Primary Particles by Vehicle Class and Model-Year Group

The number of vehicles, age, and activity determine their emissions. For all vehicles, the total annual emissions of primary particles are close to 1000 metric tons. The largest emitters are long-haul trailers with federal plates (more than 50%), followed by concession buses with local plates (25%) (Figure 5.3.). The remaining 20-25% of primary particle emissions is roughly equally split between tourism and passenger buses with federal plates, and trucks (local and federal plates). Two categories of vehicles – school & personnel buses with local plates, and long-haul trailers with local plates make inconsequential contributions to primary particle emissions.

Our cost-effectiveness analysis considers four possible controls:

1) Diesel oxidation catalyst (DOC).

2) Diesel particulate filter, active regeneration (DPF-a).

3) Diesel particulate filter, passive regeneration (DPF-p).

4) A hypothetical control – i.e., one which is 100% efficient in reducing emissions of primary PM and which has no cost. The hypothetical control provides an upper bound on the net benefits of any possible emission-control technology.

As shown in Table 5.1. DPFs, active and passive, are more efficient in reducing PM emissions than are DOCs. All DPFs trap particulate matter and must undergo a process called "filter regeneration" to burn off captured particles (releasing carbon dioxide and water). This process cleans the trap and avoids clogging, which would result in high back-pressure affecting the engine performance. There are two different technologies to regenerate the filter –passive or catalyzed and active regeneration.

Diesel oxidation catalysts are easy to retrofit and maintain. Although DOCs are less expensive, they are much less effective at removing solid PM. DOCs remove fine particulate mass by oxidizing the soluble organic fraction of the particulate matter. DPFs and DOCs are likely to remain effective for the life of the vehicle, generally five to ten years or 10,000 or more hours of operation –they have been reported to maintain performance for as much as 10 to 15 years or for over 600 000 km.

Control Technology	Emissions Control Efficiency (%)	Cost ($USD)	Fuel Penalty (%)	Ultra Low Sulfur Diesel	Other requirements
Diesel Oxidation Catalyst	~ 20 to 25	~ 500 to 1,500	None	Not required	None
DPF-Active Regeneration	~ 80 to 90	~7,000 to 9,000	2	Benefits from	Increased exhaust temperature
DPF-Passive Regeneration		~6,000 to 8,000	0.4	Required	Some highway-speed driving

Sosurces: CARB Diesel Certification & Verification Procedure, and technology-specific corresponding Executive Orders (from 2013 to 2015).

Table 5.1. Main characteristics of retrofit technologies to control primary particle emissions in heavy-duty vehicles.

We have assumed that since ultra-low sulfur fuel is the only type of diesel fuel available in Mexico City that the introduction of retrofit technology has no impact on SO_2 emissions. Similarly, we assume that oxidation catalysts and diesel particulate have no impact on NOx emissions.

Catalyzed DPF are not compatible with pre-1994 Mexican diesel technologies and require ultra-low sulfur (ULS) fuel for reliable regeneration and optimal function. ULS diesel (≤ 15 ppm) has been available in Mexico City since 2009 but is not yet available in a large portion of the country where it may contain as much as 500 ppm sulfur. Therefore, vehicles that drive outside of the city (i.e. with federal plates) are not candidates for DPFs with catalytic regeneration. Active regeneration DPFs do not require ULS diesel. Most active DPF models are suitable for 1993 and newer vehicles, and to our knowledge only one model can be used in older models (pre-1993 vehicles).

Estimates of the capital costs and annual maintenance costs are taken from recent SEDEMA bids for diesel retrofit devices (32, 33), and estimates of the fuel use penalties for each control device came from MECA (1999)(34). The equivalent annual control cost for each device was computed by converting the capital cost to an equivalent annual cost stream using the capital recovery factor and adding the result to the annual maintenance cost and any additional cost related to the decreased fuel economy of vehicles equipped with DPFs. The discount rate used in our analysis was 3% per year.

To estimate the vehicle´s contribution to population exposures we used the intake fraction, which depends on all the variables that influence the relationship between emissions and exposure, such as the nature and location of the source, the pollutant´s physicochemical properties, the population receptor features, among other factors. Using intake fraction and emissions estimates, we calculated the city-wide average annual concentration change due to the emissions of the pollutant from each vehicle type under each type of control.

The impact on mortality of the reductions in air pollution exposure caused by emissions controls from a representative vehicle was computed using the integrated exposure response function (IER) applied in our risk assessment, and that was developed to support the Global Burden of Disease analysis [26]. We applied the IER for the five diseases that the GBD analyses determined as causally associated with long-term $PM_{2.5}$ exposure: ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease (COPD), and trachea, bronchus and lung cancers in adults, and, among young children, acute lower respiratory infections.

Here we rely on a linear approximation to the IER, since for small decrements in $PM_{2.5}$ the change in relative risk can be approximated by the product of the slope of the tangent to the IER evaluated at current levels of $PM_{2.5}$ in Mexico City --the annual average $PM_{2.5}$ level in 2014 was 22.8 $\mu g/m^3$ (35). Also, we introduced a cessation lag as the reduction of risk of diseases associated with $PM_{2.5}$ exposure reductions may start immediately (first year) and continue for some time (15 years).

The monetary value of the reduction in mortality risk is calculated by multiplying the population risk reduction (i.e., the reduction in deaths attributed to PM) times the rate at which mortality risk is valued, the Value per Statistical Life (VSL). Estimates for VSL resulted from recommendations to extrapolate values from the United States to countries lacking high quality estimates of VSL.

RESULTS

Emissions within the City lead to exposures and health risks in the City and throughout the metropolitan area, so the results consider the benefits in the Mexico City Metropolitan Area. Our results are presented for the status quo, for the three control technologies, and for a hypothetical control for each type of vehicle and model-year group. These results include emissions reductions, attributable deaths avoided, monetized benefits of the avoided deaths, control costs, and the overall measure of tradeoffs between benefits and costs, that is net benefits, per vehicle and per year.

	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/veh-yr)	Control Cost (1000 USD/veh-yr)	Net Benefits (1000 USD/veh-yr)
Bus Concession-Local Plate					
Status Quo	0.00	0.00	0.00	0.00	0.00
DOC	9.35	0.83	2.41	0.14	2.27
DPF -- Active	35.56	3.14	9.17	2.42	6.75
DPF -- Passive	35.56	3.14	9.17	1.43	7.74
Hypothetical Control	40.64	3.59	10.48	0.00	10.48
Long-Haul Tractor Trailer-Federal Plate					
Status Quo	0.00	0.00	0.00	0.00	0.00
DOC	2.68	0.24	0.69	0.09	0.60
DPF -- Active	10.18	0.90	2.63	1.06	1.56
DPF -- Passive	10.18	0.90	2.63	0.86	1.77
Hypothetical Contro	11.64	1.03	3.00	0.00	3.00

Notes: DOC stands for Diesel Oxidation Catalyst. Rows in green highlight the retrofit technology that maximizes the expected net benefits. The row in light gray highlights the retrofit technology that is not adequate for such vehicle category and model-year group.

Table 5.2. Results for Bus Concession – Local Plate and for Long-Haul Tractor Trailer – Federal Plate. Model Years 1998 to 2006 US 1994/Euro II

Table 5.2. gives illustrative results for the two largest emitter categories, bus concession with local plate and long-haul trailer with federal plate, for one model-year group (1998-2006 EU 1994/Euro II).

For the approximately 4 thousand concession buses with local plates, which are heavily used (each travel ~70 thousand km per year), the largest expected net benefits are generated by choosing to retrofit with a catalyzed DPF. The catalyzed DPF retrofit is expected to reduce emissions by 35.6 kg per vehicle-year, reduce premature deaths attributable to air pollution by about 3 per 1000 vehicle-year, with benefits of US$ 9.2 thousand and costs of only 1.4 thousand US$ per vehicle-year. The expected net benefits of this strategy (health benefits minus control costs) are almost 8 thousand US$ per vehicle year.

Retrofitting the approximately 16 thousand long-haul trailers with federal plates with a catalyzed DPF would yield the largest expected net benefits of almost 1.8 thousand US$ per vehicle-year. Unfortunately, the catalyzed DPF is not an option because these long-haul trailers with federal plates, are driven both in Mexico City and outside of the city, where ultra-low sulfur fuel is not widely available. The second-best option would be to retrofit with an active regeneration DPF, with the second largest expected net benefits of close to 1.6 thousand US$ per vehicle-year. Active DPFs generate the same emission reductions (10.2 kg per vehicle-year) and health benefits (1 per 1000 vehicle-year deaths attributable to air pollution) as the catalyzed DPF but are roughly 20% more expensive.

The control options that maximize the expected net benefits for all vehicles analyzed are presented in table 5.3. Note that there is no category or model-year group for which some retrofit is not cost-effective. We must add, that there is always uncertainty about the health benefits and costs of policies to reduce air pollution.

Our analysis quantifies uncertainty about some of the most important inputs, including the relationship between emissions (in this case emission reductions) and population exposure (summarized by the intake fraction), the slope of the exposure-response functions relating mortality to air pollution, the monetary value of reductions in mortality risk (summarized by the value per statistical life), as well as the efficiency and cost of control options.

Type of Vehicle & Plate		1985-93 Pre-Control	1994-97 US 1991/EURO I	1998-06 US 1994/EURO II	2007-10 US 1998/EURO III	2011-14 US 2004/EURO IV
Transportation Buses	RTP- Public Transport Local Plate	n.a.	n.a.	DPF-p 80	DOC 70	n.a.
	School and Personnel Local Plate	DPF-a 99	DPF-p 97	DPF-p 97	DPF-p 80	DPF-p 78
	Concession Local Plate	DPF-a 96	DPF-p 99	DPF-p 99	DPF-p 99	DPF-p 99
	Metrobús Local Plate	n.a.	n.a.	DPF-p 72	DPF-p 99	DPF-p 99
	Tourism Federal Plate	DPF-a 99	DPF-a 96	DPF-a 95	DPF-a 86	DPF-a 82
	Passenger Federal Plate	DPF-a 90	DPF-a 74	DPF-a 70	DOC 98	DOC 98
Delivery Trucks >3.8 tons	Trucks Local Plate	DOC 99	DPF-p 80	DPF-p 80	DPF-p 80	DOC 96
	Trucks Federal Plate	DOC 99	DOC 99	DPF-a 65	DPF-a 74	DPF-a 58
Long-Haul Tractor Trailers >27.2 tons	Trailers Local Plate	DOC 91	DOC 93	DPF-p 84	DPF-p 93	DPF-p 87
	Trailers Federal Plate	DPF-a 95	DPF-a 88	DPF-a 95	DPF-a 97	DPF-a 94

Notes: Vehicles are grouped in five model-year groups, except for RTP public transportation and Metrobús vehicles, which have vehicles than belong to only two and three model-year groups, respectively. Delivery Trucks > 3.8 tons with local plates weigh between 4.6 to 27.2 tons, those with federal plates weigh from 11.8 to 14.9 tons; local and federal plate long-haul tractor trailers weigh >27.2 tons. DOC stands for Diesel Oxidation Catalyst; DPF-p stands for Diesel Particulate Filter with catalyzed regeneration (passive), DPF-a stands for Diesel Particulate Filter with active regeneration, and n.a. stands for not applicable.

Table 5.3. Retrofit options which maximize expected net benefits by vehicle type and model-year group, and estimated probability (%) that net benefits of indicated retrofit options are positive, Mexico City, 2014

By doing so we can estimate the probability that the benefits of the reduction in mortality risk exceed the cost of the specified retrofit technology, that is, that the net benefits of the identified retrofit program are positive. These probabilities are displayed in Table 5.3. below the specified best control option. For most vehicle types and model-year groups, the probability that the identified retrofit option will yield benefits greater than its cost is 80 percent or larger. For vehicle categories and model-year groups with lower probability values, such as trucks - federal plate of model years 1998 and newer, such probabilities are tied to the selected control option –DPF active. However, this does not imply that these vehicles should not be controlled, since a much larger probability of 99% is estimated if retrofitted with oxidation catalysts.

INTERPRETATION

It is reasonable to ask what the aggregate benefits and costs of such a strategy would be. The fully implemented strategy of retrofitting every vehicle with the control which maximizes expected net benefits for that vehicle type and model-year group would result in close to 109 million US$ net benefits.

This strategy has the potential to:

- reduce annual emissions of primary fine particles by 950 metric tons.
- cut the annual population-weighted mean concentration of $PM_{2.5}$ in the Mexico City Metropolitan Area by 0.90 µg/m^3.
- reduce the annual number of deaths attributable to air pollution by close to 85, and.
- generate expected health benefits on the order of 250 million US$ per year.

The expected annual costs would be of less than 93 million US$ per year – consisting of 61 million US$ in 'amortization' of capital cost of retrofit devices; 19 million US$ in annual maintenance costs; and 11 million US$ in fuel use penalties.
Retrofit programs have been put in place in other countries and have been on the radar of policy makers in Mexico for decades. Diesel retrofit technologies, such as DOCs and DPFs, can reduce diesel particulate matter with similar control efficiencies to emission controls from newer diesel vehicles [36].

In Mexico City, a retrofit program was put in place over 10 years ago. Two fundamental lessons were learned as key to the success of the program: Selecting appropriate buses for retrofitting through previous careful testing, and training operators on how the emissions control devices worked, how they were installed, and driving techniques for best performance of the equipment.

Retrofitting the heavy-duty diesel vehicle fleet would represent a small, but important, step towards further improvement of air quality in Mexico City. We encourage authorities in Mexico City to consider moving forward with the design and implementation of such a program.

FINAL REMARKS

Reductions in $PM_{2.5}$ and ozone over the past twenty-five years have led to substantial improvements in public health in Mexico City. We have measured health benefits from enhanced air quality as life expectancy gains, life years increased by five causes of death determined to be causally associated with fine particles or ozone, and as reductions in attributable mortality for these five causes of death. We conducted a population-based epidemiological analysis and a risk assessment to quantify and value such health benefits.

By reducing city-wide average ambient $PM_{2.5}$ concentrations from 35 μg/m^3 in 1990 to 20 μg/m^3 in 2015 and simultaneously reducing ambient ozone concentrations from over 160 ppb in 1990 to close to 84 ppb in 2015, Mexico City has been able to increase life expectancy, increase life years lived attributable to certain diseases, and reduce attributable deaths associated with air pollution. Our risk assessment shows that deaths attributable to fine particles and ozone during this 25-year period were reduced by 22.5 thousand (95% CI: 17.9 to 28.0 thousand). Roughly 18.0 thousand of these avoided deaths are due to improvements in $PM_{2.5}$ (95% CI: 14.0 to 23.5 thousand), and 4.0 thousand to ozone (95% CI: 2.7 to 5.6 thousand).

Our findings are consistent with state of the art knowledge in that long-term exposure to fine particles and ozone are related with chronic diseases that mainly affect adults. For the population between 25 and 74 years old, we found that a decrease of 10 μg/m^3 in the annual average concentration of $PM_{2.5}$ was associated with an increase in mean years of life of 0.56 (95% CI 0.28 to 0.83) years. Also, a decrease of average 1-hour peak seasonal ozone levels was associated with an increase in mean years of life of 0.10 (95% CI 0.03 to 0.17) years. Our risk assessment, also in agreement with the above, showed that most attributable deaths avoided due to air quality improvements in the last 25 years were among adults older than 25 years of age. Over 65% of avoided attributable deaths were among adults between 25 and 74 years old, and only 3% were among those of less than 19 years old.

Epidemiological analyses of data from the United States have reported for that improvements of 10 μg/m^3 in average annual fine particle concentrations are associated with an increase in life expectancy of 0.61 (95% CI 0.22 to 1.00) years (27), results that are very similar to our adult findings from Mexico City.

We also found a significant increase of life gained attributable to ischemic heart disease in adults over 25 years old of 0.094 (95% CI 0.027 to 0.160) years (equivalent to 34 days), associated with a decrease of 10 μg/m^3 in the annual concentration of $PM_{2.5}$. Our analyses using risk assessment methods, very consistently indicate that around 10 thousand attributable deaths due to ischemic heart disease were avoided because of improved fine particles levels in the past 25 years.

Results for life gained attributable to chronic obstructive pulmonary disease, among the population of over 25 years of age, showed a positive and significant increase of 0.037 (95% CI 0.007 to 0.067) years with improved annual $PM_{2.5}$ concentrations (10 μg/m^3). A positive, but non-significant association was found for ozone (P=0.14). Our risk assessment indicated that 6.5 thousand attributable deaths due to COPD were avoided as fine particles and ozone levels decreased in the city since 1990.

For life gained due to lung cancer and cerebrovascular stroke there were positive but marginally significant (p<0.10) associations with $PM_{2.5}$. These potential associations should be further explored in other studies.

Among children aged 0 to 4 years we found a modest, statistically non-significant increase in years of life lost between ages 0 and 4 years associated with $PM_{2.5}$ (0.0070 years or 2.5 days) and a significant small increase with O_3 (0.0037 years or 1.3 days). We found no positive association with years of life lost from acute lower respiratory infections (ALRI). Results from our risk assessment revealed that less than 3% of attributable deaths were among the youngest stratum of the population.

For our 25-year study period there was a life expectancy at birth increase of 1.3 and 1.9 years due to $PM_{2.5}$ and O_3 air quality improvements, respectively. With annual average $PM_{2.5}$ concentrations and seasonal hourly ozone peaks improving by close to 15 μg/m^3 and 77 ppb, correspondingly, and the estimated effects for each pollutant in our model, we calculate a net increase of almost 3.2 years in life expectancy for the population of Mexico City. Similarly, there was an important increase in temporary life expectancy for adults aged 25 to 74 of 1.6 years, with almost equal contributions by $PM_{2.5}$ and ozone of roughly 0.8 years each. Other factors likely contributed simultaneously to such improvements and they were accounted for in our models, which controlled for socioeconomic position indicators, proxy indicators for smoking, and for reference diseases.

Estimates from other countries and globally using indirect methods, i.e. with either cohort risk estimates or with integrated exposure-response functions, find that changes in life expectancy are associated with long-term changes in PM2.5 and O3 exposures. In the United States, an increase in life expectancy was esti-

mated to be between 0.35 and 0.61 years with an exposure reduction of 10 μg/m^3 (27, 28). Current global $PM_{2.5}$ and O_3 long-term exposures have been associated with a decrease in life expectancy of 1.03 years and 0.05 years respectively (29). The authors report that the potential benefits of reducing current $PM_{2.5}$ to levels that would meet the World Health Organization's guideline would be of a similar magnitude to the benefits of eliminating lung and breast cancer together. For Mexico (countrywide), they also indicate that current estimated $PM_{2.5}$ levels reduce on average 0.48 years of life lost.

The results of our epidemiological analyses and our risk assessment are an incentive to further improve air quality. This study shows that public policies that aim at improving air quality benefit public health, with gains in life expectancy and reductions in attributable mortality in large populations.

The cost-effectiveness analysis conducted for Mexico City heavy-duty vehicles clearly shows that performing retrofit with either Diesel Oxidation Catalysts (DOCs) or with Diesel Particulate Filters (DPFs) can reduce particulate matter emissions, lead to improvements in air quality, and have public health benefits among the inhabitants of the Mexico City Metropolitan Area.

For the three vehicle categories responsible for the greatest share of primary PM emissions, bus concession - local plate, long-haul trailer - federal plate, and bus tourism - federal plate, DPF retrofits, which have expected emissions reductions between 80 and 90%, provide the maximum possible expected net benefits for all model-year groups.

For other vehicle categories such as bus passenger - federal plate, the fourth largest primary PM emitter, and trucks with local or federal plates, DPFs are not cost-effective for some model-year groups, but oxidation catalysts are, for which projected emissions reductions range between 20% and 26%.

PROAIRE, 2011-2020 **Strategy 3.** **Energy Quality and Efficiency in all sources**	**Strategic Priorities**
Measure 21. Renewal of diesel vehicles with motor substitution and by adapting emission controls. ***Action 21.1.*** *Design a program to replace diesel motors that have been in use for 10 years or more, and adapt emissions control equipment.*	• SEDEMA´s Institutional Program, 2012-2018 • Mario Molina Center for Strategic Studies on Energy and Enviroment • Scientific Advisory Board of the Environmental Commission of the Megalópolis • Center of Atmospheric Sciences, UNAM

Table 6.1. Strategic Priorities for Air Quality Management in the MCMA: PROAIRE 2010-2020, Institucional SEDEMA's Program and other institutions in Mexico

If every vehicle were retrofitted with the control which maximizes the expected net benefits the aggregated net benefits would be of close to 109 million US$. Such a strategy could potentially generate expected health benefits on the order of 250 million US$ per year. Annual emissions of primary fine particles would be reduced by 950 metric tons, the annual population-weighted mean concentration of $PM_{2.5}$ in the Mexico City Metropolitan Area would decrease by 0.90 μg/m^3, and close to 85 less annual deaths attributable to air pollution would be expected.

The importance of cleaning the heavy-duty fleet in Mexico City has been recognized by experts and authorities in Mexico and Mexico City. Mexico's City and MCMA Air Quality Management Plan, PROAIRE 2011-2020, lays out a strategy and corresponding measure to clean heavy-duty diesel vehicles, either by substitution of engines of by retrofitting control technologies.

We close by noting that this one small step must be viewed from the wider perspective suggested by the air quality management program in place and by the Mario Molina Center's 2016 position paper on air quality in the Mexico City Valley (37) (Table 6.1). As these documents suggest, in addition to reducing emissions from heavy-duty vehicles, many other programs and strategies -- such as the development of an integrated public transportation system, the promotion of the rational use of cars, the reduction of emissions from industrial sources and fires, and redesign of the MCMA area to reduce urban sprawl -- must be analyzed and implemented to make significant strides forward in the control of air pollution and its public health impacts.

MAIN MESSAGES

- Air quality has dramatically improved over the last 25 years in Mexico City.
- Air quality improvements have saved 22,000 lives mainly due to reductions in $PM_{2.5}$ levels.
- Despite air quality improvements, current $PM_{2.5}$ and O_3 levels in Mexico City are still above those which lead to health effects.
- Heavy duty in-use diesel fueled vehicles are a major emissions source of $PM_{2.5}$.
- Particle emissions controls of heavy-duty vehicles (retrofits) can save more than 80 lives per year with net benefits of over $ 150 million USD, monetized health benefits of almost $ 250 million USD, and annual costs of $93 million USD.
- Our local epidemiolocal study has confirmed the mortality estimates used in the heavy-duty diesel fueled retrofit cost-effectiveness analysis.
- Our epidemiological analysis has shown that people in Mexico City live on average 3.2 more years due to air quality improvements.
- This study suggests that ozone is more important than international evidence currently suggests.
- These findings are important in Mexico City and for the Megacities throughout the World.

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Historical Analysis of Population Health Benefits Associated with Air Quality in Mexico City during1990 and 2014

Phase I. State of Knowledge and Relevance to Mexico City
Phase II. Estimation of the Health Benefits of Air Pollution Improvements

Final Report

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I. Executive Summary

The Secretaries of Environment (SEDEMA) and Health (SEDESA) of the government of Mexico City[1] initiated a program of collaboration with the TH Chan Harvard School of Public Health in 2015. The program, with focus in Mexico City's air quality, consists of four phases, to be conducted over a four-year period, which include a review of the state of knowledge relevant to Mexico City (Phase I); an estimation and verification of the health benefits of air quality improvements, first through risk assessment (Phase II), and in a following phase through epidemiological methods (Phase III); and the public policy and economic valuation of the health impacts (Phase IV).[2]

This report includes the first and second phases of work. It consists of a literature review of the state of knowledge and a description of the scientific evidence with the analysis of the most solid epidemiological studies to day, which are to be relied on when interpreting the relationship between air pollutants exposures and adverse health outcomes. Secondly, this report characterizes the methods and estimates from the risk assessment that allow to calculate the health benefits attributable to the reductions in fine particulate matter or $PM_{2.5}$ (also known as respirable fraction, which have an aerodynamic diameter ≤ 2.5 microns, μm) and ozone concentrations that have been achieved, as a result of public policy strategy implementation, from 1990 to 2014 in Mexico City.

Public policy strategies that aim at reducing air pollution diminish premature mortality risk in large populations, and so, the individuals that are affected are unknown. For this reason, we use the number of 'premature deaths avoided' to evaluate the health benefits attributable to air quality improvements. It is important to recognize that lives cannot be saved by air pollution controls or any other public policy intervention. Lives

[1] Previously called Federal District, and since 2016 called Mexico City. The Federal District and now Mexico City are conformed by 16 boroughs (called *delegaciones*, and will be eventually called *alcaldías*).

[2] This program of collaboration with the Government of Mexico City is confined to the *delegaciones* (boroughs) that conform Mexico City.

can merely be extended. Thus, the metric we use is, in fact, a *proxy* for "life expectancy increases".[3]

Phase I. State of Knowledge and Relevance to Mexico City

Air quality in Mexico City (CDMX)in the late 1980s and early 1990s exhibited the worst in its history. Most criteria pollutants (lead, sulfur dioxide, carbon monoxide, nitrogen dioxide, ozone, and particulate matter) frequently exceeded national ambient air quality standards. Since then, legal powers have been used to control and reduce air pollutant emissions, based on the faculties that are attributed to municipal, state and federal governments, to improve air quality and protect public health (Flamand and Rojas-Bracho, 2015)[4]. Public policy actions have been implemented, including federal initiatives such as standard setting to regulate emissions of mobile and point sources, improvements in fuel quality – eliminating leaded gasoline in the late 1990s and gradually reducing sulfur contents from gasoline and diesel beginning in the early 90s continuing even today- and establishing air quality maximum permissible levels for criteria pollutants.

Also, the Government of Mexico City has instrumented a series of comprehensive air quality management programs, known as ProAire. These have been developed in coordination with federal authorities, representatives from academia, and the private sector. In addition, several important public policy specific strategies have been launched, among them The Environmental Contingencies Program (PCAA) in 1988, and one year later the *Hoy No Circula*, and the Inspection and Maintenance

[3] In risk assessment and policy analysis the terms "premature deaths avoided", "reduced mortality risk", "reduced premature mortality", "avoided deaths", "excess deaths" and "lives saved" are used to refer to the benefits derived from strategies that target air pollution reductions. For the present project we use the term "premature deaths".

[4] See the General Law on Ecological Equilibrium and the Protection of the Environment (LGEEPA) (*Capítulo II. Distribución de Competencias y Coordinación*), and corresponding secondary rulings for the control and prevention of air pollution (*Reglamento para la Prevención y Control de la Contaminación de la Atmósfera (Capítulo I. Disposiciones Generales*).

Programs, among other. These programs have been modified in numerous occasions.

These programs have been intertwined and used to promote the fleet renewal to accelerate the entry of more advanced pollution control technologies, and the continued maintenance of vehicles that circulate in Mexico City, and in the neighboring urbanized area of the State of México. Such strategies have been based in assigning privileges to new vehicles, less frequently required inspections and the right to circulate every day, versus the lack of privileges to older cars which need to inspect twice a year and aren't allowed to circulate once or twice a week (contingent on model-year). In turn, the PCAA seeks to trigger corrective actions to reduce pollutant emissions -including banning circulation of certain vehicles- and the exposure of sensitive population sub-groups to air pollutants when levels of ozone or PM_{10} (particulate matter ≤ 10 μm in aerodynamic diameter, also known as inhalable fraction) exceed a certain threshold. The threshold for ozone has been reduced importantly through the years, from about 2.5 times the 1-hour ambient air quality standard when the program first entered into force, to 1.5 times the value of the current standard (which was tightened in 2014) in July of 2016. It must be added that, in spite of having a lower threshold level, these high pollution events called *Contingencias* have decreased importantly since the program was first launched. For instance, in 1992 and 1993 there were 33 and 14 *Contingencias*, respectively. In clear contrast, there were no *Contingencias* between 2006 and 2014.[5]

Sizeable improvements in air pollution have been observed in Mexico City. Concentrations of $PM_{2.5}$ (expressed as annual averages of daily 24-hour concentrations) have decreased from between 30 and 66 micrograms per cubic meter ($\mu g/m^3$) in 1990[6] to less than 25 $\mu g/m^3$ in 2015 -as measured by Mexico City

[5] The most recent years that are directly comparable in terms of number of *Contingencias* are 2012 to 2014, since they shared the same threshold to activate a *Contingencia* and the same value of the 1-hour ambient air quality standard. No *Contingencias* were triggered in those years. It was only in 2016 that ten *Contingencias* were triggered, when more stringent values for both, the threshold and the ambient air quality standard were in place.

[6] As $PM_{2.5}$ was not directly measured in the City but after the end of 2003, concentrations prior to this year were estimated by means of a predictive Generalized Additive Model developed for this project,

environmental authorities. Ozone concentrations (expressed either as annual or as seasonal (six month) daily 1-hour maxima[7]) have decreased from between 85 and 185 parts per billion (ppb) in 1990 to less than 92 ppb in 2015 -computed from official data from fixed-site monitoring stations.

Today, there is robust evidence regarding the adverse health impacts of ambient air pollution. This evidence stems primarily from epidemiological studies, mostly time-series and cohort study designs. These complement each other since together the adverse health effects are evaluated for short-term and long-term exposures. In Mexico City time-series studies, conducted since the early 1990s, have been the dominant epidemiological design to evaluate the relationship between air pollutant exposures and adverse health impacts (Borja-Aburto et al., 1997; Borja-Aburto et al., 1998; Loomis et al., 1999; Castillejos et al., 2000). These studies reported associations between particle and ozone exposures and total mortality, and between ozone and cardiovascular mortality. Sensitive populations sub-groups were identified, people over 65 years old being more sensitive to ozone exposures, and infants to particle exposures.

The most recent times-series analysis, ESCALA (Study of Air Pollution and Health Effects in Latin America) was conducted as a multicity project -- that included Mexico City -- to estimate all-natural cause, cause-specific and age-specific daily mortality associated with daily exposures to PM_{10} and to ozone (Romieu et al., 2012). This study found positive associations between daily levels of PM_{10} and all-cause mortality, and the highest risk reported was for chronic obstructive pulmonary disease mortality. Ozone was more weakly associated with increased all-cause mortality than were particles.

Even considering the differences and regional variations in the quantitative relationship between air pollution and mortality that have been found among time-

that includes as predictors PM_{10} concentrations, meteorological, spatial and time variables. These numbers reflect the range of values estimated for five monitoring stations used to develop the model.

[7] Seasonal maxima of daily 1-hour maxima ozone concentrations is the exposure metric used in epidemiological cohort studies and will be used repeatedly in this report.

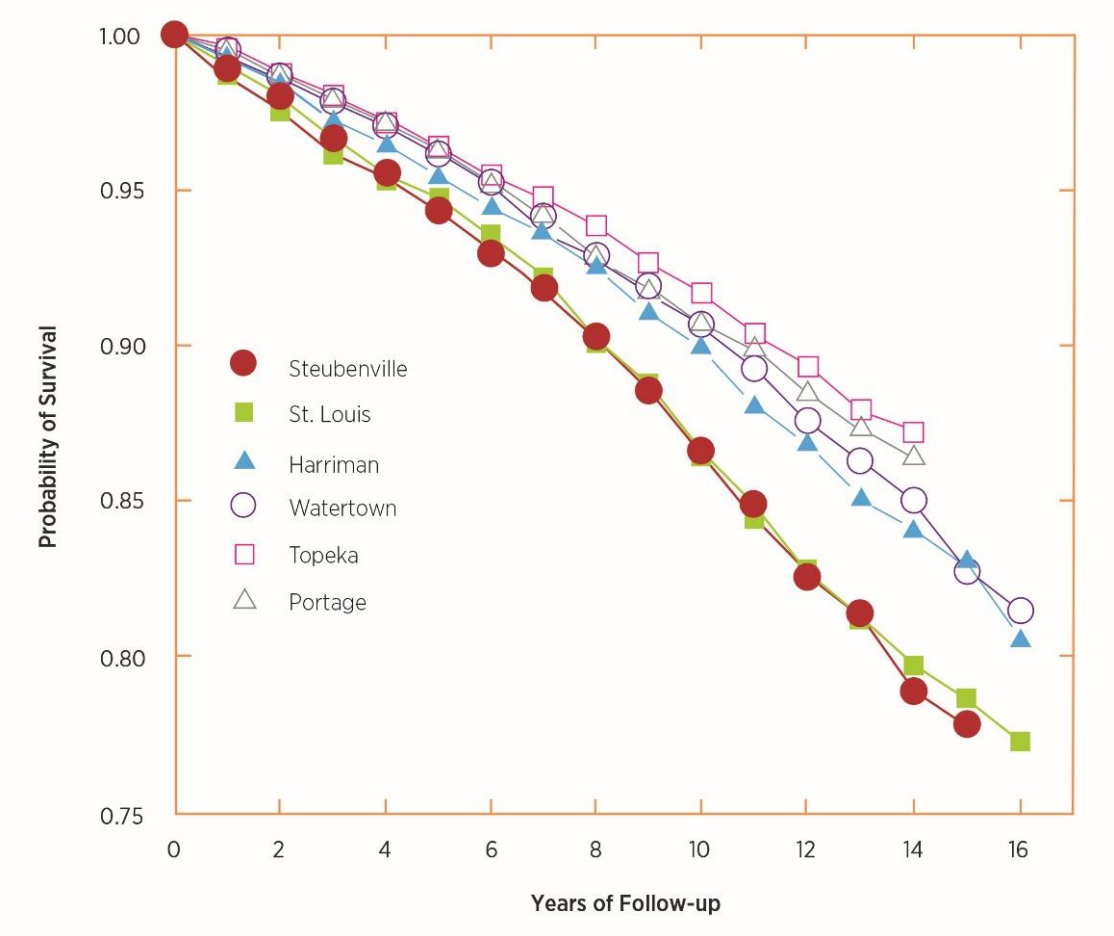
series studies conducted in cities around the world, the scientific consensus is that daily fluctuations in particulate matter and ozone have an adverse impact on daily mortality (Atkinson et al., 2014).

In Mexico no epidemiological studies to evaluate the long-term health effects associated with chronic air pollution exposures have been conducted. For this reason, we will describe the most relevant evidence from cohort studies conducted elsewhere.

The first cohort study to examine the mortality impacts of air pollution exposure was the Harvard Six Cities Study (Dockery et al., 1993). The hypothesis of the study was that individuals living in cities with higher levels of $PM_{2.5}$ air pollution would experience higher rates of cardiovascular, respiratory, and lung cancer mortality. Results indicate that for $PM_{2.5}$ exposures survival was lower and mortality rates higher in the dirtiest city than in the cleanest one (Fig. I.1). For every 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations, mortality rates increased by approximately 1.5%.

This study was soon followed by a larger cohort study, the American Cancer Society (ACS) study (Pope et al., 1995). Consistent with the Six City study, the ACS also found an association between $PM_{2.5}$ concentrations and mortality. However, the size of the effect was about one third smaller, for each 1 $\mu g/m^3$ increase in ambient levels of $PM_{2.5}$ mortality rates would show an increment of about 0.4%. The difference in the size effect between the two studies may be partly explained because the ACS cohort is more than 50 times larger, triples the number of deaths that occurred during the study period; includes white, black and Hispanic subjects (not only white participants); and, improves the statistical analysis and design to control for certain risk factors.

Figure I.1. Six Cities Study: Crude probability of survival vs. years of follow-up



Source: Dockery et al., 1993.

The Six Cities and the ACS study have been vetted thoroughly and have been extended to include prolonged periods of follow-up, that have increased the number of deaths that occurred during the periods under study, and the statistical power of the analysis (Lepeule et al., 2012; Krewski et al., 2009). During the extended follow-up periods, air quality improved in the cities included in these cohorts, and the authors found that mortality was reduced and life expectancy was extended. This is relevant for our project in Mexico City, given the better air quality that we have today relative to pollution levels found in the 90s.

The qualitative consistency of results from these studies is noteworthy. Both found that cardiovascular mortality (a broad category that includes ischemic heart disease and cerebrovascular stroke) and lung cancer mortality are associated with long-term $PM_{2.5}$ exposures. Also, the concentration-response function was found to be nearly linear within the range of concentrations observed in the cities included in each study -from 5.8 to ~30 $\mu g/m^3$ in the ACS, and from 8 to ~30 $\mu g/m^3$ in the Six Cities (Lepeule et al., 2012; Pope et al., 2002; Krewski et al., 2009).

For ozone, only the ACS found a significant association with mortality, because there was a broader range of ozone exposures in the cities that were included in this cohort. The association between seasonal (six month) 1-hour maximum concentrations and mortality was preserved when controlling for $PM_{2.5}$, and the primary effect was on respiratory causes of death (Jerrett et al., 2009).

Several new cohort studies have been conducted in the United States, Europe, Asia, and Oceania. Results have been qualitatively consistent, although there is heterogeneity among their estimated risk coefficients. This coefficient variability arises because each study yields a concentration-response relationship for a different population sample (for instance, sub-groups with pre-existing medical conditions or particular trades), in addition to differences in analytical methods as well as in the elements that comprise the causal chain of the exposure-response relationship. By means of a meta-analysis that evaluated over a dozen cohort studies summary coefficients show that a 1 $\mu g/m^3$ increase in annual average $PM_{2.5}$ concentrations is associated with a 0.6% increment in all-cause mortality, and an 1.1% increment in cardiovascular mortality (Höek et al., 2013).

At present, in Mexico as in most countries around the globe, major regulatory projects, and the burden of air pollution are assessed via health benefit analysis (and frequently complemented with cost estimates). Health benefits are estimated by means of risk assessment methods, and use exposure-response functions from cohort studies of ambient air pollution. In Mexico City the support to use effect estimates derived from cohort studies conducted in other countries for risk

assessment purposes stems principally from the consistency between locally and internationally generated evidence from the short-term exposure studies (HEI, 2012).

Phase II. Estimation of the Health Benefits of Air Pollution Improvements in CDMX, 1990-2014

Air Pollution as a Risk Factor Worldwide and in Mexico

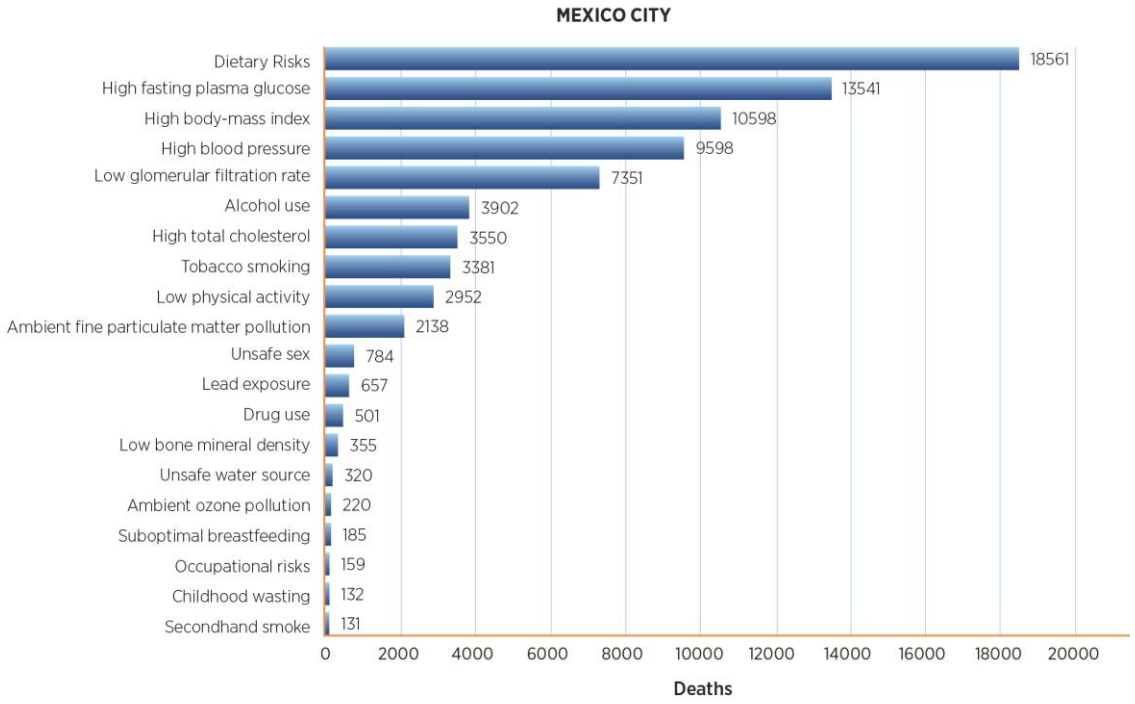
The impact of air pollution exposures on premature mortality can be measured as "premature deaths" when the assessment refers to the adverse health impacts of air pollution or as "premature deaths avoided" when the assessment refers to the health benefits of air quality improvements. Risk assessment and burden of disease methods have been applied for this purpose globally and locally.

Recently, a worldwide effort known as the Global Burden of Disease (GBD), found that ambient $PM_{2.5}$ and ozone air pollution are ranked in the 10^{th} and 21^{st} positions among the nearly 70 risk factors analyzed for 2010 and 2013 (Lim et al., 2010; Forouzanfar et al., 2015). The GBD and Lelieveld and coauthors (2015) found that $PM_{2.5}$ exposures cause around 3 million premature deaths (GBD 95% uncertainty intervals: 2.6 million to 3.6 million premature deaths). For ozone, the GBD estimated approximately 220 thousand premature deaths (95%UI: 160 thousand to 272 thousand premature deaths) (Lim et al., 2013; Forouzanfar et al., 2015; Lelieveld et al., 2015). The GBD assessments and Lelieveld and coauthors (2015) show that exposure to $PM_{2.5}$ causes predominantly premature deaths from ischemic heart disease, cerebrovascular stroke, and lung cancer, whereas exposure to ozone is related with chronic obstructive pulmonary disease.

Few risk assessments have been conducted for Mexico or for Mexico City to assess health impacts of air pollution. Results have shown that $PM_{2.5}$ chronic exposures are responsible for 7,600 annual premature deaths per year in Mexico (Stevens et al.,

2008). For the Metropolitan Area of the Valley of Mexico (MAVM)[8] roughly 3,000 premature deaths were related with chronic exposures to $PM_{2.5}$, and for Mexico City, 6,100 premature deaths were attributable to PM_{10} chronic exposures (Stevens et al., 2008; Riojas-Rodríguez et al., 2014).

Figure I.2. Main risk factors and associated premature deaths for Mexico City in 2013



Source: Prepared by the authors with information from IHME, 2016.

The GBD 2010 and 2013 studies analyzed the per-country and per-state burden of disease, including Mexico and Mexico City. For Mexico over 13,000 premature deaths could be attributed to $PM_{2.5}$ chronic exposures, and close to 2,000 to chronic

[8] The definition of the Metropolitan Area of the Valley of Mexico that prevailed until 2005, period during which this study was conducted (2001 to 2005), included 16 boroughs of the then called Federal District (called *delegaciones*), and 18 boroughs of the State of Mexico (called *municipios*). The definition was officially modified after December 2005 to include 59 boroughs from the State of Mexico, 1 from the state of Hidalgo, and 16 from the Federal District (INEGI, 2016).

ozone exposures (IHME, 2016). For Mexico City, $PM_{2.5}$ and ozone are responsible for about 2,100 and 220 premature deaths annually, respectively (IHME, 2016).[9] In Mexico City ambient exposures to $PM_{2.5}$ and ozone are among the first 20, out of 70, risk factors that were evaluated (Fig. I.2).

Methods for Estimating the Health Benefits from Improved Air Quality in Mexico City

The risk assessment is the method we relied on to estimate the benefits associated with air quality improvements in Mexico City in the past 25 years. Risk assessment comprises four elements: to analyze the stressor (***hazard identification***), characterize quantitatively its contact with humans (***exposure assessment***), quantify the relationship between the exposure and the risk of an adverse health effect (***concentration-response***), and, lastly, integrate all of the above to provide a final estimate of the health risk and the uncertainty associated with it (***risk characterization***).

Specifically, benefits from reductions of ambient fine particulate matter and ozone are examined for the 1990 – 2014 period by analyzing air quality improvements achieved every year for every *delegación* (Mexico City is divided into sixteen *delegaciones* for administrative purposes). Therefore, we first assess the exposure of the population of Mexico City for the study period, and then calculate how many premature deaths were avoided (benefits) due to better air quality in the City.

The basis for this risk assessment is air pollution data and mortality counts officially available for Mexico City. SEDEMA and SEDESA provided air quality and mortality data, respectively. Our exposure assessment metrics are in agreement with those from the cohort studies that have yielded the most robust relative risk estimates for the relationship between chronic exposure to $PM_{2.5}$ and ozone and mortality. For

[9] Such studies compared estimated population exposure levels to an alternative concentration under which no health effects had been observed in epidemiological studies (Stevens., et al., 2008, and the GBD, Lim et al., 2013; Forouzanfar et al., 2015), or to the World Health Organization recommended values (Riojas-Rodríguez., et al., 2014).

$PM_{2.5}$ we use annual average $PM_{2.5}$ concentrations, and for ozone, seasonal (six month)[10] averages of daily 1-hour maximum levels. These exposure metrics are calculated using data from monitoring stations and then are spatially interpolated to each *delegación*, to match the spatial resolution of official mortality data –needed for the next steps of the risk assessment.

Our risk assessment was conducted from 1990 to 2014. Air pollution data has been routinely monitored by SEDEMA, $PM_{2.5}$ since the end of 2003 and ozone before 1990. Fine particle concentrations were estimated for the earlier years of our analysis (1990 – 2003) by means of a Generalized Additive Model (GAM). The GAM included predictive variables such as PM_{10} (PM_{10}, in contrast with $PM_{2.5}$, has been routinely monitored since the late 1980s), meteorological variables (wind speed, relative humidity and temperature), monitoring station, year and month (as a proxy for season).[11] We used $PM_{2.5}$ and PM_{10} data from the five fixed-site monitoring stations that have consistently used High Volume Air Samplers (Federal Reference Method). An alternative model was evaluated to include SO_2 and NO_2, however, missing data precluded its use –missing data would have reduced by almost two thousand the number of 24-hour predicted values, that were then used to estimate the annual averages per year and per station.

To compute our ozone exposure metric all available daily data from fixed-site monitoring stations was used. The number of monitoring sites with available data varied yearly, starting with less than six sites in the early nineties, then increasing to over a dozen, and finally exceeding 20 since 2012.

[10] For consistency, this project defined "seasonal" as closely as possible as the ACS cohort study, because we base our risk assessment for ozone on the relative risk derived from such study. The ACS study defined "seasonal" as the warmer months that tend to have higher ozone concentrations (April-September) (Jerrett et al., 2009). For this project we defined "seasonal" as the period between February 1st and July 31st, in order to include the highest ozone months (March-June), adding one month before and one after, and at the same time exclude the months that have had historically the lowest ozone concentrations (September-December). This is in line with the definition of the "ozone season" by the Environmental authorities from Mexico City, which runs from the second week in February through June, when the rainy season begins.

[11] The GAM that we developed and selected to predict $PM_{2.5}$ 12-hour concentrations, had an adjusted R-square equal to 0.73, and available data allowed to predict 6761 24-h $PM_{2.5}$ concentrations (equivalent to an average of 54 daily concentrations per station on an annual basis).

We found that annual average $PM_{2.5}$ concentrations[12] for the early 1990s ranged from around 30 to over 65 µg/m^3. After 2007 there was an average decrease of about 20 µg/m^3 and the downward trend leveled off since 2008. From 2010 onwards a slight increase of less than 10% has been observed. For ozone, the early 1990 were characterized by extremely high concentrations, with seasonal averages of daily 1-hour maxima in the range of between 85 and 200 ppb levels. By the late 1990 seasonal average daily 1-hour maxima highest value was slightly below 160 ppb. The lowest seasonal 1-hour maximum level was reported in 2012 (67 ppb).

Fine particle and ozone exposure metrics (average values and their corresponding standard error of the mean) that were estimated for each monitoring station were interpolated spatially to the *delegación* level. The analysis was first conducted at a much finer spatial resolution – the basic geostatistical area (known as an AGEB) within an *delegación*. The values assigned to each AGEB were determined by how close the AGEB was to one or more of the $PM_{2.5}$ or ozone monitoring sites. More specifically, the methods applied were Proximity Analysis (nearest monitor) and Inverse Distance Weighting (IDW), using weights proportional to 1/distance^2. We restrained the area of influence of the Xalostoc monitoring site on surrounding *delegaciones*, since it has a micro-scale spatial representativeness (according to the definition of the US-EPA) which implies that the very high concentrations monitored at this station are impacted by local sources and are unlikely to represent the exposure of populations that are not in the immediate vicinity.[13] For that purpose, Xalostoc $PM_{2.5}$ concentrations were estimated using weights proportional to 1/distance^3 with a probability of 2/3, and no weights were applied with a probability of 1/3.

Figure I.3. Estimated annual average $PM_{2.5}$ concentrations at the *delegaciones* of Mexico City from 1990 to 2015

[12] The GAM model predicted daily $PM_{2.5}$ concentrations were used for the entire 1990-2014 period under study. These are meteorology-adjusted concentrations.

[13] Personal communication with Armando Retama, Director of Atmospheric Monitoring, General Direction on Air Quality Management, SEDEMA, Government of Mexico City.

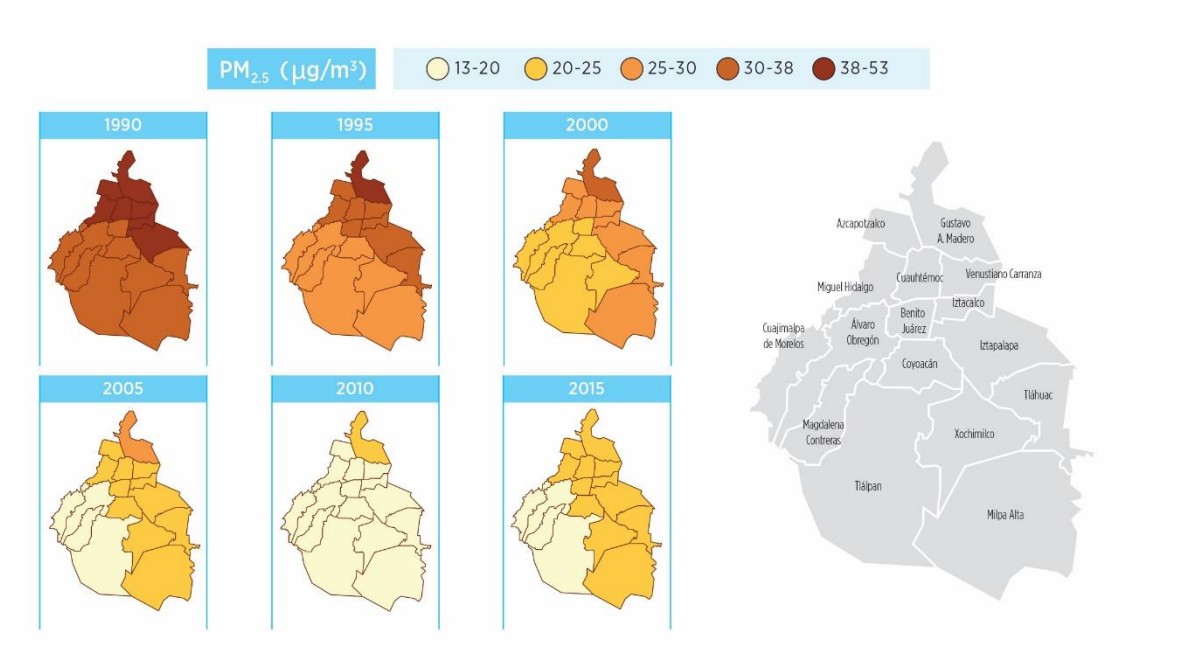
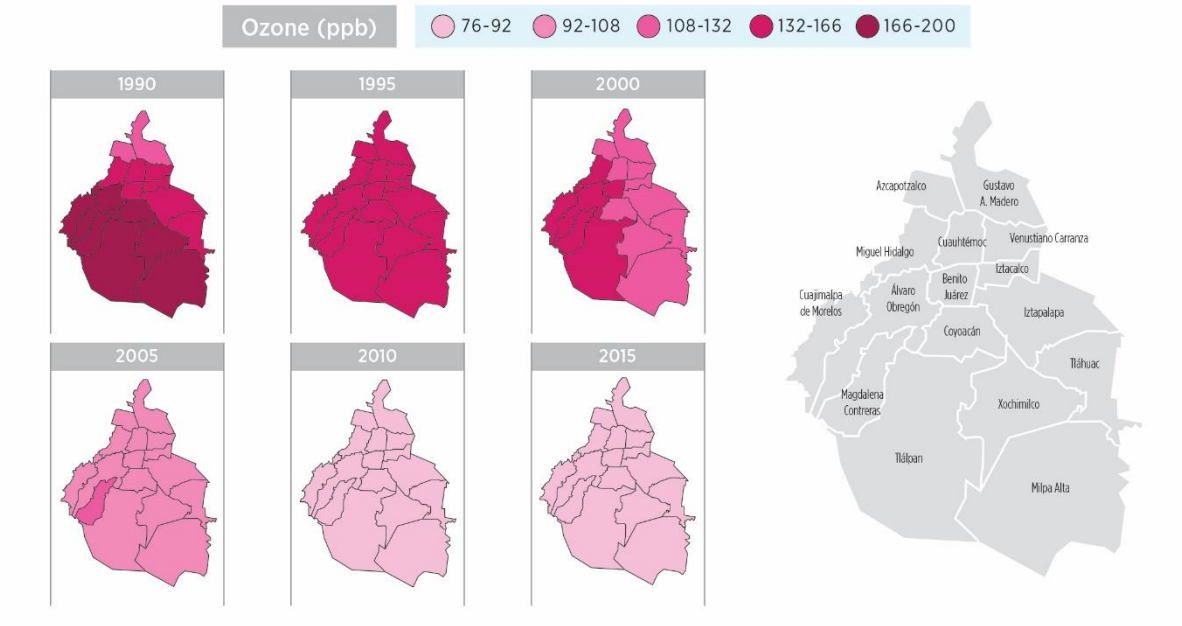


Figure I.4. Estimated seasonal (six month) daily 1-hour maximum ozone concentration at the *delegaciones* of Mexico City from 1990 to 2015



We observe at the *delegación* level, substantial air quality improvements in $PM_{2.5}$ concentrations since the early 90s (Fig. I.3). In the early 1990s annual average $PM_{2.5}$ concentrations could be close to 40 $\mu g/m^3$ in some *delegaciones*, and by 2015 a significant reduction is observed, levels were below 25 $\mu g/m^3$ throughout the city. Interestingly, in 2010 concentrations reached their lowest levels in most *delegaciones* in the city and then some of them presented slightly higher concentrations.

Ozone's behavior at the *delegación* level shows that seasonal 1-hour maximum levels have also decreased importantly in Mexico City (Fig. I.4). In the 1990s concentrations between 120 and 200 ppb were found in the City. By 2012 the ozone scenario had improved and since then, all the *delegaciones* show levels below 95 ppb.

Overall the observed gradual reduction of ozone concentrations reflects the success of air management public policy strategies in the City. For $PM_{2.5}$, attention should be paid to whether the trend continues to increase or whether it stabilizes or decreases as it did before 2010, to determine the most adequate control measures that may need to be implemented.

The next step in our risk assessment involves data management of mortality statistics for Mexico City. Official mortality numbers for Mexico City for 1990–2014 were obtained from SEDESA, which is the authority in charge of integrating and forwarding health mortality statistics from Mexico City to INEGI (National Institute for Statistics and Geography). Official mortality counts were evaluated for the five causes of death to be included in our risk assessment: for adults (30 years and older), cardiovascular diseases, including ischemic heart disease and cerebrovascular stroke (hemorrhagic and ischemic), chronic obstructive pulmonary disease, and trachea, bronchus and lung cancers; and, for young children (less than five years old), acute lower respiratory infections.

Mortality data was corrected for certain coding problems. We corrected for deaths that had been registered in the first few months of the year after the death had

actually occurred. Such deaths were recoded to reflect the actual year of occurrence. Also, deaths assigned to causes that are not underlying causes of death or that are immediate or intermediate causes of death, known as “garbage codes”, were redistributed to certain target causes. An important target cause is cardiovascular disease, and the main target code is ischemic heart disease. Because Mexico has a well-established cause-of-death registry, low levels of garbage codes are present. As such, the number of ischemic heart disease deaths increased by 7% during the whole study period.

The stage that involves characterizing the exposure-response relationship is needed to estimate the health benefits accrued due to the improvements in air pollution that have occurred in Mexico City since 1990. To do so, it is necessary to know how much mortality risk decreases for every unit decrease ($\mu g/m^3$ of $PM_{2.5}$ or ppb of ozone) in ambient air pollution levels. We addressed this question by considering the following issues: the uncertainty in the risk coefficient near the values of PM and ozone observed in the individual studies; the synthesis of the risk coefficients from cohort studies and the uncertainty in the pooled risk coefficient; and the shape of the dose-response function to understand its behavior at concentrations higher than those seen in the cohort studies.

To estimate premature deaths avoided attributable to air pollution we relied on exposure-response functions developed and used to support the GBD analysis for 2010 and 2013. For $PM_{2.5}$ we apply the integrated exposure-response (IER) function developed by Burnett and coauthors (2014). We chose this exposure-response function because it represents a widely-scrutinized synthesis of estimates from epidemiological results from 8 cohort studies of ambient air pollution. This function also incorporates risk estimates from studies that have evaluated exposures to particles emitted by non-ambient sources, such as second-hand smoke, indoor air pollution from cooking and heating, and active smoking. The GBD analysis of the IER coefficients for $PM_{2.5}$ was conducted separately for the five causes of death that are included in our risk assessment, four classes of disease in adults and one for young children. Also, we incorporated the approach applied by the GBD research

group to characterize the within study parameter uncertainty and between study variability by using the set of values that were estimated for the parameters of the IER function.

For ozone, we relied on the exposure-response function that stems from the Jerrett and coauthors' (2009) analysis of the ACS study and was used for the GBD analysis. The health outcome analyzed for ozone is COPD in adults.

In our analysis the shape of the exposure-response functions for $PM_{2.5}$ and for ozone constrained the risk estimates in the high-exposure regions. The IER applied for $PM_{2.5}$ constrains the risk by incorporating the relative risks that have been estimated in epidemiological studies of non-ambient particle sources for which observed exposures are much higher than those found in ambient air cohort studies. For ozone, we constrained the function from the analysis of Jerrett and coauthors' (2009) at higher levels of exposure following the approach by Lelieveld and coauthors (2015), first suggested by Ostro for particles (2004).

If the concentration-response functions seen in the cohort studies are not constrained, but are extrapolated linearly to elevated concentrations, the resulting relative risk estimates are implausibly large. By constraining the concentration-response functions we were able to better model the risk for $PM_{2.5}$ and ozone elevated concentrations that were observed Mexico City in the 1990s, which were higher than those observed in the cities where the cohort studies were conducted. In the United States and Europe annual average $PM_{2.5}$ concentrations were lower than 30 $\mu g/m^3$ and ozone concentrations did not exceed 104 ppb. In contrast, in Mexico City $PM_{2.5}$ concentrations from 1990 to 1996 were often in the order of 35 $\mu g/m^3$ and were not uncommonly in the range of 50 to 60 $\mu g/m^3$. In the early 1990s ozone seasonal averages of daily 1-hour maxima were frequently in the range of 120 to 180 ppb, and even reached 200 ppb.

To calculate the health benefits realized as a result of the improvements in air pollution, we first estimate the fraction of deaths attributable (attributable risk fraction,

ARF) to air pollution in each *delegación* between 1990 and 2014 using the exposure-response functions. For $PM_{2.5}$ the GBD team developed IER functions for ischemic heart disease and for cerebrovascular stroke by every five-year age-group, thus, we estimate ARF for each age-group. To account for variability in exposures across the population, the ARF must be evaluated at each exposure level and then integrated over all levels of exposure using weights corresponding to the number of deaths due to the disease of interest found in that exposure level.

The fractions of mortality attributable to these pollutants are estimated for five ($PM_{2.5}$) and one (ozone) cause of death, and are then multiplied by the number of deaths from each cause in each *delegación*. The resulting estimates are aggregated across diseases and *delegaciones* to obtain estimates of the total number of premature deaths avoided for Mexico City.

Health Benefits ("Premature Deaths Avoided") and Improved Air Quality in Mexico City

By reducing average ambient $PM_{2.5}$ concentrations from 45 $\mu g/m^3$ in 1990 to 20 $\mu g/m^3$ in 2014 and simultaneously reducing average ambient ozone concentrations from over 130 ppb in 1990 to close to 80 ppb in 2014,[14] Mexico City has been able to reduce the number of premature deaths avoided attributable to air pollution during this 25-year period by more than 22 thousand (95%CI: 17.9 to 28.0 thousand) (Table 1.). Roughly, 80% of the benefits were due to improvements in $PM_{2.5}$. Central estimates of the benefits for $PM_{2.5}$ and ozone were approximately 18.0 thousand and 4.0 thousand premature deaths avoided, respectively.

The benefits from both $PM_{2.5}$ and ozone air quality improvements range from almost 18 to 28 thousand premature deaths avoided. The intrinsic uncertainty in our estimates is not due to careless analysis, but to the sources of uncertainty that are

[14] For $PM_{2.5}$ annual average estimates were computed from annual averages from four sites in 1990 and from five sites in 2014 -which were in turn estimated from predicted 24-h concentrations from the GAM developed for the project. For ozone, seasonal (February to July) 1-hour maxima levels were computed from measurements from 6 and 27 sites, respectively, for 1990 and 2014.

propagated from the exposure assessment and the concentration-response functions to the final results.

Table I. Expected number of premature deaths avoided by reduction of $PM_{2.5}$ and ozone exposures in Mexico City, 1990 – 2014

	Premature Deaths Avoided (Thousands)	95% Confidence Interval
Air quality improvements in $PM_{2.5}$ and ozone*	**22.5**	**(17.9 - 28.0)**
Reductions in annual average $PM_{2.5}$ concentrations	18.2	(14.0 – 23.5)
Reductions in seasonal 1-h maxima concentrations	4.1	(2.7 – 5.6)

* Totals may not exactly equal the sums of disease-specific estimates due to rounding errors

The evaluation of the relative importance of each of the variables as individual sources of uncertainty show that the single largest source of uncertainty comes from the $PM_{2.5}$ concentration-response function. Uncertainty about the ozone concentration-response function is the second largest source of uncertainty. Such findings reflect the scientific uncertainty about the **true** concentration-response functions for $PM_{2.5}$ and ozone, that arise because of quantitative differences in the findings of the major cohort studies, and for $PM_{2.5}$, because of the need to rely on evidence from studies of smoking and exposure to second-hand smoke, and smoke from cooking and heating in poorly ventilated homes to characterize risks at high levels of exposure.

Complementarily, uncertainty about $PM_{2.5}$ and ozone exposure estimates were not important determinants of uncertainty in our results. Even if small, our exposure estimates incorporate uncertainty from the actual measurements at monitoring sites, the scarcity of monitoring sites in the earlier years of the study period, the spatial interpolation of concentrations from these sites to the *delegaciones*, and the need to estimate concentrations of $PM_{2.5}$ for the 1990 – 2004 period, when $PM_{2.5}$ was not directly monitored.

The vast majority of the benefits accrued from air quality improvements result from a reduction in premature deaths avoided from ischemic heart disease, chronic obstructive pulmonary disease, and cerebrovascular stroke, which account for almost 45%, 30% and 20%, respectively. Premature deaths avoided due to lung cancer among adults, and acute lower respiratory infections among young children also contribute, but together sum less than 10% of the mortality benefits of air pollution improvements.

It is important to note that premature deaths avoided among young children from acute respiratory infections become much more important when viewed from the perspective of their impact on longevity. Each such death involves a loss of life expectancy of many decades. In contrast, premature deaths avoided among adults due to ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, or lung cancer typically involve losses of life expectancy of perhaps one or two decades.

The primary results that are presented derive from our ***base case analysis*** which is defined by certain choices about several assumptions. A sensitivity analysis was conducted to assess how much these choices impact our central estimates and uncertainty results. If we had not constrained the ozone concentration response function, our central effect estimate would have been about 20% larger. Likewise, if we had used 1993 instead of 1990 as the reference year, our central estimate would have been about 20% lower. In contrast, the decision about whether to down-weight the influence of $PM_{2.5}$ concentrations from Xalostoc into neighboring *delegaciones* appears to have had only an infinitesimal impact on our results.

The essential story – i.e., that reductions in $PM_{2.5}$ and ozone over the past 25 years have led to substantial improvements in health and reductions in mortality – with more than 20 thousand premature avoided deaths over the period, is not modified when we change the choices about the assumptions from our base-case analysis, as shown by our sensitivity analysis.

Finally, our study assumes that if it hadn't been for the rigorous air pollution controls implemented since the late 1980's, air pollution would have been increasingly worse than in 1990. This would have resulted from the population growth, vehicular fleet increase, and expansion of the urban sprawl, that has occurred in Mexico City and the surrounding urbanized areas since 1990. Thus, the true health benefits of government public policy strategies and programs are likely to be considerably larger than the values estimated in this study.

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II. Final Report

Introduction

In the late 1980s and early 1990s air quality in Mexico City (CDMX[15]) was the worst in its history. Lead, sulfur dioxide, carbon monoxide, ozone, and particulate matter concentrations far exceeded national ambient air quality standards. Since then, the Government of Mexico City and agencies from the Federal Government have made substantial efforts to control air pollution. As a result of these efforts, ambient levels of fine particulate matter and ozone have decreased almost 50% from the levels that prevailed in the early 1990s. $PM_{2.5}$ concentrations (expressed as annual averages of daily 24-hour averages) have decreased from between 30 and more than 60 micrograms per cubic meter ($\mu g/m^3$) in 1990[16] to between 20 and 30 $\mu g/m^3$ in 2015 (as measured by Mexico City environmental authorities). Ozone concentrations (expressed either as annual or as seasonal maxima of daily 1 hour maxima[17]) have decreased from between 85 and 200 parts per billion (ppb) in 1990 to between close to 55 and 90 ppb in 2015, as computed from official data from fixed-site monitoring stations. These control measures, which have not been inexpensive, were implemented with the hope that they would contribute to an improvement in public health.

Results of epidemiological studies, which have found that $PM_{2.5}$ and ozone exposures contribute to increased mortality, are used in the evaluation of proposed regulatory programs. In the United States, for example, the Environmental Protection Agency (EPA) conducts benefit-cost analyses of all major regulations.[18] In Mexico, regulatory impact assessments that include benefit-cost analysis were introduced in

[15] The Federal District officially became Mexico City since 2016. The Federal District and Mexico City are conformed by 16 boroughs, called *delegaciones*.
[16] As $PM_{2.5}$ was not directly measured in the City but after 2004, concentrations prior to this year were estimated by means of a predictive model that includes PM_{10}, meteorological, spatial and time variables. (See Phase II Report for details on model development.)
[17] Seasonal maxima of daily 1 hour maxima ozone concentrations is the exposure metric used in epidemiological cohort studies and will be used repeatedly in this report.
[18] The Office of Management and Budget defines major regulations as those expected to incur regulatory costs of US $100 million or more.

the early 2000s for the systematic evaluation of potential social impacts of regulations (Rojas-Bracho et al., 2013). The Ministry of Economy is required by law to carry out benefit-cost analyses in support of regulations and standards, and the Ministry of Finance must also complete a benefit–cost analysis when making a decision about federal funding of a major investment project (Rojas-Bracho et al., 2013; SHCP, 2013).[19]

In benefit-cost analysis, regulatory costs are subtracted from monetized estimates of health benefits to determine whether the program is likely to generate positive net social benefits. Health benefits are calculated on the basis of the findings of epidemiological studies and are monetized using estimates of society's willingness to pay to reduce morbidity and mortality risks.

A National Academy of Science review of EPA benefit-cost analyses for various air pollution regulations indicated that, of all the health benefits generated by these regulations, reduction of mortality was typically responsible for 80% of the total monetized health benefit (NAS, 2002).[20] That is why the Government of Mexico City, in considering the health benefits of its public policy strategies to control air pollution, must ensure that the analysis focuses on their impact on mortality, measured as avoided premature deaths. It must be said that what is actually involved is a change in the timing of deaths, specifically a reduction in life expectancy.

In 2015 the Ministries of Environment (SEDEMA) and Health (SEDESA) of Mexico City launched a program of collaborative research with the TH Chan Harvard School of Public Health intended to estimate and verify the public health benefits of air pollution control public policy strategies implemented over the previous twenty years.

[19] For the Ministry of Economy, "high impact" regulations require a detailed cost-benefit analysis. For all other regulations the sponsoring agency is only required to answer a few questions about the quantifiable and non-quantifiable costs and benefits of the proposed regulation. For the Ministry of Finance, a detailed cost-benefit analysis is required for major projects (that is all investments over $150 million pesos), as well as for other investment projects at the Ministry's discretion (Rojas-Bracho et al., 2013).

[20] Regulations that are targeted to reduce air pollution diminish premature mortality risk in large populations, and the individuals that are affected are unknown. The terms "reduced mortality risk", "premature mortality", and "lives saved" are used in risk assessment. For the present project these terms are used indistinctly.

The program involves four phases – (I) State of knowledge and relevance to Mexico City; (II) Estimation of the health benefits of air pollution improvements (using risk assessment); (III) Verification of the health benefits of air pollution improvements (using epidemiology); and (IV) Evaluation of health economic impacts of policy interventions. The program is to be conducted over a period of four years, beginning in 2015.

This is the final report for the first phase. It consists of an overview of the public policy strategies to control air pollution that have been implemented in Mexico and in Mexico City, and a description of how air quality has improved in Mexico City from 1990 to 2015. It ends with a literature review and a description of the evidence that will sustain the risk assessment on the health benefits attributable to $PM_{2.5}$ concentration improvements since the early 1990s for Mexico City. That risk assessment will be the second phase of this project.

Phase I. State of Knowledge and Relevance to Mexico City

1. Overview of Public Policy Strategies for Air Pollution Control in Mexico and in Mexico City

a. Air Quality Management Programs Implemented in Mexico City

Over the last 25 years Mexico City has implemented many plans and programs to manage and improve air quality. An essential public policy strategy is to measure air pollutants in fixed-site monitoring stations; this effort began in the late 1970s. A few years later, federal and local and local environmental authorities launched a multitude of initiatives to manage and regulate air pollution. In 1987, a first plan with "100 Necessary Actions" included reducing lead and sulfur in gasoline. Other early plans were the Environmental Contingencies Program (*Programa de Contingencias Ambientales Atmosféricas*, PCAA), in 1988, and one year later the *Hoy No Circula* and the Inspection and Maintenance Programs. The PCAA established tiered threshold ozone and PM_{10} levels to activate either *Pre-contingencias* or *Contingencias* and trigger increasingly tighter actions to reduce both air pollutant emissions and exposures of sensitive populations to ambient air pollutants. *Hoy No Circula* aimed at taking almost half a million private vehicles (20%) a day out of circulation, based on the last digit of the license plate. The objective of the Inspection and Maintenance Program was to identify high-emitting vehicles in need of repair, through visual inspection and emissions testing, and force owners to repair them as a prerequisite to vehicle registration. These programs are still in effect in Mexico City, although they have been substantially modified over time.

For instance, the *Hoy No Circula* Program, was modified in several occasions to promote the introduction of newer vehicles with increasingly more advanced emission control technologies. Circulating restrictions for newer vehicles (1996 and 1998 modifications) and then for new vehicles (modification of 2007) were abolished. In contrast, the restriction for cars 10 years and older was maintained and they were further banned from the streets two days per week during atmospheric Contingencies ("double" *Hoy No Circula*).

In 1990, federal authorities, PEMEX, local governments, scientists and academics worked together to develop the Integrated Program for Air Pollution Control (PICCA). The primary goal of PICCA was to improve air quality specifically in Mexico City, but its analyses of strategies to control pollutant emissions also led to the setting of national standards. These standards included fuel quality improvements and emission limits of private and public transportation vehicles. In 1996 the federal Ministry of Environment and the governments of Mexico City and the State of Mexico published an air quality management plan (ProAire), covering the period from 1995 to 2000. The Environmental Metropolitan Commission was also created that year to coordinate all efforts related with air pollution control in the Metropolitan Area of the Valley of Mexico (MAVM)[21]. More air quality management plans followed: ProAire 2002-2010 and ProAire 2011-2020. The latter sets forth eight strategies, including protection of public health; reductions in energy consumption; energy quality and energy efficiency; mobility and regulation of energy efficiency explicitly for vehicles, and changes in emission control technologies.

In 2013 the federal government and the governments of Mexico City, the State of Mexico, Hidalgo, Puebla, Tlaxcala, and Morelos created the Environmental Commission of the Megalopolis (CAMe) to coordinate and manage more efficiently environmental public problems that this spread-out urban and industrialized area is currently facing. In early 2016, as a result of high ozone levels in the MAVM, the CAMe modified the *Hoy No Circula* and the PCAA to curb peak ozone concentrations. All private vehicles, regardless of their model-year, were banned from circulating four weekdays and one Saturday per month. Also, the threshold level to activate an atmospheric *Contingencia* was tightened (See below, **2.c. Recent Air Quality Emergencies**). In addition, authorities announced that new strategies to fight air pollution would be put in place, and that over 160 specific control measures were being analyzed.

[21] The definition of the Metropolitan Area of the Valley of Mexico (MAVM) that prevailed until December, 2005 included 16 boroughs (called *delegaciones*) of the then called Federal District, and 18 boroughs (called *municipios*) of the State of Mexico. After that date the definition was officially modified to include 59 boroughs from the State of Mexico, 1 from the state of Hidalgo, and 16 from the Federal District (INEGI, 2016).

As of July 1st, 2016, an emergency standard applies to all vehicles circulating in the Megalopolis. It introduces for the first time the use of the On–Board Diagnostics System (OBD) for the inspection of emissions.[22] The standard also reduces emission limits for vehicles 10 years and older, both gasoline (75% reduction) and diesel (40% reduction) fueled vehicles (CAMe, 2016). Furthermore, the Federal Attorney's Office for Environmental Protection (Procuraduría Federal de Protección al Ambiente, PROFEPA) will inspect verification stations in the Megalopolis to ensure compliance with the new standard. This emergency standard is temporary in nature, it will be in place for six months, can be extended for six additional months if necessary, and becomes permanent in those states that enforce vehicle-emission verification programs.

b. Federal and Local Public Policy Strategies: Vehicle Emissions, Fuel Quality, and Ambient Air Quality[23]

In general, health and environmental regulations have developed under a framework of "order and control", based on standards to protect public health and the environment. Some regulations aim to regulate pollutant emissions by setting limits on mobile and point sources. Others, based on the best international scientific evidence, set maximum permissible limits (MPL) for concentrations of air pollutants in the urban atmosphere.

Control and reduction of air pollutant emissions are regulated by the legal powers established by the Mexican federal system for each level of government (Flamand and Rojas-Bracho, 2015).[24] The federal government has the power to issue and

[22] The emergency standard (Norma Oficial Mexicana de Emergencia NOM-EM-167-SEMARNAT-2016) relies on current regulations for new vehicles that required vehicles model-year 2006 or newer to be equipped with OBD, which constantly monitor the performance of pollutant emissions control systems.

[23] A different version of this section was published in Rojas-Bracho L., Leonor Cedillo-Becerril, and Ania Mendoza Cantú. 2014. “Salud y medio ambiente. Metodología PEIR”. 2014. En: Juan Manuel Castro Albarrán, Martha E. Palacios Nava, María del Pilar Paz Román, Guadalupe S. García de la Torre, Laura Moreno Altamirano (eds.). Salud, Ambiente y Trabajo. McGraw Hill Education. México, D.F. ISBN: 978-1-4562-2254-3.

[24] See the General Law on Ecological Equilibrium and the Protection of the Environment (LGEEPA) (*Capítulo II. Distribución de Competencias y Coordinación*), and corresponding secondary rulings for

monitor the compliance of new vehicle emission standards and fuel quality specification standards. Governments at the state and municipal level[25] are granted the power to control air pollution generated by in-use vehicles. The Mexico City government uses this authority to implement programs such as inspection and maintenance, *Hoy No Circula* and mandatory school-bus transportation for schools with certain characteristics, such as number of students. It has also developed other strategies to promote clean and efficient public transportation, such as the bus transit systems in restricted lanes (a modification of the so-called Bus Rapid Transit systems, BRT), less expensive than the subway.

Regulation of Air Pollutant Emissions and Fuel Quality

New vehicle emissions have been regulated for at least 30 years. Current standards in the United States, the European Union and Japan are regarded as best international practices, and emission limits in the United States and the European Union are now practically equivalent.

The Mexican standards for new vehicular emissions are more lenient than those in developed countries, which results in a technological lag of over 10 years. Barring that lag, vehicles circulating in Mexico could be over 10 times cleaner. For diesel vehicles, in Mexico City these are predominantly heavy duty vehicles (such as trucks and buses) technological developments would result in over 98% particle emission reductions and changes in diesel exhaust composition. Furthermore, carcinogenic emissions from vehicles that comply with best international practices are so low that they can no longer be detected by pollution testing instruments[26] (Blumberg et al.,

the control and prevention of air pollution (*Reglamento para la Prevención y Control de la Contaminación de la Atmósfera (Capítulo I. Disposiciones Generales*).

[25] In Mexico City "municipal" refer to the political demarcations called *delegaciones*.

[26] Diesel exhaust is a mixture composed by carbon dioxide, oxygen, nitrogen, nitrogen compounds, carbon monoxide, water vapor, sulfur compounds, numerous low and high molecular weight hydrocarbons, and particulate matter aggregates of elemental carbon nanoparticles with associated hydrocarbons. Technological developments in diesel engines and use of ultra-low sulfur fuel have modified the relative contribution of such a mixture. The emissions of particulate mass are reduced significantly and are free of the elemental carbon particles. In contrast, the exhaust of older diesel engines that still use high sulfur content fuel contains elevated concentrations of carbonaceous particulate matter with associated elevated concentrations of polycyclic aromatic hydrocarbons, nitrogen oxide (NOx) and gas phase hydrocarbons. The potential of carcinogenicity associated with

2003; Khalek et al., 2011). These emission reductions are achieved with improved engine control, fuel injection systems, and by combining advanced pollution control technologies, such as three-way catalytic converters and high-efficiency diesel exhaust particulate filters, together with ultra-low sulfur (ULS) gasoline and diesel (McClellan et al., 2012).

Mexico, due to its more lenient standards, does not benefit from these technological advances. For new cars, Premium ULS gasoline (best quality, used mostly in new vehicles) is available. However, for older cars, which account for the vast majority of the fleet, use Magna gasoline, which is not widely available with ULS quality. ULS diesel has been available for a few years in Mexico City, Monterrey, Guadalajara, and the Northern border region. In November 2015 Federal authorities published an emergency fuel quality standard; the first deadline of this standard was fulfilled, and ULS diesel has been available since December 2015, in 11 corridors linking 200 cities and towns. A second deadline set for July 2018 is to expand availability to the rest of the country.[27] Vehicles with older technologies that are fueled with ULS fuels may gain certain pollutant emission reductions. However, the optimum control of exhaust emissions can only be achieved by combining ULS fuels together with vehicles with advanced engines and exhaust after-treatment systems. Such vehicles are available in developed country vehicle markets since 2006.

Air Quality Regulation

Mexico and most other countries use MPL set in their health and environmental regulations as a yardstick to evaluate air quality and the potential adverse impact of criteria pollutants on human health. The basis for developing permissible limits at

diesel exhaust relates, among other, with organic solvents of the particulate matter (McClellan et al., 2012).

[27] The National Energy Strategy (Estrategia Nacional de Energía 2013-2027, Secretaría de Energía, 2013) includes the production of ULS gasoline and diesel in the six refineries that operate in Mexico. Complementarily, the Emergency Fuel Quality Standard (Norma emergente sobre calidad de combustibles, NOM-EM-005-CRE-2015) was published on October 30, 2015 in the Diario Oficial de la Federación (http://www.dof.gob.mx/normasOficiales/5848/cre/cre.html).

country level is the best available international evidence on the specific impact on human health of acute (hours or days) or chronic (months or years) exposures to ambient air pollutants. The World Health Organization (WHO) issues recommendations on the concentrations that must not be exceeded in order to protect public health; these recommendations are tighter than Mexico's national regulations (WHO, 2006).

National limits are set as Mexican Official Norms (NOM) issued by the federal Ministry of Health. The most relevant pollutants for health and public policy are suspended particulate matter, specifically $PM_{2.5}$, and ozone. The association between fine particulate matter and ozone and the rest of the criteria pollutants is unquestionable, for they share both pollutant emission sources and many chemical processes that happen in the atmosphere. Therefore, $PM_{2.5}$ and ozone are indicators of the complex mix of pollutants found in urban and industrial atmospheres.

Since particulate matter is regulated in terms of its aerodynamic diameter, MPL are established for $PM_{2.5}$ and for PM_{10} (DOF, 2014a). For both size fractions, the standard includes limits for two averaging times, one for acute exposures (24-hour average), and one for chronic exposures (annual average). Ozone is also regulated with two averaging time maximum permissible limits, one for one hour (maximum hourly average per calendar year), and another one for eight hours (annual maximum eight hour moving average) (DOF, 2014b).

In 2014 the $PM_{2.5}$, PM_{10} and ozone standards were updated (Table 1.1). The 24-hour average standard for $PM_{2.5}$ was reduced from 60 to 45 $\mu g/m^3$ and the annual average standard was reduced from 15 to 12 $\mu g/m^3$. The limits for PM_{10} were reduced from 120 to 75$\mu g/m^3$ for the 24-hour average standard, and the annual standard was reduced from 50 to 40 $\mu g/m^3$. The standard for ozone was also reduced from 110 to 95 ppb for the hourly average, and from 80 to 70 ppb for the 8-hour average.

Table 1.1. Mexican ambient air quality standards and WHO recommendations for PM_{10}, $PM_{2.5}$ and ozone

		Maximum Allowable Limits		
		Mexican Official Norm*		WHO
Pollutant	Exposure / Averaging Time	Previous	In force	
PM_{10} ($\mu g/m^3$)	Chronic exposure (Annual average)	50	40	20
	Acute exposure (24-hour average)	120	75	50
$PM_{2.5}$ ($\mu g/m^3$)	Chronic exposure (Annual average)	15	12	10
	Acute exposure (24-hour average)	65	45	25
O_3 (ppb)	Chronic exposure (8-hour moving average)	0.08	0.07	0.05
	Acute exposure (1-hour average)	0.11	0.095	n.a.

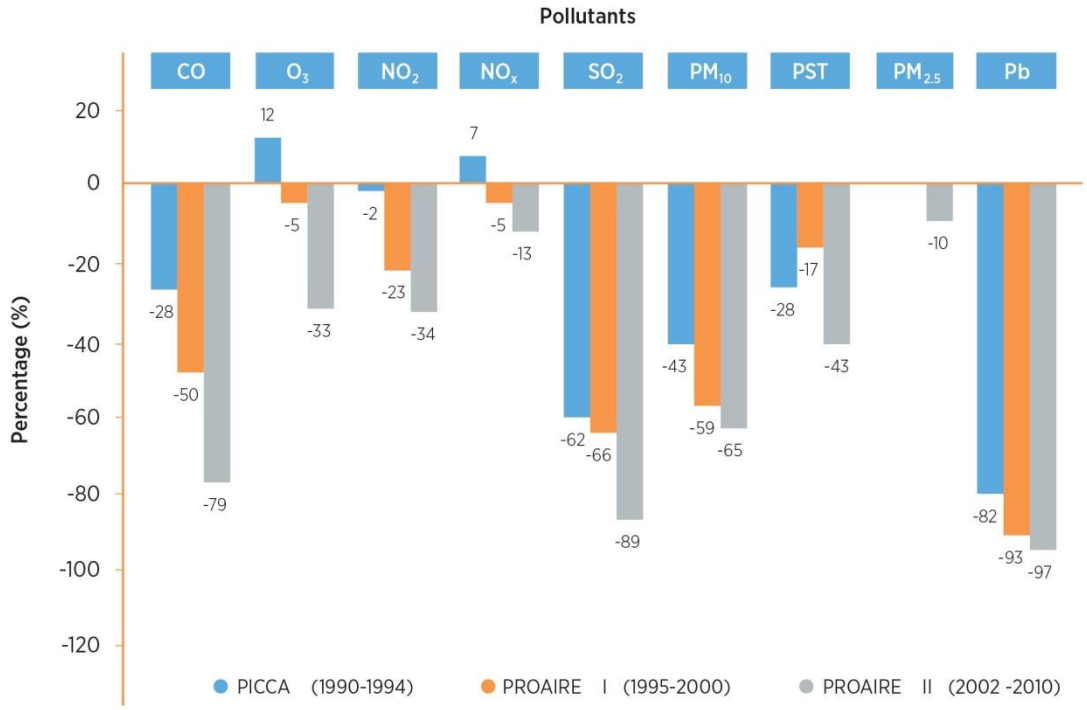
Notes: * The tighter NOM-025 for particulate matter and NOM-020 for ozone were published in August, 2014. They are in force since October, 2014. n.a. = not available; WHO does not have a 1-hour ozone recommended limit.

Sources: WHO, 2006; DOF, 2014a; DOF, 2014b.

2. Air Quality Improvements in Mexico City from 1990 to 2015

The MAVM air-monitoring network put in place in the 70s makes it possible to assess changes in pollutant concentrations over time. At that time Mexico City's authorities started regular sampling of criteria pollutants: total suspended particles (TSP), ozone (O3), carbon monoxide (CO), nitrogen dioxide (NO_2), nitrogen oxides (NO_x) and sulfur dioxide (SO_2). Later, lead (Pb) was added to the monitoring network. Particles with a diameter of less than 10 microns (PM_{10}) were first monitored in the late 1980s, and particles with a diameter of less than 2.5 microns ($PM_{2.5}$) by the end of 2003.

Figure 2.1. Effective reduction (percentage) in air pollutant concentrations due to air quality management programs. Baseline: annual average concentration in 1989



Source: SMA-DF, 2010.

Over the past decades, due to the implementation of different air quality management plans, concentrations of criteria pollutants have decreased in the MAVM (Figure 2.1)[28]. We initially describe changes in particulate matter, with more emphasis on $PM_{2.5}$, and in ozone concentrations because these pollutants raise the larger concerns in terms of public health. We also describe briefly changes in the other criteria pollutants.

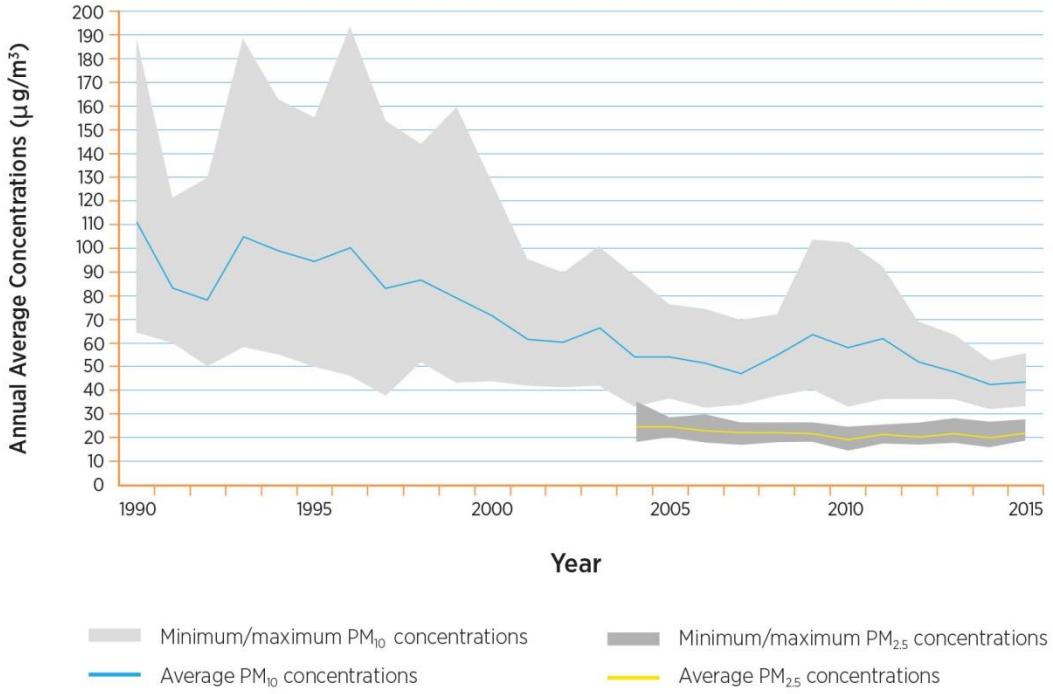
a. Particulate Matter ($PM_{2.5}$, PM_{10}) and Ozone

Concentrations of $PM_{2.5}$ have decreased slightly since 2004 (Fig. 2.2). Between 2004 and 2015 annual average $PM_{2.5}$ concentrations in the MAVM decreased from almost 25 µg/m^3 to close to 22 µg/m^3.[29] The annual standard, equal to 15 µg/m^3 until 2014 and tightened to 12 µg/m^3 by the end of that year, has been exceeded every year and in every single monitoring station (ProAire 2011-2020; Garibay Bravo et al., 2011; INECC, 2016). Historically, the highest annual concentrations have been reported in the northern part of the MAVM, at the monitoring stations of Xalostoc and, more recently, Camarones. The lowest levels are reported in the southern areas of the metropolitan area, such as Pedregal.

PM_{10} decreased about 60% between 1990 and 2015, from over 110 µg/m^3 to less than 45 µg/m^3. In 2014, the MAVM complied with the previous 24-hour standard (120 µg/m^3). However, if the stricter standard in force since the end of 2014 had been applied, the MAVM would have been out of compliance with both the 24-hour and the annual standards (75 µg/m^3 and 40 µg/m^3, respectively) (INECC, 2016).

[28] Figure 2.1. presents reductions of $PM_{2.5}$ for ProAire II only, due to the fact that measurements in monitoring stations did not start but until 2004.
[29] Authors' estimate based on official data from five fixed-site monitoring stations (Pedregal, UAM-Iztapalapa, Merced, Tlalnepantla y Xalostoc) that use manual sampling equipment (Hi-Volume Sampler).

Figure 2.2. Annual $PM_{2.5}$ and PM_{10} Concentrations in the Metropolitan Area of the Valley of Mexico, 1990-2015



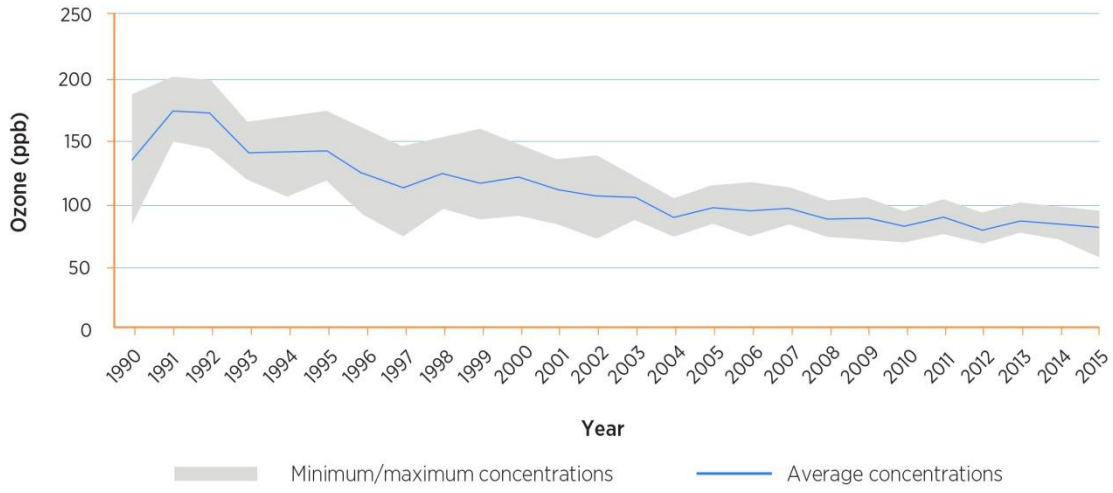
Note: Data available for $PM_{2.5}$ from 2004-2015, and for PM_{10} from 1990-2015 from official monitoring sites. Values estimated from five fixed site monitoring stations that use manual sampling equipment.

Source: Elaborated by the authors with official data from fixed-site monitoring network (*Red Manual de Monitoreo Atmosférico*, SEDEMA, CDMX)

Ozone concentrations have decreased significantly since 1989, showing a decrement of over 30% in 2010 (Fig. 2.3). In the 1990 annual 1-hour maximum concentrations during high ozone season reached 200 ppb. In contrast, since 2010 levels have been below 100 ppb (Fig. 2.3). Important reductions were observed during the '90s, with a slower downward trend for the following decade (SEDEMA, 2014).

In the 1990s the 1-hour standard was exceeded in 320 days. Ten years later the standard was exceeded in almost 200 days, and in close to 120 days in 2014. The 8-hour average standard has been exceeded every year since the late 1980s. In 2014, all but one site, Xalostoc, exceeded this standard (INECCC, 2016).

Figure 2.3. Ozone concentrations in the Metropolitan Area of the Valley of Mexico: Average of 1-hour maximum concentrations during peak ozone months, 1990-2015 1990-2015



Source: Elaborated by the authors with official data from fixed-site monitoring network (*Red Automática de Monitoreo Atmosférico,* SEDEMA, CDMX).

b. Other Criteria Pollutants: Lead, Carbon Monoxide, Sulfur Dioxide and Nitrogen Dioxide

The scenario for the other criteria pollutants is better. All of them are in compliance with their corresponding air quality standards. The significant reductions of worldwide atmospheric lead concentrations resulting from improvements in the environmental quality of fuels represent an important achievement. Ensuing benefits for human health have been extensively documented. Mexico began to reduce lead levels in fuels in the late 80s and culminated these efforts in 1997 with the full elimination of tetraethyl lead in gasoline. Lead levels have remained below the standard since 1992.

Carbon monoxide and sulfur dioxide levels have decreased significantly since the first air quality management program was launched. Concentrations of carbon monoxide have decreased since 1992, and remain low until today. Starting in 2000, the concentrations have been below the hourly maximum permissible limit at all monitoring stations.

Concentrations of sulfur dioxide have decreased constantly; since 2003 ambient sulfur dioxide levels have been in compliance with the 8-hour, 24-hour, and annual national standard limit values (Garibay Bravo et al., 2011; ProAire 2011-2020; INECC, 2016).

Concentrations of nitrogen dioxide have decreased from levels found in the late 1980s. However, the hourly limit was exceeded in 2003, 2004 and 2005 (Garibay Bravo et al., 2011). As of 2006, concentrations have been below the standard limit. Regardless of these low concentrations, nitrogen dioxide will remain a concern in Mexico City, for it is a precursor of ozone, a pollutant that constantly exceeds the national standards.

c. Recent Air-Quality Emergencies

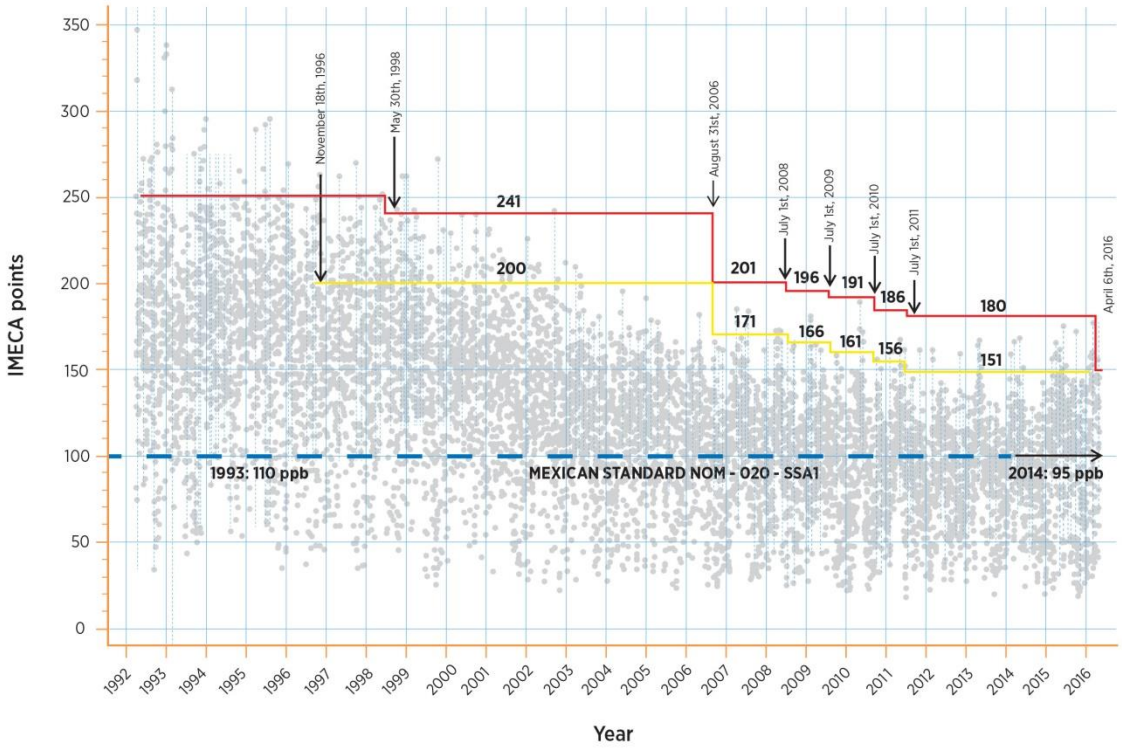
From January to July of 2016, the MAVM experienced at least nine ambient ozone emergencies, or *Contingencias*.[30] This was unprecedented since 2002 and has nurtured a misconception, in the media and public opinion, that air quality in the MAVM has deteriorated since last decade. These *Contingencias*, however, are a result of a stricter threshold value to activate them in hand with tighter ambient air quality standards for ozone, not of having worse air quality.

In 1989 a Metropolitan Index of Air Quality (IMECA) was developed as a tool to communicate to the public the state of air quality in the MAVM in clear and understandable terms. IMECA points, or IMECAS, were assigned equivalent concentrations for each air pollutant. A value of 100 IMECA points is equivalent to

[30] These atmospheric *Contingencias* were activated, among other days, on March 14, April 5, May 2, 14, 20, 24, 27 and 31, and July 8 (http://www.aire.cdmx.gob.mx/descargas/ultima-hora/calidad-aire/pcaa/pcaa-historico-contingencias.pdf).

the maximum allowable limit set in the Mexican air quality standard for each criteria pollutant, in force at a certain point in time.

Figure 2.4. Ozone Daily Maximum IMECA Points, 1992-2016



Note: Daily Maximum IMECA points (blue circles), threshold values to trigger *Pre-contingencias* (yellow line) or *Contingencias* (red line); Maximum Permissible Levels for the ozone standard in place (blue solid line) and for the previous standard (blue dotted line).

Source: Elaborated by the authors with data from SEDEMA, 2016.

The *Programa de Contingencias Ambientales Atmosféricas* (PCAA, see above, 1.a.) establishes threshold values, expressed in IMECAS, for ozone and PM_{10}. When these values are surpassed, a *Contingencia* is declared and emergency measures are applied. These control measures include reducing activity levels from point and mobile emission sources, for instance, by increasing the number of motor vehicles banned each day from the city's streets through the *Hoy No Circula* program.

Threshold values to activate a *Contingencia* through the PCAA have become stricter

throughout the years, decreasing from 250 IMECAS back in 1998 to 180 in 2012 (Fig. 2.4). As of April, 2016, a *Contingencia* is triggered when threshold values for ozone reach 150 IMECAS, a historical low. Furthermore, as the maximum allowable limit in national ambient air quality standard for ozone and PM_{10} were tightened in 2014, 100 IMECAS are now equivalent to lower concentrations of these pollutants.

As a result, the *Contingencias* declared in the first semester of 2016 are related to the lower threshold of 150 IMECAS, and to the lower maximum allowable limits in the air quality standards, rather than due to the deterioration of air quality.

Air quality in the MAVM continues to be a major problem but it is considerably better than it was in the 80s and 90s. For comparative purposes, in 1999, three *Contingencia* were activated with the threshold value of 240 IMECAS, as was set then by the PCCA. However, at least 36 *Contingencia* would have been triggered had the threshold been 150 IMECAS, as it is today.

3. Air Pollution and Adverse Health Impacts: International Body of Evidence

The effects of ambient air pollution on public health have been studied extensively and are relatively well established. For ambient air pollutants like particulate matter, ozone there is biological plausibility for health impacts. They can both cause oxidative stress that leads to chronic inflammation primarily in respiratory airways, degrade pulmonary function and impair gas exchange. Adverse health effects of ozone and particulate matter have been a concern for governments around the world for several decades. Recently this concern has increased, given the evidence regarding the impact of these pollutants on increased mortality and thus reduced life expectancy.

A great deal of epidemiological research on the health effects of air pollution has been conducted in Mexico. Studies have shown that short-term exposures are associated with health outcomes; most notably, day–to–day fluctuations in the levels of air pollution are correlated with day–to–day fluctuations in mortality. Furthermore, researchers have demonstrated that children living in areas with higher levels of ozone experience higher rates of asthma. Air pollution scientists around the world have made similar observations.

Epidemiologists have relied primarily on two study designs: (a) the time-series study, which assesses the effects of short-term exposures; and (b) the cohort study, which evaluates adverse health impacts of long-term exposures.

This section includes the nature and main findings of these two major epidemiological study designs that have evaluated air quality and its impacts on population health worldwide. These results are a key input to estimate the burden of disease associated with air pollution and, conversely, to evaluate health benefits associated with improvements in air quality. We also establish the basis for some sources of variability and uncertainty that stem from the epidemiological studies and affect the results of the risk assessment conducted in the next phase of this project.

a. Short-Term Exposures and Adverse Health Impacts

Time-series studies examine the relationships between day–to–day variations in levels of air pollution and day–to–day variations in mortality. More than 100 studies of this kind have been conducted in cities around the world. This body of evidence includes several studies in Mexico City, starting in the early 90s, and a multi-city study in Mexico, Brazil and Chile using 2005 air pollution and mortality data. Virtually all of the studies have found positive associations between daily levels of air pollution and daily mortality. Although there are both study-to-study differences and regional variations in the quantitative relationship between air pollution and mortality, the consensus among experts is that the time-series literature has clearly established the impact of daily fluctuations in air pollution on mortality (Atkinson et al., 2014).

The first study to investigate the health impacts of short-term pollution exposures in Mexico City was conducted with data for 1990 (Borja-Aburto et al., 1997). The study found that total mortality was associated with increments in ozone concentrations measured as daily averages and as 1-hour maximum concentrations. However, the ozone effect disappeared when daily TSP concentrations were added into the analysis, and only TSP remained significantly associated with mortality showing excess mortality of 0.6% per 10 $\mu g/m^3$ increase in TSP levels.

Other studies followed to assess the effects of daily changes in particles and ozone on daily changes in mortality in Mexico City. They were conducted between 1993 and 1995 with independent fine particle monitoring in one southwestern site in the city, near the official fixed-site monitoring site of Pedregal, and found an effect of fine particles on mortality (Borja-Aburto et al., 1998). Researchers also reported that ozone had a significant effect on cardiovascular mortality, which increased when $PM_{2.5}$ was included in the analysis. Two groups sensitive to ambient pollutant exposures were identified. People over 65 years of age were shown to be sensitive to the effects of ozone, with a larger increment in deaths from cardiovascular and respiratory causes. Infants were shown to be sensitive to particle exposures, exhibiting increases in relative risk roughly ten times larger than the general

population for similar increases in fine particle exposure (Loomis et al.,1999). Infant deaths were less consistently associated with ozone exposures.

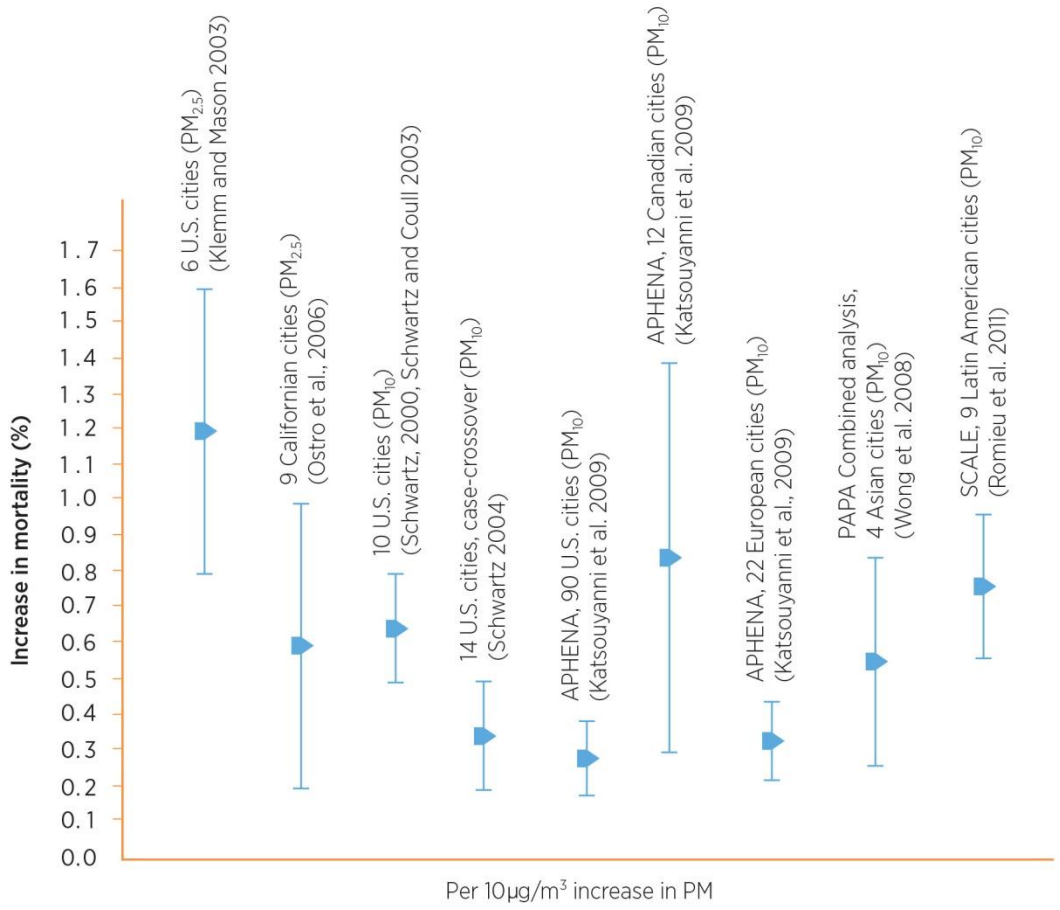
A mid-1990s study investigated the effects of daily changes of mortality of daily changes of three PM size fractions: $PM_{2.5}$, $PM_{2.5-10}$ (coarse particulate matter, with a diameter between 2.5 and 10 microns) and PM_{10} (Castillejos et al., 2000). Unexpectedly, results showed that the coarse fraction presented stronger effects than fine particles in single pollutant models, as opposed to most results in other cities of the world. Furthermore, the coarse particle effect prevailed in two-pollutant models that included fine and coarse particles. The effect of was stronger for coarse particles for total and for respiratory mortality; in fact, the effect of $PM_{2.5}$ was reduced to close to the null.

The multicity time-series study ESCALA (Air Pollution and Health in Latin America) evaluated the effect of daily exposures to PM_{10} (daily 24-hr mean average) and ozone (daily 8-hr maximum moving average) on mortality in three cities in Mexico (Mexico City, Toluca and Monterrey) and six cities in Brazil and Chile (Romieu et al., 2012). For all the cities, ESCALA reported that a 10 $\mu g/m^3$ increase in PM_{10} concentrations is associated with a 0.77% increase in all-natural cause mortality; for Mexico City, the increase was of 0.61 percent. For the nine cities, the study reported a 2.44% increase in chronic obstructive pulmonary disease mortality, and lower risks for cardiopulmonary, cerebrovascular-stroke, respiratory disease, and cardiovascular mortality.

For ozone, ESCALA found a smaller mortality effect than for PM_{10}. In Mexico City when ozone was included with PM_{10} in two pollutant models, the effect of PM_{10} remained significant. For Mexico City, associations, albeit small, were also found between ozone concentrations and all-natural cause, cardiopulmonary, cardiovascular, respiratory, and chronic obstructive pulmonary disease mortality. Some of these associations with ozone were found only in Mexico City.

ESCALA also identified socioeconomic status and age-group as possible effect-modifiers of the exposure-response relationship. Thus, population groups in the lowest socioeconomic level are at higher risk of mortality due to respiratory causes, especially COPD.

Figure 3.1. Findings on the association of daily change in $PM_{2.5}$ and PM_{10} concentrations and total mortality



Note: APHENA = Air Pollution and Health in Europe and North America.

Source: Based on the HEI Review Committee Commentary in the ESCALA report, 2012.

Evidence found in other studies conducted in Mexico is consistent and similar to that from other countries, especially regarding the effects of particulate matter and ozone on mortality (Fig. 3.1). An analysis of over 100 time-series studies conducted in cities around the world, most of them in North America and Europe, ratified the adverse health effects of particulate matter. For all-age all-cause mortality studies, the authors reported a pooled risk increase of 1.04% (CI 95% 0.52, 1.56) for a 10 $\mu g/m^3$ increment in $PM_{2.5}$, with significant regional differences (Atkinson et al., 2014). The specific causes of death which appear to be responsible for the observed increase in all-age all-cause mortality include cerebrovascular stroke, respiratory diseases, chronic obstructive pulmonary disease, and ischemic heart disease. Among these, cerebrovascular stroke and respiratory disease were not only more strongly associated with fine particles than other causes, but exhibited no regional variability.

b. Mortality and Long-Term Exposure

Cohort studies examine the differences in mortality experienced by people living in places with different levels of air pollution. They allow us to understand the effects of chronic exposures, that is cumulative exposures, to air pollutants. Cohort studies are much more expensive than time-series studies and, as a result, to date there are a relatively small number of cohort studies of air pollution and mortality and these have been conducted principally in the United States and Europe. Recently a few cohort studies have been initiated in Mexico, and some of them include air quality as one of the exposures of interest. However, these studies have not yet yielded results regarding the relationship between air pollutants and mortality.[31]

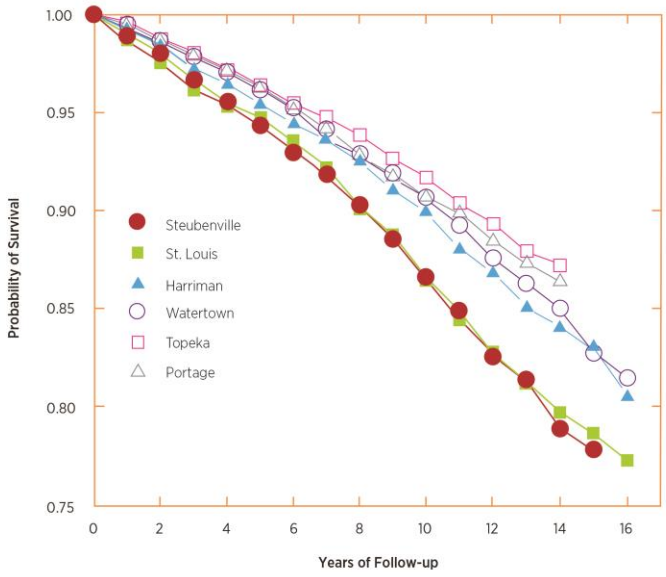
For this reason, risk assessments in Mexico have relied on results from the studies carried out to-date in other parts of the world. While this approach introduces some uncertainty, it is considered reasonable given the consistency of results between

[31] Two cohorts are worth mention for their potential to produce results on the association between air pollution and health outcomes: The “Estudio de seguimiento de la salud de las maestras” and the PROGRESS cohort (Program Research in Obesity, Growth, Environment and Social Stressors). These two cohort studies started recruitment in 2006 and 2007, respectively, and now consist of over 100 000 women, and 1000 mother-child pairs, correspondingly (Romieu, 2011; Braun et al., 2014).

study results from Mexico and studies conducted in other countries regarding short-term exposures and health effects (HEI, 2012).

The Harvard Six Cities study was the first cohort study to examine the relationship between air pollution and mortality (Dockery et al., 1993). The hypothesis of the study was that individuals living in cities with higher levels of particulate air pollution would experience higher rates of cardiovascular, respiratory, and lung cancer mortality. The study cohort consisted of 8111 white individuals, over the age of 25 at the time of recruitment, from six cities in the United States. The cities[32] were selected to provide a wide range of exposures to particulate matter. Levels of fine particulate matter in Steubenville (the dirtiest city) at the time were about 30 µg/m^3. In contrast, levels of fine particulate matter in Portage (the cleanest city) were about 10 µg/m^3.

Figure 3.2. Six Cities Study: Crude Probability of Survival vs. Years of Follow-up



Source: Dockery et al., 1993.

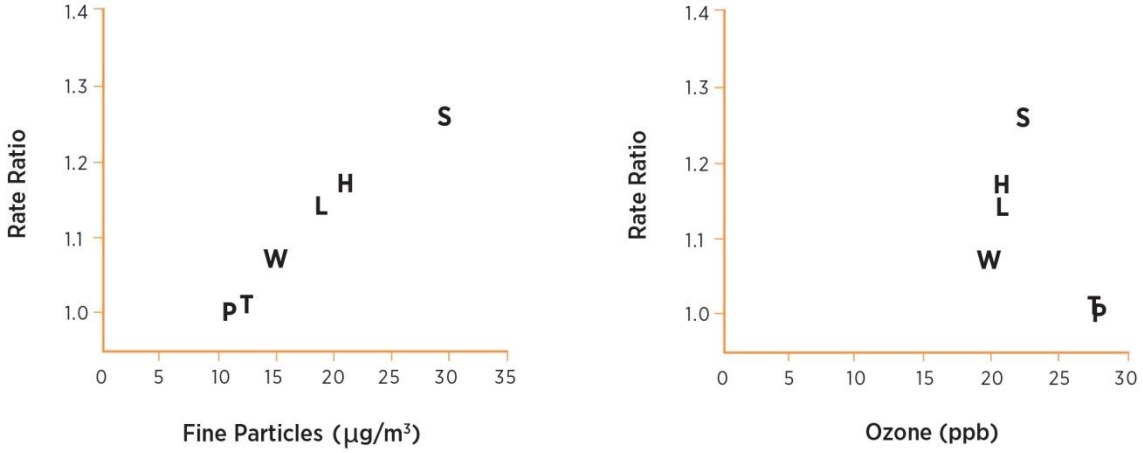
Analysis of the first 15 years of follow-up of the Six Cities Study indicated that survival was lower and mortality rates were higher in the dirtiest city (Steubenville, Ohio) than in the cleanest city (Portage, Wisconsin) (Dockery et al., 1993). Figure 3.2 shows the survival curves for study participants in each of the six cities over the first 15

[32] Steubenville, Ohio; St. Louis, Missouri; Kingston-Harriman, Tennessee; Watertown, Massachusetts; Portage, Wisconsin; and Topeka, Kansas.

years of follow-up. Figure 3.3. presents the mortality rate ratios plotted against the average concentrations of fine particles and ozone in each of the six cities.

Results reveal that the mortality rates in these cities, after adjustment for smoking and other risk factors,[33] were strongly associated with the average levels of fine particles in the cities: for every 1 μg/m^3 increase in fine particle concentrations, mortality rates increased by approximately 1.5%. As for ozone, the very narrow range of concentrations seen across these six cities (ranging approximately between 20 and 30 ppb) limited the study's power to detect an association.

Figure 3.3. Six Cities Study: Mortality-Rate Ratios and Fine Particle and Ozone Ambient Air Pollution



Note: The names of the cities are: P = Portage, Wisconsin; T = Topeka, Kansas; W = Watertown, Massachusetts; L = St. Louis; H = Harriman, Tennessee; and S = Steubenville, Ohio. Particles and ozone mean concentrations.

Source: Dockery et al., 1993.

Shortly after the Harvard Six Cities Study was published, Pope and his colleagues conducted a second analysis based on a much larger cohort, the American Cancer Society (ACS) study (Pope et al., 1995). Pope matched the residences of almost 500,000 individuals participating in the ACS Cancer Prevention Study II with air pollution data from the nearest ambient monitor. The ACS study had an initial 7-year

[33] The risk factors that were adjusted for include occupational exposures to pollutants, body mass index, level of education, among other (Dockery et al., 1993).

follow-up period (1982-1989), during which annual fine particle concentrations in the cities studied were strikingly similar to those found for the Six Cities Study, ranging from less than 11 to 30 $\mu g/m^3$ and with typical concentrations around 21 $\mu g/m^3$ (Pope et al., 1995; Pope et al., 2002). For ozone, the range of 1-hour maximum levels observed for the Summer (1988) were between 11.7 ppb and 56.4 ppb (Krewski et al., 2009).

Consistent with findings from the Six Cities, the ACS study also found an association between fine particle concentrations and mortality, after adjusting for smoking and other relevant individual risk factors (Pope et al., 1995). It must be said that results suggested an effect size about one third as large as that seen in the Harvard Six Cities Study, indicating that for each 1 $\mu g/m^3$ increase in ambient levels of $PM_{2.5}$ mortality rates would rise by about 0.4%.

The ACS study differs from the Six Cities study in many ways that help understand risk estimate dissimilarities: (i) ACS involves more than 50 times as many participants as Six Cities, and it triples the number of deaths; (ii) ACS participants came from over 100 cities; (iii) the ACS include not only white participants, but also black and Hispanic subjects; (iv) ACS improved the control of certain risk factors, including occupational exposures to gases and fumes, and diet (Pope et al., 2002).

Since then there have been several re-analyses and extensions of the Harvard Six Cities and American Cancer Society studies. (Pope 2002; Laden et al., 2006; Lepeule et al., 2012; Krewski et al., 2009). An independent team of researchers re–analyzed these two cohort studies to verify the validity of their results and conclusions (Krewski et al., 2000). Furthermore, additional years of follow-up were incorporated to test how robust the associations were between $PM_{2.5}$ chronic exposures and mortality; increase the power of the analysis; test lags of exposure and the shape of the concentration-response relationship; and reexamine specific causes of death.

The Six Cities cohort was extended to a total of 36-year follow-up from 1974 to 2009 (Laden et al., 2006; Lepeule et al., 2012), and the ACS extension encompassed a total of 18 years of follow-up (1982-2000) (Jerrett et al., 2009; Krewski et al., 2009). These extensions substantially increased the number of person-years of follow-up and of deaths, giving these studies greater statistical power.

Interestingly, air quality improved in the cities during the extended follow-up periods, for Six Cities average levels across the cities fell from around 18 $\mu g/m^3$ to close to 10 $\mu g/m^3$ by the end of the last extension of the follow-up, and for ACS annual average $PM_{2.5}$ concentrations dropped from over 20 $\mu g/m^3$ to close to 14 $\mu g/m^3$ (Dockery et al., 1993; Lepeule et al., 2012; Pope et al., 2002; Krewski et al., 2009). In the Six Cities the authors found that air quality improvements translated into a reduction in mortality (Laden 2006). These findings are relevant in for Mexico City, where substantial air quality improvement has been observed over the past 25 years.

For particles, the consistency in results from these two important cohort studies is noteworthy. Although the central estimates of the risk coefficients from the two studies differ by a factor of ~ 3, both studies have found that cardiovascular (a comprehensive category of diseases that include ischemic heart disease and cerebrovascular stroke), and lung-cancer mortality are associated with long-term fine particle exposures. And both have found that the concentration–response relationships appear to be linear or nearly linear within the range of observed concentrations – i.e., from 5.8 to ~30 $\mu g/m^3$ (ACS) and from 8 to ~30 $\mu g/m^3$ (Six Cities) (Lepeule et al., 2012; Pope et al., 2002; Krewski et al., 2009).

For ozone, the small range of exposures in the Six Cities Study precluded meaningful analysis of the issue. But because of the broader range of ozone exposure across the cities included in the ACS study from 1997 to 2000, which ranged from 33.3 ppb to 104.0 ppb (measured as daily maximum 1-hour concentrations from April to September), it was able to detect, for the first time, an adverse effect of chronic ozone exposures in mortality (Jerrett et al., 2009). The authors reported that ozone exposures were associated with deaths from

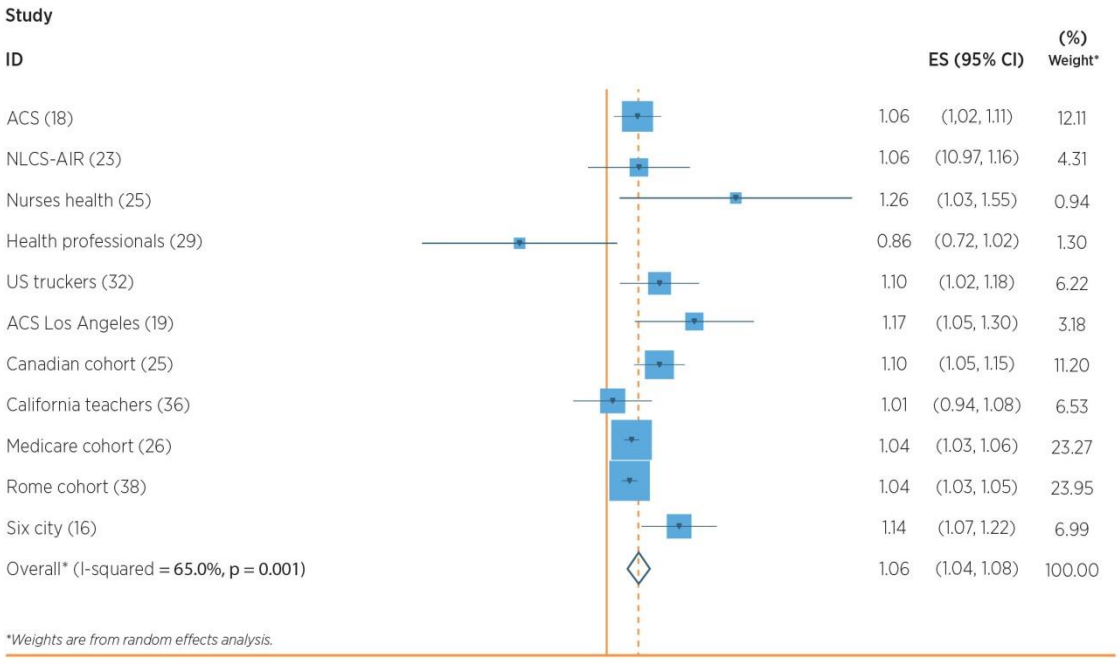
cardiopulmonary disease, a very broad category which includes ischemic heart disease, stroke and chronic obstructive pulmonary disease (Jerrett et al., 2009; Krewski et al., 2009). However, a two-pollutant model with fine particles and ozone indicated that ozone was only associated with the risk of death for respiratory causes, which means that the primary effect of ozone is on respiratory (chronic obstructive pulmonary disease) and not cardiovascular diseases (ischemic heart disease and stroke) (Jerrett et al., 2009).

Over the past years, several entirely new cohort studies have been conducted in the United States, as well as in Europe, Asia and Oceania. These include the California Teachers study (Lipsett et al., 2011); the Nurses' Health study (Puett et al., 2009), the Male Health Professionals Study (Puett et al., 2011); the US Trucking Industry Study (Hart et al., 2011); the Electric Power Research Institute Veterans Cohort (Lipfert et al., 2000); the energy workers (Bentayeb et al., 2015); the Taiwanese civil servants (Tseng et al., 2015); the Seventh Day Adventist study (Chen et al., 2005); the Dutch Diet and Cancer study (Beelen et al., 2008); the Women's Health Initiative (Miller et al., 2007); the US Medicare National Cohort (Zeger et al., 2008); the Vancouver Cohort (Gan et al., 2011); the Canadian National Cohort Study (Crouse et al., 2012), and the Rome Cohort Study (Cesaroni et al., 2013).

Although these cohort study results are qualitatively consistent, there is inevitable heterogeneity among the values of the risk coefficients estimated from observational epidemiological studies (Höek et al., 2015; Shin et al., 2015; Atkinson et al., 2016). Variability in the coefficients may be explained by the fact that each cohort study yields an estimate of the concentration-response relationship for a different population sample. Some of these cohorts consist of population sub-groups with specific characteristics, pre-existing medical conditions, or particular occupational groups (e.g., nurses, truckers, teachers). Also, there are differences in study specifications, analytical methods, as well as in the many elements that comprise the causal chain of the exposure-response relationships. Among these, relevant factors include the nature and mixture of pollutant sources, the composition of emissions, the elemental or source composition of the particulate matter inhaled, the

duration and time-trends of pollutant concentrations during the study period, air exchange rates in buildings, individuals time-activity patterns and behavior, meteorology, demographics and socioeconomic position distribution within populations, among others.

Figure 3.4. Meta-analysis of the association between chronic $PM_{2.5}$ exposure and all-cause mortality (Relative Risk per 10 µg/m^3)



Source: Höek et al., 2013.

Figure 3.4 summarizes the results from cohort studies that have evaluated long-term exposures to fine particulates and total mortality (Höek et al., 2013). It illustrates the variability discussed above and also provides a summary estimate of the weighted average coefficient from the studies derived by meta-analysis.

The scientific community has mostly reached consensus on the causal relationship between $PM_{2.5}$ and increased mortality from cardiovascular and pulmonary diseases (Shin et al., 2015). The causality between ozone and respiratory mortality is not yet supported with as ample evidence (Atkinson et al., 2016).

The risk assessment that constitutes the next phase of this project builds upon the information that has been presented in this report, air pollution data from the 1990s to 2015, and results from epidemiological studies. Phase II aims at estimating the health benefits, as well as variability and uncertainty sources that influence estimates accrued from improvements in air quality that have occurred in Mexico City during this time-period.

Phase II. Estimation of the Health Benefits of Air Pollution Improvements in Mexico City

4. Risk Assessment for Policy Making

The risk assessment that constitutes Phase II of this project integrates air pollution and mortality data from 1990 to 2014, and uses results from epidemiological studies to estimate health benefits from improvements in air quality that have occurred in Mexico City (CDMX) during this time-period.

Risk assessment consists of four elements:

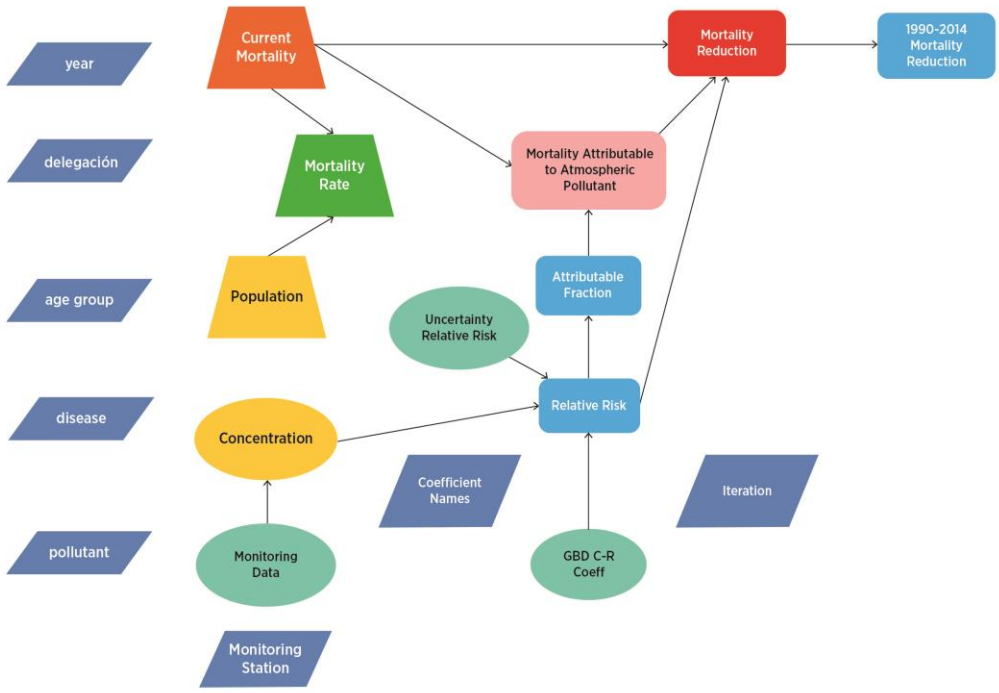
1) Hazard identification
2) Exposure assessment
3) Concentration-response analysis, and
4) Risk characterization.

The first element, ***hazard identification***, is qualitative and seeks to characterize the nature and strength of evidence underlying claims that exposure to a pollutant can impact health. The material discussed in the preceding phase I report of this project serves this purpose – demonstrating clearly that there is sufficient evidence to support the view that exposure to ambient fine particulate matter and ozone results in reductions in survival and increases in mortality.

The remaining three elements of risk assessment – exposure assessment, concentration-response analysis, and risk characterization – are all quantitative. Because risk assessors realize that the public, and decision makers who serve them, need to understand 'how good' estimates of risk are, in both the ***exposure assessment*** and the ***concentration-response analysis*** phases of the analysis they seek to provide quantitative estimates of both the central tendency and the uncertainty of key parameters. In the final ***risk characterization*** stage, risk assessors rely on tools which allow them to study the propagation of uncertainty throughout the entire risk model and to provide (i) central estimates of health

impacts; (ii) estimates of the uncertainty in these values; and (iii) information about the contributions of uncertainty in various key parameters to the overall uncertainty in estimates of risk.

Figure 4.1. Risk Assessment conceptual model for health benefits associated with air quality improvements in Mexico City, 1990-2014



Source: Prepared by the authors.

In this report we provide an introduction to ideas central to understanding the nature, elements, and results of the risk assessment for CDMX air quality improvements during the past 25 years. We use the risk assessment methodology as a tool to evaluate the health benefits that result from air pollution reductions, achieved by the ensemble of public policy strategies that have been implemented in the city. Because regulations oriented at reducing air pollution diminish premature mortality risk in large populations (and the individuals that are affected are unknown), we use the number of 'premature deaths avoided' to evaluate the health benefits attributable to

air quality improvements. In this context, it is key to note that air pollution controls or any other public policy intervention cannot save lives. Lives can only be extended. Thus, the metric of 'premature deaths avoided' is in fact a *proxy* for "life expectancy increases".[34]

a. Recent Risk Assessment Findings Worldwide and for Mexico

Several recently published risk assessments have evaluated the impact of air pollution exposure on premature mortality. A comparative risk assessment known as the Global Burden of Disease (GBD) with base year 2010 and updated for 2013 found that ambient air pollution is among the first 10 most important risk factors worldwide. Among the close to 70 risk factors evaluated in these 2010 and 2013 studies, particulate matter ranked 9th and 10th, respectively. Both assessments estimated that fine particles cause around 3 million excess deaths, with 95% uncertainty intervals ranging between 2.6 million and to 3.6 million deaths (Lim et al., 2013; Forouzanfar et al., 2015). Furthermore, GBD analyses also showed that ambient ozone ranks in the 21st position within all the risk factors that were assessed in 2013. Ozone adversely impacts premature mortality with a toll of approximately 200 thousand deaths (95% UI: 161 thousand deaths to 272 thousand deaths) (Forouzanfar et al., 2015).

In a similar manner, Lelieveld and coauthors estimated worldwide mortality due to fine particles and ozone in 2010 at 3.3 million (Lelieveld et al., 2015). Results of both GBD assessments and of Lelieveld et al. show that the major causes of death that contribute to mortality associated with exposure to $PM_{2.5}$ (particles with an aerodynamic diameter of less than 2.5 microns, also called fine particles) are ischemic heart disease, cerebrovascular stroke, and lung cancer.

[34] In risk assessment and policy analysis the terms "premature deaths avoided", "reduced mortality risk", "reduced premature mortality", "avoided deaths", "excess deaths" and "lives saved" are used to refer to the benefits derived from strategies that target air pollution reductions. For the present project these terms are used indistinctly.

These burden of disease evaluations and risk assessments are calculated with reference to an alternative or counterfactual exposure, or, to the exposure that may allow the greatest improvement in the health of the population (Stevens et al., 2008; Lim et al., 2013; Forouzanfar et al., 2015; Lelieveld et al., 2015). Since current epidemiological research has not identified a threshold for $PM_{2.5}$ nor for ozone exposures (that is, a concentration under which no adverse health effects are observed) the minimum pollutant concentrations at which health effects have been observed are often used as counterfactuals (Pope et al., 2002; Cohen et al., 2004). Effects have been seen down to concentrations in the range of 5.9 – 8.7 $\mu g/m^3$ annual average $PM_{2.5}$ concentrations, and of 33.3 – 41.9 ppb seasonal (warmer six months assumed to have higher ozone concentrations) hourly maximum ozone concentrations (Pope et al., 2002; Jerrett et al., 2009; Stevens et al., 2008; Lim et al., 2013; Forouzanfar et al., 2015).

Very few risk assessments have been conducted for Mexico or Mexico City. One such study found that fine particle exposures at concentrations estimated for 2001 to 2005 are responsible for 7,600 annual excess deaths per year (Stevens et al., 2008). This burden of disease resulted from comparing nation-wide average $PM_{2.5}$ levels of approximately 23 $\mu g/m^3$ (range between about 15 and 40 $\mu g/m^3$)[35] with an alternative scenario deemed the lowest concentration at which health impacts were observed in cohort studies (7.5 $\mu g/m^3$). The authors estimated that 38% of those deaths (2,900 deaths) occurred in the Metropolitan Area of the Valley of Mexico (MAVM)[36], as a result of its large population which accounts to roughly 35% of Mexico's urban population. A second study conducted in Mexico City with 2005 data reported that a reduction of annual average PM_{10} (particles with an aerodynamic diameter of less than 10 microns) levels from slightly over 50 to 20 $\mu g/m^3$ –level set as the WHO air quality recommended value to protect public health-- would prevent

[35] Annual average $PM_{2.5}$ concentrations estimated by the authors from stratified mean $PM_{2.5}$ concentrations and percent of population assigned to each category in Stevens et al., 2008.

[36] The definition of the MAVM that prevailed until 2005, period during which this study was conducted (2001 to 2005), included 16 boroughs (called *delegaciones*) of the then called Federal District, and 18 boroughs (called *municipios*) of the State of Mexico (INEGI, 2016).

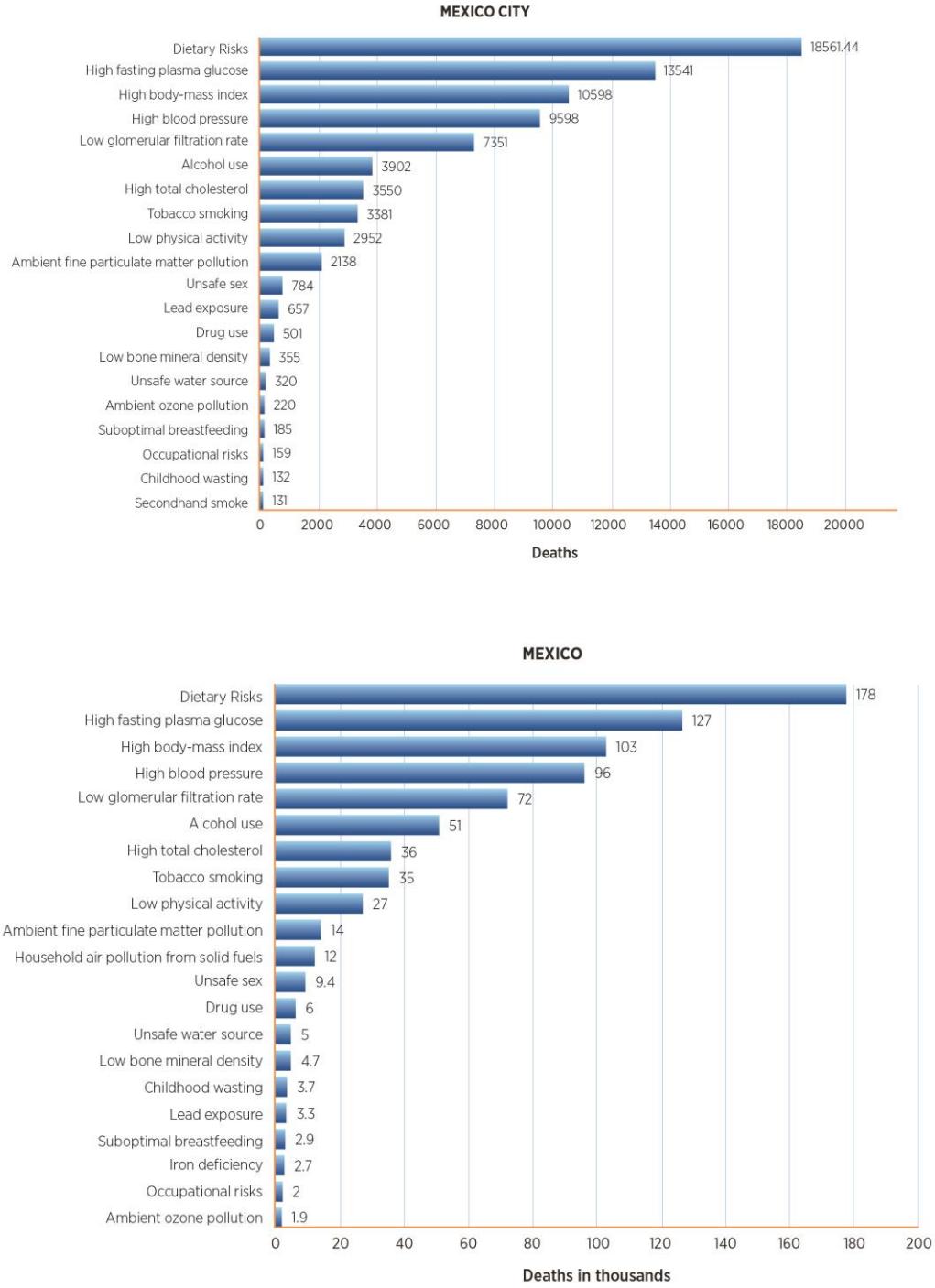
more than 6,100 annual deaths from total mortality in people over 30 years old (Riojas-Rodríguez et al., 2014).[37]

More recently, the GBD 2010 and 2013 included the per-country and state analysis, including Mexico and Mexico City (Fig. 4.2). GBD results show that ambient exposures to $PM_{2.5}$ and ozone are among the first ten and 21st risk factors, respectively, of the almost 70 risk factors that were evaluated. These two assessments find that between 13,200 and 13,700 premature deaths could be attributed to $PM_{2.5}$ exposure reductions. Such results stem from a countrywide fine particle annual average reduction of close to 50%, from 13.0 µg/m^3 (with a 95% uncertainty interval between 11.7 to 12.2 µg/m^3) to the counterfactual modelled as a range between 5.9 and 8.7 µg/m^3 (IHME, 2016).[38] For ozone, the number of premature deaths is smaller and it is estimated to range approximately between 1,800 and 1,900. These excess deaths are caused by a reduction of almost 50% in seasonal hourly maximum ozone concentrations, from 60 ppb (with a 95% uncertainty interval between 57 and 64 ppb) to a range of between 33.3 and 41.9 ppb (Lim et al., 2013; Forouzanfar et al., 2015; IHME, 2016). For Mexico City, the GBD results, recently released, indicate that exposure to fine particles is responsible for over 2,100 premature deaths. Again, ozone's toll is smaller, with approximately 220 premature deaths for 2013 (IHME, 2016). For the Mexico City analysis baseline (2013) fine particle and ozone concentrations that are evaluated versus the alternative levels were not available.

[37] This study did not estimate avoided deaths from long-term ozone exposure reductions. However, a short-term exposure decrease from 65 to 50 ppb (maximum 8-hour moving average) using a concentration-response function from a meta-analysis of time-series studies, resulted in an estimate of 390 avoided deaths (Riojas-Rodríguez et al., 2014).

[38] The GBD 2010 analysis used an almost identical range for the alternative concentration, 5.8-8.8 µg/m^3 (Lim et al., 2012)

Figure 4.2. Main risk factors and premature mortality for Mexico (lower graph) and for Mexico City (upper graph) in 2013



Source: Prepared by the authors with information from IHME, 2016 and Forouzanfar et al., 2015.

Several methodological inconsistencies between the GBD work and the comparative risk assessment of both, Stevens and coauthors (2008) and Riojas-Rodríguez and coauthors (2014), may explain higher excess death estimates in the latter assessments. Of main importance is that the latter authors applied functions that relate fine particle concentrations with the health response as a linear function. Applying such functions at concentrations that exceed fine particle levels observed in the epidemiological studies that yield the relative risks, results in higher excess deaths than when utilizing a more realistic function that allows to flatten the risk above certain concentrations (as will be extensively discussed in section **2.c. Concentration-Response Analysis**). Fine particle upper range concentrations in Stevens and in Riojas-Rodríguez were 39 $\mu g/m^3$, and 35 $\mu g/m^3$, respectively, whereas the upper range found in cohort studies did not exceed 30 $\mu g/m^3$.

5. Risk Assessment and Air Quality Improvements in Mexico City, 1990-2014

In the early 2000's a group of researchers, led by Luisa Molina and Mario Molina, conducted an analysis of air pollution in Mexico City Metropolitan Area[39] (Molina & Molina, 2002). One element of their work was an assessment of the health benefits expected to accompany a 10% reduction in the levels of inhalable particulate matter (PM_{10}) throughout the entire Mexico City Metropolitan Area. Results showed that approximately 2000 premature deaths could be avoided each year if levels of inhalable particles were reduced by 10% from the approximately 80 $\mu g/m^3$ annual average concentrations reported at the time. The estimate relied on evidence from the cohort studies, using the concentration-response coefficient from the 1995 reanalysis of the ACS study. It was assumed that fine particles were responsible for the entire effect seen in the ACS study, and that in the late 1990's and early 2000's 60% of PM_{10} in Mexico City Metropolitan Area was fine particulate matter. The above implied an increase in mortality rates of 0.3% with an increment in 1 $\mu g/m^3$ of PM_{10}. The baseline mortality rate used in the analysis was 10 deaths/10,000 person-years among adults (people 30 years of age and older) in Mexico City Metropolitan Area. Twenty million inhabitants populated the entire Metropolitan Area at that time.

As noted by Molina and Molina, several issues confronted anyone interested in estimating health risks from particulate matter in Mexico using evidence from the United States cohort studies. First, the mortality estimate was based on a study that was not conducted in Mexico. However, the results were supported qualitatively by the consistency of results from time-series mortality studies carried-out locally with results from similar studies from other countries, including the United States. Second, it was unclear whether the slopes of the concentration-response functions from cohort studies conducted in the United States would be relevant to estimating

[39] Throughout the book the authors referred to the Mexico City Metropolitan Area, which was not explicitly defined. However, the figures presented on number of inhabitants, vehicles, and extension imply that it's equivalent to the official term of the Metropolitan Area of the Valley of Mexico (INEGI, 2016).

health impacts in Mexico City, where PM levels were much higher. It was in part because of this concern that the Molina analysis focused on estimating the impact of a 10% reduction in PM_{10} levels instead of attempting to estimate the overall mortality impact attributable to current ambient levels of PM in Mexico City. Third, there are significant differences in terms of population and baseline mortality rates, in the age structures and disease-specific causes of mortality between Mexico and the United States. All of these differences imply that such estimates must be viewed as plausible, but less precise than those obtained when risk assessments are conducted in settings more similar to those where the underlying epidemiological studies have been conducted.

In this project we estimate the health benefits associated with air quality improvements in Mexico City since the early 1990's and up to 2014. We use the methods that have been recently refined to better estimate the mortality impact attributable to higher concentrations of pollutants observed in Mexico City in the 1990s than those observed in the cities where cohort studies have been conducted. During the period of interest, Mexico City has grown tremendously, in terms of population, vehicle use, commercial activity, and energy use. For instance, the population grew from 8.4 million to almost 8.9 million inhabitants from 1990 to 2015 (INEGI, 2016). Similarly, energy use showed a growth rate of 1.3% (1992 to 2012), with transportation being the largest contributor accounting for over 60% of total energy consumption in the city.

Our analysis does not take account of the large increases in air pollution that would have been expected during this period if the government had made no effort to reduce air pollution. It takes credit only for the reduction in air pollution levels from their 1990 levels, not for the differences between the levels that would have been expected given the growth in population, vehicle use, commercial activity and energy use. In this sense our estimates of the impacts of government regulations almost certainly underestimate the real benefits that have been achieved.

In this report, health benefits have been estimated for the 1990 - 2014 period by considering the air quality improvements achieved every year. Our analyses start in 1990 with data from monitoring sites used either to compute our exposure estimates (as happened with ozone) or to predict concentrations from a model that includes covariates directly measured in stations that were already in operation. Data for health outcomes were available from official sources since the beginning of our analyses. We selected 2014 as the final year for analysis because it is the most recent year with available official health (mortality) data.

a. Exposure Assessment for $PM_{2.5}$ and Ozone in Mexico City[40]

The exposure assessment provides quantitative information on the pollution levels to which the population in CDMX is exposed. Our analysis covers a period of 25 years. The exposure metrics used in the risk assessment for 1990 through 2014 are: (i) for $PM_{2.5}$, the annual average concentration in each *delegación* and year, and (ii) for ozone, the seasonal average of the daily 1-hour maximum ozone concentrations in each *delegación* and each year. These estimates are derived from data collected by the network of fixed-site monitoring stations in Mexico City run by SEDEMA.

Exposure estimates for $PM_{2.5}$ result from predicted values that used measurement values from monitoring sites. Measurements of fine particle concentrations began by the end of 2003. $PM_{2.5}$ concentrations from 2004 through 2014 are available from five stations that monitor particles with manual sampling equipment.[41] For the years with no direct $PM_{2.5}$ measurements (1990 to 2003) a predictive model (Generalized Additive Model, GAM) was developed to estimate $PM_{2.5}$ concentrations. This model

[40] Monitoring data was provided by Armando Retama, Director of Atmospheric Monitoring, General Direction on Air Quality Management, SEDEMA, Government of Mexico City.

[41] The Federal Reference Method is the Sierra-Anderson High Volume Air Sampler System. Data from these sites is used for regulatory purposes by environmental authorities in CDMX. Data from 9 additional sites that measure $PM_{2.5}$ with automatic samplers were not included because there have been changes in the sampling method and adjustments to control for volatile losses through the years.

used as main a-priori predictor PM_{10} concentrations that were measured at these five sites.

Uncertainty in the estimates of the annual mean $PM_{2.5}$ exposure in each *delegación* and each year arises due to – (i) fundamental errors in the measurements themselves; (ii) the need to estimate the annual mean from measurements taken at 6 day intervals throughout the year; (iii) the need to estimate the exposures in each *delegación* from a small number of monitoring sites; and (iv) for the years before 2004, the necessity to estimate $PM_{2.5}$ values based on a predictive model that includes measurements of PM_{10}, meteorological conditions (relative humidity, temperature and wind speed), and variables reflecting seasonality (month) and time trends (year). Of these, the largest source of uncertainty is the need to predict values for the early years and so the uncertainty in $PM_{2.5}$ estimates decreases over time.

Ozone exposure estimates are computed directly from monitoring data because measurements started since the 1970. The number of ozone monitoring sites has grown through the years. During the early years of our study period (1990 to 1993) measurements were available for 2 to 6 sites. Since 1994, data from between 10 and 25 sites are available. A paucity of measurements for the earlier years and a more abundant set for the latter is reflected in smaller ozone exposure uncertainties over time.

The continuous efforts of CDMX environmental authorities, SEDEMA, to improve methods for quality control and quality assurance protocols, together with regular external audits, have led in more recent years to reduced uncertainty in fine particle and ozone measurements, and thus in our exposure estimates.

i. Annual Average $PM_{2.5}$ Concentrations at Monitoring Sites

The five stations that measure particles ($PM_{2.5}$ and PM_{10}) using manual sampling equipment are located at Pedregal (PED), UAM-Iztapalapa (UIZ), Tlalnepantla (TLA), Merced (MER), and Xalostoc (XAL). These stations are in the southwestern, southeastern, northwestern, center, and northeastern areas of the Metropolitan Area

of the Valley of Mexico, respectively. PED, UIZ, and MER are located in Mexico City, and are mostly urban, with PED being the foremost residential site, and UIZ and MER a combination of small businesses and service areas. TLA and XAL are in the southerly portion of the neighbouring and very industrialized State of Mexico.

For $PM_{2.5}$ and PM_{10} daily values were obtained from every sixth day 24 hour-integrated measurements. Complementarily, daily values for meteorological variables were computed from hourly measurements. Relative humidity, temperature and wind speed are measured at the five sites for which we have PM data, PED, MER, UIZ, TLA and XAL. City-wide 24-hour averages for each meteorological variable were estimated for the days with measured PM_{10} and $PM_{2.5}$ concentrations.[42] City-wide average values were deemed convenient to represent the variability of the micro-climates within the city because there are only two large geographic climatic regions defined by topography and land use, the urbanized and the rural. The urbanized region is the one of most relevance to this project, for the majority of the population is settled there and for its high population density.

Completeness criteria for the project was set to 75% of valid measurements for all variables (For a full description of the criteria refer to Annex I). All sites had complete information for $PM_{2.5}$ (2004-2014) and for PM_{10} (1990-2014), except UIZ. For UIZ 1998 was the first year with $PM_{2.5}$ and PM_{10} measurements, and no PM_{10} data were available for 2003 and 2008, which implies that we could not predict fine particle concentrations for 1990 to 1998, 2003 and 2008.

Model Building for $PM_{2.5}$ Predictions

[42] SEDEMA's data for temperature and for relative humidity were incomplete in 2007. Missing data, 45 and 120 24-hour measurements for temperature (TEMP) and relative humidity (RH), respectively, were imputed from the meteorological network of the Program of High School Meteorological Stations (*Programa de Estaciones Meteorológicas del Bachillerato Universitario*, PEMBU, *Universidad Nacional Autónoma de México*, UNAM). Ordinary least-square regressions between these two data sets (2004 to 2014) for each of the variables showed a correlation coefficient of 0.99 for TEMP and of 0.98 for RH, and a t-test showed no significant difference between the means.

Twelve years (1994-2015) of daily $PM_{2.5}$, PM_{10} and meteorological data were included in the GAM. By developing a hybrid model that included PM_{10}, an important predictor of $PM_{2.5}$ as it accounts for between 30 and 60% of its mass (Stevens et al., 2008), plus wind speed, temperature and relative humidity, we were able to estimate meteorology-adjusted $PM_{2.5}$ concentrations. Annual average concentrations for $PM_{2.5}$ and PM_{10} for five sites and for the whole of the Metropolitan Area of the Valley of Mexico (MAVM)[43] for the period with direct simultaneous $PM_{2.5}$ and PM_{10} measurements, 2004 to 2014 are presented in Table 5.1.

Table 5.1. Annual average PM_{10} and $PM_{2.5}$ concentrations at fixed-site monitoring stations in the Metropolitan Area of the Valley of Mexico, 2004 – 2015

Fixed-Site Monitoring Stations												
Year	Pedregal		Merced		Tlalnepantla		UAM-Iztapalapa		Xalostoc		MCMA	
	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)
2004	33	18	51	24	55	24	45	22	88	36	54	25
2005	36	20	51	24	53	24	53	25	77	29	54	24
2006	32	18	51	23	50	22	49	23	74	30	51	23
2007	34	17	46	22	46	22	40	24	70	26	47	22
2008	37	18	56	23	53	22	n.a.	n.a.	72	26	55	22
2009	40	18	60	22	63	21	52	21	104	26	64	22
2010	33	14	55	21	53	19	48	18	102	25	58	20
2011	36	18	73	22	57	21	50	20	92	25	62	21
2012	36	17	56	21	51	20	47	19	69	26	52	21
2013	36	18	47	21	48	22	45	21	64	28	48	22
2014	32	16	42	19	44	19	41	20	52	27	42	20
2015	33	19	43	21	45	23	40	19	55	28	43	22

n.a. = Not available

Source: Estimated by the authors. Annual average observed $PM_{2.5}$ concentrations were calculated from daily measurements from monitoring stations (SEDEMA, CDMX).

[43] The definition of the MAVM that prevailed until 2005, period during which this study was conducted (2001 to 2005), included 16 boroughs of the then called Distrito Federal (called *delegaciones*) and 18 boroughs of the State of Mexico (called *municipios*). The definition was officially modified after December, 2005 to include 59 boroughs from the State of Mexico, 1 from the state of Hidalgo, and 16 from the Distrito Federal (INEGI, 2016).

The relationships between $PM_{2.5}$ and PM_{10}, the main a-priori predictor, relative humidity, mean temperature, wind speed, year, and month were evaluated with a GAM of the following form:[44]

$$PM_{2.5measured} = \alpha + \beta_1 PM_{10measured} + \beta_{2i}\,station + \beta_{6i}\,month + s(year) + s(ws) + (temp) + s(rh) + \varepsilon_{ij} \quad \text{(Model 1)}$$

Where α is the regression intercept, and the β correspond to the regression coefficients for station (β_{2i}, i=2-5) and month (β_{6i}, i=6-16), included as categorical variables. Year, wind speed, temperature, and relative humidity are modelled as continuous variables with penalized smoothing spline functions, s(x), to characterize nonlinear relationships.

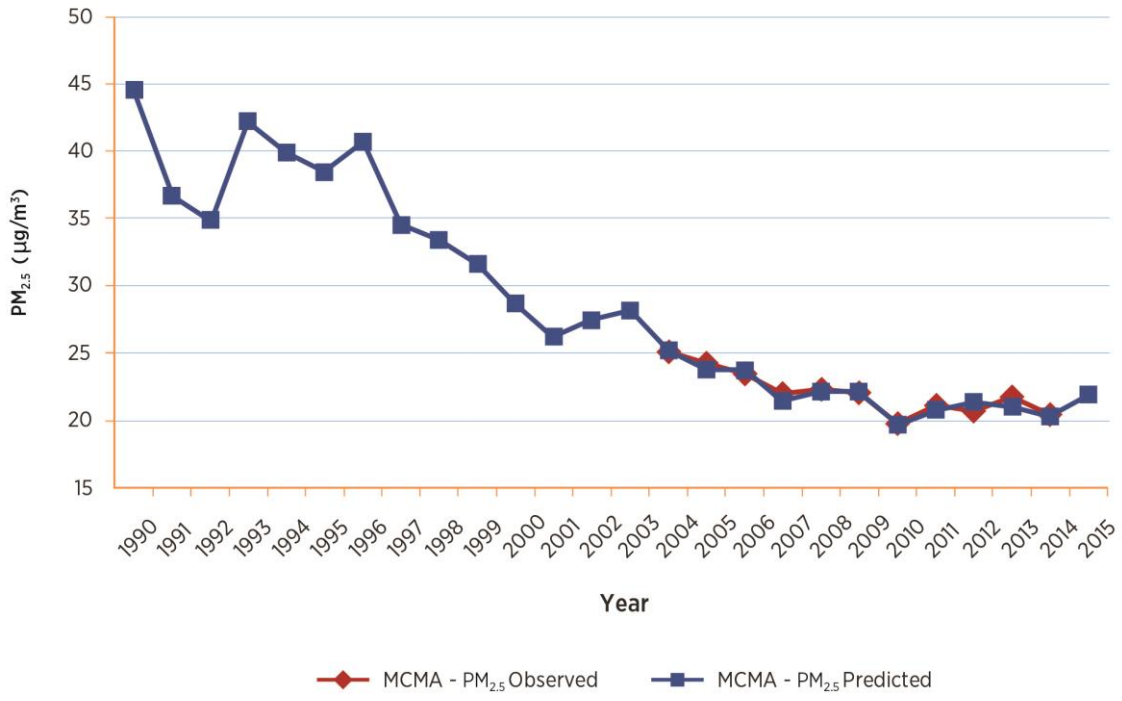
The criteria to select this model (Model 1) included having the most complete data for the covariates to allow predicting the least uncertain and largest possible set of $PM_{2.5}$ daily concentrations. Model 1 predicted 6761 24-h $PM_{2.5}$ concentrations, and had an adjusted R-square equal to 0.73. In comparison, a marginally better performing predictive GAM also included SO_2 and NO_2 (Model 2 not shown, adjusted R-square = 0.77). However, missing data for these two gaseous pollutants were substantial. From 1990 to 1993 all stations were lacking measurements for SO_2 and most of them for NO_2. In 1994, 1997, and 2002 one station missed SO_2 data. In 1999, 2000, and 2014, two, three and one station, respectively, missed NO_2 values. Such data gaps would have resulted in a lack of almost 2000 $PM_{2.5}$ predicted values -equivalent to almost 7 years of every 6th day 24-hour value- distributed among the five stations, which would have added uncertainty to our exposure estimates.[45]

[44] Jhun and coauthors (2013) developed a similar GAM in Santiago, Chile. While the objective of building the model was to analyze anthropogenic impacts in fine particle concentrations, it showed that wind speed was the most important predictive variable. Even after adjusting for wind speed, temperature and relative humidity, seasonal variability –modeled as month- still remained (Jhun et al., 2013).

[45] There was a slight difference between Model 1 and Model 2 in terms of goodness of fit indicators, Akaike Information Criterion (AIC) and Residual Maximum Likelihood (REML). Model 1, AIC = 18979.51, REML = 9495; Model 2, AIC = 16950.55, REML = 8484.7.

Figure 5.1. shows annual average $PM_{2.5}$ concentrations for the MAVM, computed from annual average values from the five monitoring sites. The graph includes predicted meteorology-adjusted (1990-2015) and observed (2004-2015) concentrations. **Appendix I** contains the graphs for all sites. This appendix also comprises GAM resulting independent variable parametric coefficients, and the significance of smooth terms. Table 5.2 shows annual average predicted meteorology-adjusted $PM_{2.5}$ concentrations and the corresponding standard error of the mean[46] for the five sites, computed from daily concentrations derived from the GAM for 1990 to 2015.

Figure 5.1. Annual average observed and predicted $PM_{2.5}$ concentrations for the Metropolitan Area of the Valley of Mexico, 1990-2015



Source: Estimated by the authors. Annual average predicted $PM_{2.5}$ concentrations were computed from daily concentrations derived from the Generalized Additive Model. Annual average observed $PM_{2.5}$ concentrations were calculated from daily measurements from five monitoring stations (data from SEDEMA, CDMX).

[46] The standard error of the mean was computed for each year and site from 24-hour concentrations.

Table 5.2. Annual average predicted $PM_{2.5}$ concentrations for five sites in the Metropolitan Area of the Valley of Mexico, 1990-2015

Year	Fixed-Site Monitoring Stations											
	TLA		MER		UIZ		PED		XAL		ZMVM	
	Annual average and standard error or the mean $PM_{2.5}$ concentrations (μg/m³)											
	Annual average	SE	Annual average	SE	Annual average	SE	Annual average	SE	Annual average	SE	Annual average	SE
1990	42	1.5	40	1.3	n.a.	n.a.	31	0.9	66	1.8	45	1.4
1991	35	1.5	34	1.2	n.a.	n.a.	31	1.6	46	2.5	37	1.7
1992	33	1.3	31	1.1	n.a.	n.a.	27	1.3	47	2.7	35	1.6
1993	39	1.6	35	1.6	n.a.	n.a.	29	1.1	66	2.7	42	1.8
1994	41	1.7	34	1.8	n.a.	n.a.	28	1.0	57	2.4	40	1.7
1995	38	1.6	35	1.4	n.a.	n.a.	26	1.1	55	2.3	38	1.6
1996	36	1.8	35	1.5	n.a.	n.a.	26	1.2	67	2.7	41	1.8
1997	32	1.5	30	1.5	n.a.	n.a.	22	1.4	54	2.8	34	1.8
1998	31	1.9	31	1.7	32	1.7	24	1.4	49	2.4	33	1.8
1999	28	1.8	28	1.1	27	1.2	22	0.9	54	2.1	32	1.4
2000	26	1.1	26	1.2	28	1.3	21	1.0	43	2.3	29	1.4
2001	25	1.0	25	1.0	25	1.0	21	0.9	35	1.5	26	1.1
2002	27	1.1	27	1.1	26	1.4	22	1.0	34	1.4	27	1.2
2003	25	1.1	29	1.4	n.a.	n.a.	22	1.1	37	1.5	28	1.3
2004	25	1.0	24	1.1	24	1.4	19	1.0	34	1.3	25	1.2
2005	24	1.2	23	1.2	24	1.3	19	1.0	29	1.5	24	1.3
2006	23	0.9	24	1.1	24	1.1	19	0.8	29	1.4	24	1.1
2007	21	0.8	21	1.0	20	0.9	18	0.9	27	1.2	21	1.0
2008	22	1.0	23	0.9	n.a.	n.a.	18	0.8	26	1.1	22	1.0
2009	22	1.0	21	1.0	19	0.9	16	0.8	33	1.8	22	1.1
2010	18	1.0	19	1.3	17	1.1	13	0.8	31	1.9	20	1.2
2011	19	1.1	24	1.4	18	1.2	14	0.9	28	1.7	21	1.3
2012	21	0.9	22	1.1	21	1.0	17	0.8	25	1.3	21	1.0
2013	21	1.0	21	1.1	21	1.0	18	0.9	24	1.3	21	1.0
2014	21	0.7	21	0.8	21	0.8	17	0.6	23	1.2	20	0.8
2015	23	0.8	22	0.8	22	0.7	19	0.7	24	0.9	22	0.8

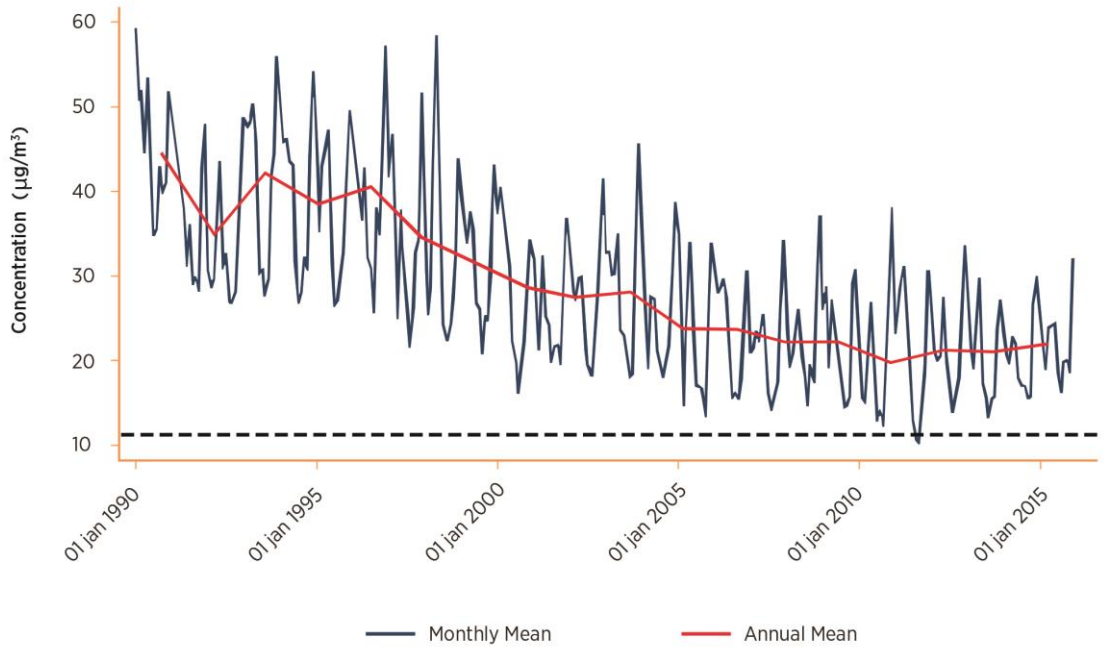
n.a. = Not available

Note: For UIZ site, lack of PM_{10} monitoring data between 1990-1997 and missing data for 2003 and 2008 resulted in the inability of the GAM to predict $PM_{2.5}$ concentrations for these years.

Source: Estimated by the authors. Annual average predicted $PM_{2.5}$ concentrations were computed from daily concentrations derived from the GAM.

Predicted $PM_{2.5}$ concentrations in Figure 5.2 reflect the substantial air quality improvements in fine particle concentrations since the early 90s achieved by environmental authorities in CDMX. Annual average $PM_{2.5}$ concentrations for the early 1990s were frequently in the range of 50 to 60 µg/m^3, and by 2007 there was an average decrease of about 20 µg/m^3. After 2008 the trend has levelled off, and since 2010 a slight increase of less than 10% has been observed. Concentrations It should be added, that the annual average $PM_{2.5}$ Maximum Allowable Limit (15 µg/m3 until 2014, and reduced to 12 µg/m^3 since that year) has been exceeded throughout the 1990 – 2014 period.

Figure 5.2. Annual and monthly average predicted $PM_{2.5}$ concentrations for the Metropolitan Area of the Valley of Mexico, 1990–2015



Note: The dotted line corresponds to the Mexican ambient air quality standard (12 µg/m^3).

Source: Estimated by the authors. Annual and monthly average predicted $PM_{2.5}$ concentrations were computed from daily concentrations derived from the Generalized Additive Model for five monitoring stations.

ii. Maximum Hourly Ozone Concentrations at Monitoring Sites

The sites that monitor ozone are located throughout the City except for the most southerly areas, which are more rural and less densely populated.

Ozone is measured with continuous sampling equipment and hourly data are reported. Sufficiency criteria for ozone was set to 75% of valid measurements per day, per quarter, and yearly. (For a full description of sufficiency criteria refer to **Appendix II.**) In applying such criteria, the sites with valid data for our analysis varied by year. Between two and five sites with valid data were available from 1990 to 1993. Then the number of sites oscillated from between 8 and 19 up to 2011 (with the exception of 2008, with 21 sites), since 2012 there were over 22 sites, and by 2015 there were 27 sites.

The exposure metric for our risk assessment is the seasonal (six month) one-hour maximum ozone concentration. We selected this metric to be consistent with the metric used to evaluate exposure in the ACS cohort study, the most robust study that has reported an association with mortality, and from which we took the relative risk for our risk assessment (Jerrett et al., 2009). Cohort studies and the GBD have defined "seasonal" as the period during which higher ozone concentrations are observed, and have used averages of three or six months. For our study the behaviour of ozone concentrations year-round in Mexico City led us to define "seasonal" as the period between February 1st and July 31st. Highest ozone months are March through June, so we added one month prior and one after, and at the same time we excluded the months that have had historically the lowest ozone concentrations (September-December). Our definition is well in line with the Environmental authorities from Mexico City's definition of the "ozone season", which runs from the second week in February through June, when the rainy season begins (SEDEMA, 2014).

Ozone exposures for each site and each year were computed, first by selecting daily maximum 1-hour concentrations. Then, the average of all daily maximum 1-hour concentrations was estimated for each quarter. Finally, the average of the averages

of the first quarter and of the second quarter (six months) were computed for every year.

For this project “seasonal” is defined as the six-month period that tends to have elevated ozone levels in Mexico City. The first quarter ranges from February through April, and the second from May through July. Since 1990 and up to 2014, atmospheric *Precontingencias* and *Contingencias* from the PCAA have prevailed from February to May (and oddly enough, December), and have had the lowest frequencies in September and November (SEDEMA, 2016).

Figure 5.3. Seasonal (six month) one-hour maximum ozone concentrations by site in the Metropolitan Area of the Valley of Mexico, 1990-2015

Source: Estimated by the authors from hourly measurements from monitoring stations. The number of sites per year ranged from 2 to 27, depending on available and valid data (SEDEMA, CDMX).

Average seasonal one-hour maximum ozone concentrations, with the corresponding standard error of the mean,[47] were estimated for each monitoring site and for each year. Figure 5.3 presents the results for all monitoring sites in the MAVM. This figure shows that in early 1990 extremely high concentrations prevailed, with seasonal averages of daily 1-hour maxima commonly in the range of 120 to 180 ppb levels, and even reaching 200 ppb. By the late 1990 daily 1-hour maxima were of around 150 ppb and by 2010 they approached 100 ppb. Overall the gradual reduction of mean, minimum and maximum one-hour maximum averages reflects that air management policy strategies in the City have yielded positive results.

iii. $PM_{2.5}$ and Ozone Concentrations from Monitoring Sites to *Delegaciones*

In this section we describe the methods used to assign $PM_{2.5}$ and ozone concentrations to each of the 16 *delegaciones* in Mexico City,[48] for every year between 1990 and 2014. To evaluate the health impacts of air quality improvements, $PM_{2.5}$ and ozone exposure estimates for each *delegación* are needed since official data for mortality statistics, the main health outcome in our risk assessment, are aggregated at the *delegación* level.

Each *delegación* is assigned $PM_{2.5}$ and ozone concentrations based on values estimated at monitoring sites using geostatistical interpolation methods.[49] For both pollutants a mean value and a standard error of the mean for each *delegación* and for each year were interpolated based on the corresponding yearly values of the

[47] The standard error of the mean was computed for each quarter and then averaged over the two high ozone quarters selected for this analysis. The mean was estimated from the 1-hour maximum averages for each quarter (for each year).

[48] In Mexico City the *delegaciones* (equivalent to US counties) are: Azcapotzalco, Coyoacán, Cuajimalpa de Morelos, Gustavo A. Madero, Iztacalco, Iztapalapa, La Magdalena Contreras, Milpa Alta, Álvaro Obregón, Tláhuac, Tlalpan, Xochimilco, Benito Juárez, Cuauhtémoc, Miguel Hidalgo, and Venustiano Carranza.

[49] Data were geoprocessed with ArcGIS 10.1® (Environmental System Research Institute; Redlands, CA, USA). Layer of monitoring sites was generated with information from SINAICA (National System of Air Quality Information) and the webpage from each monitoring site.

mean and standard error of the mean that had been computed or estimated for each monitoring site and year.[50]

To develop concentration estimates for each *delegación*, the analysis was first conducted at a much finer spatial resolution – the basic geostatistical area (known as an AGEB) within an *delegación*.[51] The values assigned to each AGEB were determined by how close the AGEB was to one or more of the fixed site $PM_{2.5}$ or ozone monitoring sites. More specifically, the methods applied were Proximity Analysis (nearest monitor) and Inverse Distance Weighting (IDW). The first method simply assigns the concentration of the site that is closest to the AGEB –shortest straight line from the site to the AGEB. The IDW method integrates information of two or more neighbouring sites by applying weights that are a function of the inverse of the distance between the AGEB and each relevant monitoring site. Our analysis uses the square of the inverse distance ($1/distance^2$), which is the exponent most commonly used with IDW. As will be explained later in this section, our analysis reduced the influence of XAL over neighbouring AGEB by using a higher value of the exponent for this site.

Krieging, a method that also uses weights to estimate the average concentration of several sites, was considered as an alternative approach to use in this project. Weights result from variograms which are a function of distance that consider autocorrelation among sites. The selected method for the project was IDW, as a study conducted in the MAVM concluded that Krieging and IDW estimated $PM_{2.5}$ and ozone concentrations (measured as daily concentrations in 2008) were highly correlated (Pearson correlation coefficients equal to 0.94 and 0.97, respectively), with very similar means and standard deviations (Rivera et al., 2015).

[50] For this report when "mean and standard error" $PM_{2.5}$ or ozone concentrations are omitted when referring to monitoring site concentrations to be used for the spatial interpolation, their presence should be taken as implicit. The same principle applies when "mean value and a 95% confidence interval" for each *delegación* are dropped when referring to resulting concentrations at this level of resolution.

[51] Layer of AGEB was provided by INEGI (National Institute for Statistics and Geography) with data from the Population and Household Census 2010.

The following process was applied for the spatial interpolation of $PM_{2.5}$ and ozone.[52] Buffer zones, with 5 and 10 km radii, were drawn around each monitoring station. If an AGEB was within a single 5 km radii buffer zone, it was assigned the pollutant concentration (either $PM_{2.5}$ or ozone) from the monitoring site which defined that zone (nearest monitor). If the AGEB was within two or more 5 km radii buffer zones (intersections), it was assigned a concentration by IDW that would weigh the distance between the AGEB and each relevant monitoring site. AGEB that were assigned concentrations with the method of nearest monitor or with IDW using 5 km radii buffer zones were removed from the steps that follow for the 10 km radii buffer zones.

An AGEB that was within a single 10 km radii buffer zone, was assigned the pollutant concentration from the nearest monitor. If and AGEB was within two or more 10 km radii buffer intersections, IDW was applied to assign a weighted concentration by the distance between the AGEB and each relevant monitoring site.

Finally, if an AGEB did not lie within a 5 or 10 km radii buffer zone, it was assigned the pollutant concentration found at the nearest monitoring site.

Following the process described above, an AGEB may be classified into one of four groups that determine how the pollutant concentration is assigned: 1) AGEB that fell within a site's buffer zone (either 5 or 10 km radii) – nearest monitor method; 2) AGEB that fell within a two site intersection zone (5 or 10 km radii) – IDW of the two sites; 3) AGEB that lie within a three or more site intersection zone (5 or 10 km radii) – IDW of the three or more intersecting sites; and 4) AGEB that did not lie within any of the buffer zones – nearest monitor method. Less uncertainty in exposure estimates is expected for AGEB in group 3, especially those within intersection zones of 5 km radii, because there is a larger number of sites to interpolate

[52] The spatial interpolation methods will be described for $PM_{2.5}$ and ozone. Due to the fact that the starting point for the interpolation are the concentrations at the monitoring sites and that these differ among pollutants, we will first describe the steps that are shared by both pollutants, and will follow with the specificities for each one.

concentrations from. In contrast, more uncertainty is expected for AGEB in group 4, which lied further away from any of the monitoring sites.

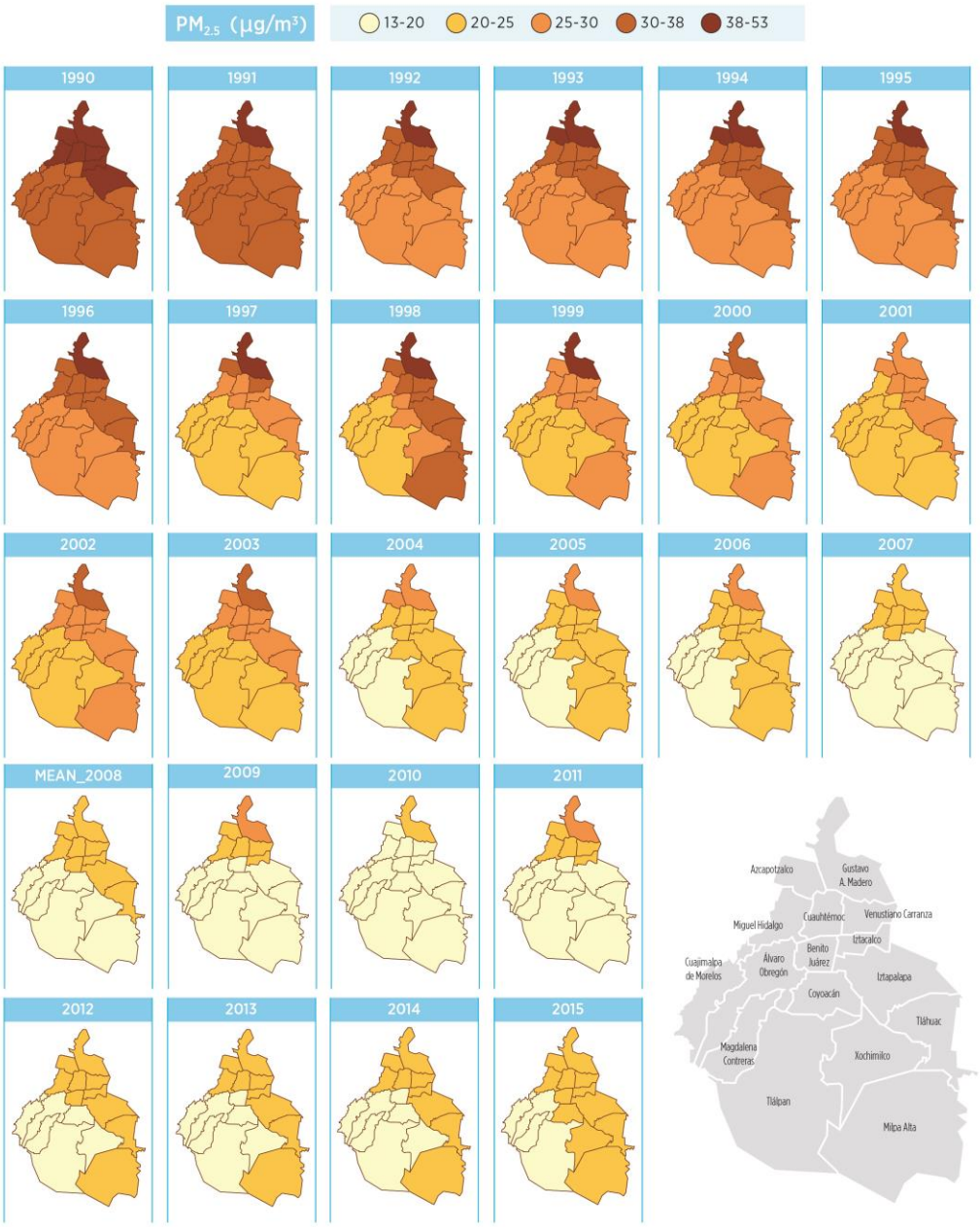
Five sites were used to predict spatial variation in $PM_{2.5}$ concentrations. The exception were the years for which site UIZ had no $PM_{2.5}$ predicted concentrations (1990 through 1997, 2003 and 2008). When 5 km buffer zones were drawn, only the zones of MER and UIZ sites intersected, so AGEB within this intersection were assigned a weighted average of the two values using $1/distance^2$. When 10 km buffer zones were drawn, there were seven cases in which two buffer zones intersected (MER-PED; MER-TLA; MER-XAL; MER-UIZ; TLA-XAL; PED-UIZ; XAL-UIZ), and three cases in which three buffer zones intersected (PED-MER-UIZ; TLA-MER-XAL; UIZ-MER-XAL). AGEB within two (or three) intersecting zones were assigned a weighted average of the two (or three) values using $1/distance^2$ weighting.[53]

The interpolation method gave XAL a special treatment. This site is impacted by local sources (an area with very heavy traffic and highly industrialized) and its measurements are unlikely to represent the exposure of populations further than 5 km from the station.[54] Thus, its area of influence over neighbouring *delegaciones* was restrained with a probability of 2/3 (downweight), and its influence was not restrained with a probability of 1/3 (non-weighted). To downweight the influence of XAL, only the 10 km buffer zone was drawn. AGEB within intersecting zones of XAL with other sites were assigned $PM_{2.5}$ concentrations using weights proportional to $1/distance^3$. Intersecting two buffer, and three buffer zones with XAL were TLA-XAL and MER-XAL, and TLA-MER-XAL. By down weighting the $PM_{2.5}$ concentrations, XAL exerted less spatial influence over the *delegaciones* than did the other four stations.

[53] Spatial interpolation analyses used only four sites (PED, MER, TLA and XAL) for the years for which UIZ had no $PM_{2.5}$ predicted concentrations (1990 through 1997, 2003 and 2008).

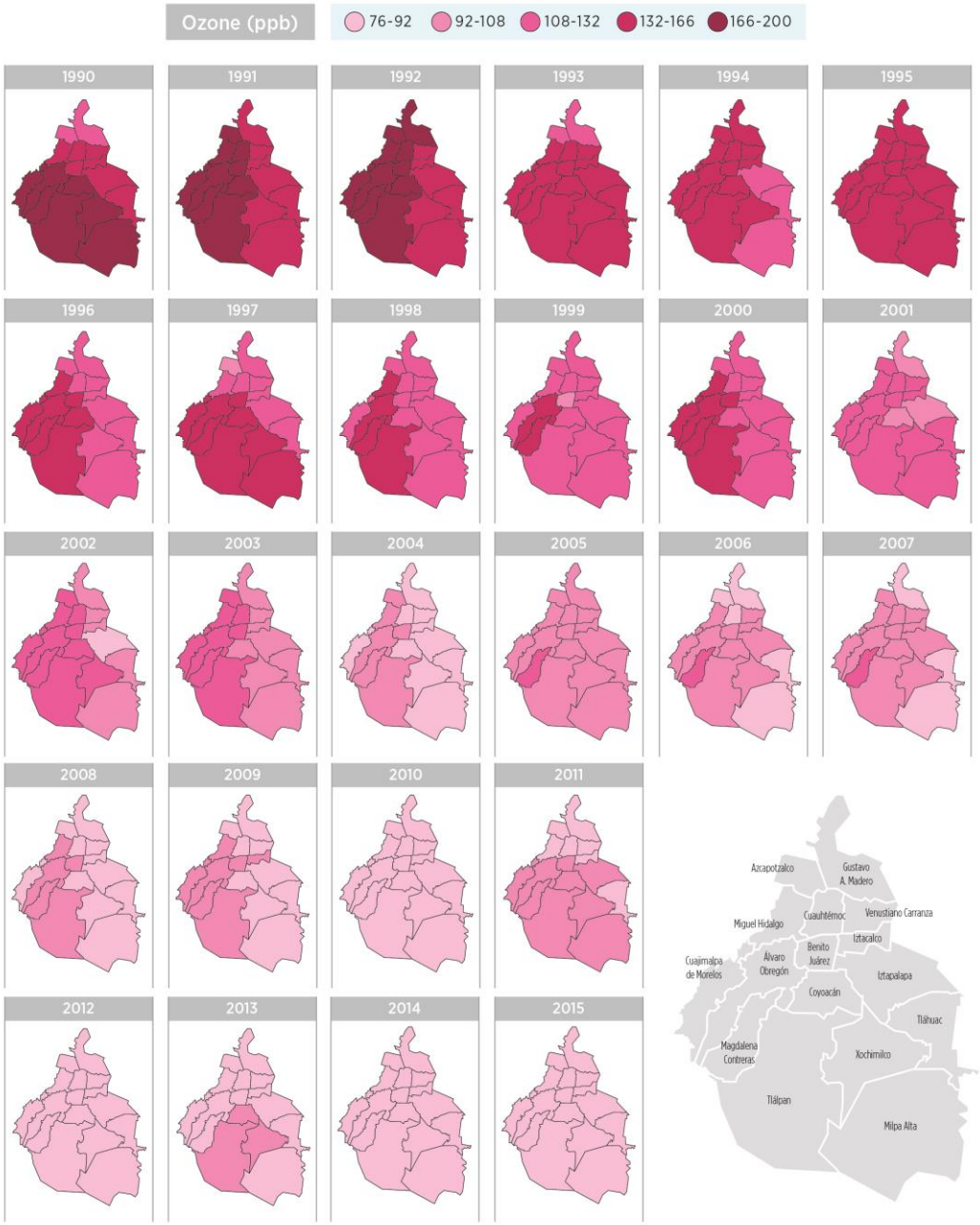
[54] Personal communication with Armando Retama, Director of Atmospheric Monitoring, General Direction on Air Quality Management, SEDEMA, Government of Mexico City.

Figure 5.4. Estimated annual average $PM_{2.5}$ concentrations at the *delegaciones* of Mexico City from 1990 to 2015



Source: Prepared and estimated by the authors. $PM_{2.5}$ concentrations were first predicted for five monitoring sites using a Generalized Additive Model, and further spatially interpolated to the *delegación* level.

Figure 5.5. Estimated seasonal (six month) daily 1-hour maximum ozone concentration at the *delegaciones* of Mexico City from 1990 to 2015



Source: Prepared and estimated by the authors. Ozone concentrations were estimated for each monitoring site and were further spatially interpolated at the *delegación* level.

For ozone there were many more sites than for fine particles for most years of the analysis to use for the spatial interpolation process. The number of sites varied from year to year, with between 6 and 27 sites used to predict spatial variations in ozone concentrations. Therefore, multiple AGEB were within intersecting 5 km radii buffer zones of two and more sites. Complementarily, even more AGEB fell within the 10 km buffer intersecting zones. AGEB that did not lie within any of the buffer zones or intersecting buffer zones were mostly in the southern most area of the CDMX, where AGEB are predominantly rural and less densely populated.

The interpolated pollutant concentrations for each AGEB within a *delegación* were then combined to calculate a mean value and corresponding standard errors of the mean for each *delegación* and year. The maps in Figures 5.4 and 5.5 show the estimated annual average $PM_{2.5}$ concentrations and 1-hour maximum ozone concentrations for the high ozone season, respectively, for the 16 *delegaciones* in Mexico City from 1990 to 2014.

The risk assessment that follows treated $PM_{2.5}$ exposures and ozone exposures in each *delegación* as normally distributed variables, with an estimated mean and 95% confidence intervals**.**

b. Mortality in Mexico City. Causes of Death Associated with Air Pollutants

Official mortality numbers for Mexico City were used in this project. In Mexico, general death statistics are under the responsibility of INEGI (National Institute for Statistics and Geography). Deaths are registered based on death certificates filled-out by physicians or others authorized by the Ministry of Health, and those certificates provide information on deaths by sex, age, cause, place of death, place of residence, and probable underlying causes of death, among other. Mexico follows the General Health Law (*Ley General de Salud*, LGS) as well as WHO International Classification of Diseases and Injuries (ICD) standards and rules, set to allow international comparability. Vital registration with medical certification of causes

used the ICD-9 from 1990 through 1997, and switched to tabulations of ICD-10 starting in 1998.

Statistics on mortality are released annually, and as early as 1893 they have been captured and reported by federal entity, by municipality (equivalent to *delegaciones* for Mexico City), and by locality (INEGI, 2014). Health authorities at state level –in the case of Mexico City the Secretariat of Health, SEDESA- are responsible for forwarding health mortality statistics to INEGI. INEGI integrates country-wide vital statistics and is responsible for their publication.

This analysis includes the causes of death associated with chronic exposures to ambient fine particles and ozone that were analysed in the 2010 and 2013 Global Burden of Disease studies (Lim et al., 2013; Forouzanfar et al., 2015) (Table 5.3). These causes of death were selected because as was discussed in Phase I, the epidemiological and scientific evidence point to a causal relationship between chronic exposures to fine particle, with more limited evidence for chronic exposures to ozone, and mortality due to specific diseases. Also, concentration-response functions developed for these relationships permit the estimation of the burden of disease, ie., attributable deaths to fine particle and ozone exposures.

Mortality data for 1990–2012 were obtained directly from SEDESA.[55] Data were stratified by year, cause of death, age-group, for each of the 16 *delegaciones* of Mexico City. A quality control process was followed to guarantee that all records complied with the characteristics specified for this project: place of death within a *delegación* of Mexico City, causes of death included in Table 5.3, by-gender and specific age-group stratification, and year of death (1990-2014). Additionally, some deaths were reassigned to a different year or cause of death than first appeared in the data set as briefly explained below (For a thorough description see **Appendix III)**.

[55] Information was provided by Jorge Gerardo Morales Velázquez, Director of Health Information, General Direction on Planning and Sectorial Coordination, SEDESA, Government of Mexico City.

INEGI releases official statistics by year of registry, and deaths that occur in November and December are frequently registered in January of the following year. November and December deaths are under 10% of deaths registered every year. However, we recoded the year of such deaths in order to assign them to the year in which they actually occurred.

One of the problems faced by cause of death registries is that deaths are assigned to causes that are not underlying causes of death (underlying cause of death should be codified) or that are immediate or intermediate causes of death. These wrongly assigned causes are sometimes known as 'garbage codes'.[56] There are multiple challenges in filling in death certificates, even by well-trained physicians. Several approaches, including algorithm development and physiology and ethology of diseases, have been combined to adjust for this problem by redistributing garbage codes to target causes, which are the probable underlying cause of death (Naghavi et al., 2010).

Garbage codes are redistributed to certain target causes that may vary by age and sex. An important target cause is cardiovascular disease, for which assigned deaths are expected to vary by age group and sex. Globally, cardiac arrest is a widespread garbage code in mortality registries. The main target code is ischemic heart disease.

The fraction of deaths assigned to garbage codes varies by country. Mexico is considered to have a well-established cause-of-death registry that uses standardized death certification and ICD coding. As such, Mexico has low levels of garbage codes (Naghavi et al., 2010; Stevens et al., 2010). It is estimated that less than 10% of deaths are assigned to garbage codes since the use of the ICD-10 in 1998 (Naghavi et al., 2010). Nonetheless, to improve the registry of ischemic heart disease mortality, we corrected for heart failure death assignment using the Naghavi adjustment factors (Naghavi et al., 2010). See **Appendix III** for further details related with garbage code reassignment to target codes.

[56] Examples of garbage codes are cardiopulmonary arrest, senility, natural or sudden death, among other.

Table 5.3. Mortality by cause associated with ambient $PM_{2.5}$ and ozone chronic exposures [(1)]

Causes of Mortality		ICD-10 Code (1998-2014)	ICD-9 Code (1990-1997)	Ages
Lower Respiratory Infections		J09-J11 J13 J14 J12.1 J12 (except J12.1) J15-J22 J85 P23	487 481 482.2 480.1 466 480.0-480.9 (except 480.1) 482.0- 4829 (except 482.2) 483-486 513 770	<5 years
Trachea, bronchus and lung cancers		C33-C34 D02.1-D02.2 D38.1	162-162.9 231.1* 231.2* 231.8* 235.7*	≥25 years
Ischemic Heart Disease		I20-I25	410-414	≥25 years
Cerebrovascular Disease	Hemorrhagic Stroke	I60-I62	430-431	≥25 years
	Ischemic Stroke	163	432	≥25 years
Chronic Obstructive Pulmonary Disease		J40-J44 J47	490-492.8, 494, 496	≥25 years

Notes: 1) All five causes of mortality are associated with chronic exposures to fine particles. Chronic exposure to ozone is associated only with COPD mortality. 2) The analysis for ischemic heart disease and cerebrovascular stroke is conducted with age-specific risk parameters, by five-year age groups, as explained in section 2.c. Concentration-Response Analysis in Mexico City.

Source: IHME, 2015.

Redistribution of heart failure deaths to ischemic heart disease increased the average number of deaths by 7.5%, with a 6 or 7% increase in 8 *delegaciones*, and 13% in one of the largest and less populated *delegación* (Milpa Alta). These results closely match the erroneously assigned cardiovascular deaths to heart failure in Mexico, estimated to be equal to 8% (Stevens et al., 2010).

c. Concentration-Response Analysis in Mexico City

To characterize the health benefits accrued due to the improvements in air pollution that have occurred in Mexico City since 1990, it is necessary to know how much mortality risk decreases for every unit decrease ($\mu g/m^3$ of $PM_{2.5}$ or ppb of ozone) in ambient air pollution levels. To answer this question, it is essential to consider several issues:

- how to characterize the uncertainty in the risk coefficient near the values of PM and ozone observed in the individual studies;
- how to synthesize risk coefficients across the many cohort studies now available and how to characterize the uncertainty in the resulting pooled risk coefficient;
- what to assume about the shape of the dose-response – i.e., how does it behave at concentrations higher than those seen in the cohort studies themselves;
- for particles, what, if any, additional uncertainty is introduced by the need to estimate risks for particulate matter with elemental (or source) composition different from that seen in the underlying epidemiological studies; and
- any residual concerns (e.g., uncontrolled confounding, effect-modification, biological plausibility) about the plausibility of causal interpretation of the findings.

i. Fine Particles, $PM_{2.5}$

For fine particles, the original publication from the Six Cities Study (Dockery, 1993), showed a central estimate of the risk coefficient for all-cause mortality equal to 1.25% per $\mu g/m^3$, but the 95% confidence interval for the risk coefficient was from 1.08% $\mu g/m^3$ to 1.47% per $\mu g/m^3$. This so-called 'parameter uncertainty' arises simply from the need to draw inferences from a relatively small sample of over 8000 individuals, who at the time had experienced about 1400 deaths (Dockery et al., 1993).

In the original publication from the American Cancer Study (Pope, 1995) for fine particle exposures, the central estimate of the risk coefficient, also for all-cause mortality, was 0.6% per $\mu g/m^3$, with a 95% confidence interval ranging from 0.2% per $\mu g/m^3$ to 1.1% per $\mu g/m^3$ (Pope et al., 2002). The ACS study involved many more participants – almost 500,000, and a correspondingly larger number of deaths, more than 20 thousand (Pope et al., 1995).

With results available from just two studies it is readily apparent that an approach for synthesizing evidence is necessary. Regulatory risk assessments in the US initially adopted the approach of conducting their primary analysis based on the ACS results and using the Six Cities Study results as a basis for sensitivity analysis. A 2002 report of the United States National Academy of Science criticized this approach and recommended that formally elicited expert judgment be considered. In response, two such studies were conducted – one by Cooke et al. in 2007 and another by Roman et al. in 2008 (NAS, 2002; Cooke et al., 2007; Roman et al., 2008). Both studies used rigorous methods to elicit expert judgment. Cooke et al. interviewed six eminent European environmental epidemiologists. Roman et al. interviewed eight well-known environmental epidemiologists and four highly-regarded toxicologists from the US. Both studies reported the individual judgments of each scientist. Cooke et al. also provided pooled estimates, using two approaches (equal weighting and performance weighting). Roman did not report pooled estimates, but recently Krutilla, Good and

Graham provided pooled estimates (using equal weighting) of Roman's results (Krutilla et al., 2015).

The results of these two studies were quite similar. When expert opinions were pooled with equal weight assigned to each expert, they both gave a central effect estimate for all-cause mortality of 1.0% per μg/m^3 (roughly centered between Six Cities and ACS) and 95% upper estimates between 2.4% per μg/m^3 (Roman) and 4.5% per μg/m^3 (Cooke). Both groups of experts gave 5% lower estimates which were quite small – essentially zero (Roman) and 0.02 % per μg/m^3 (Cooke).[57] They give much wider confidence intervals for the true effect than the parameter uncertainty reported by either study, because the experts consider the differences in the results between the studies and also the more complex issues of residual confounding, effect modification, generalization to populations other than those studied, and causality.

With the much larger number of cohort studies now available, attention has turned to meta-analysis as a means for combining results. One highly regarded meta-analysis of $PM_{2.5}$ risk estimates is that of Höek and colleagues (2013) – which was described in the Report of Phase I. As shown in Figure 3.4 of Phase I Report, Höek's pooled estimate of the slope of the concentration response for all-cause mortality from $PM_{2.5}$ is 0.6% per μg/m^3 (95% CI: 0.4% per μg/m^3 to 0.8% per μg/m^3). The confidence interval reported by Höek reflects a combination of within study uncertainty and between study variability in the coefficient estimates.

The Höek meta-analysis does not address the issue of the behaviour of the concentration-response function at concentrations above the values commonly seen in the underlying epidemiological studies. As noted previously, even in the most

[57] Note that when Cooke et al. combined experts' opinions using 'performance weights' – i.e., weights based on their ability to provide accurate and informative probabilistic estimates to a series of calibration questions (for which the analysts know the answers, but the experts do not) – the pooled median effect estimate was 0.6% per μg/m^3 (equal to the ACS result…and equal to the center of the interval later found by Höek et al. using meta-analysis…the lower 5% estimate was 0.06% per μg/m^3 and the upper 95% estimate was 3.8% per μg/m^3).

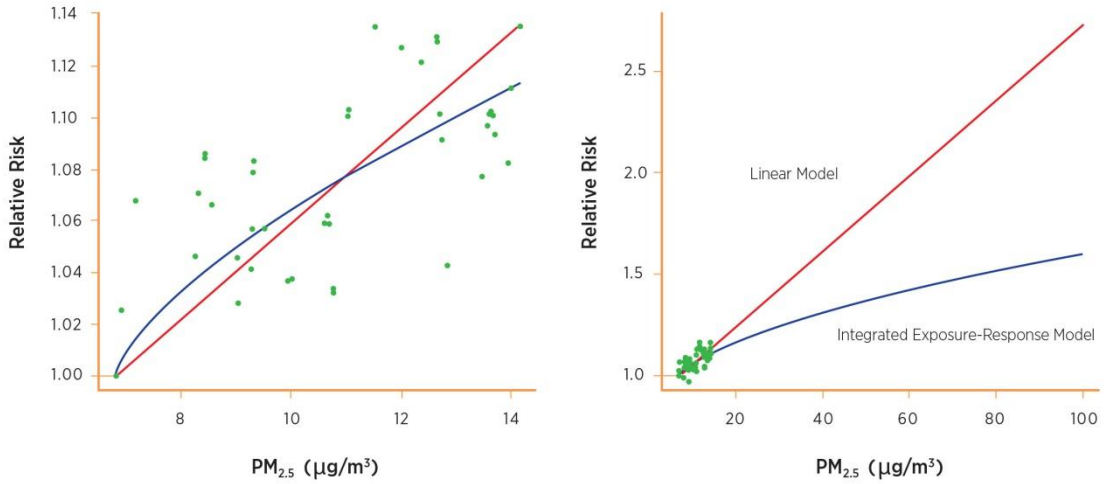
polluted cities included in the Six Cities and American Cancer Society studies the annual average concentrations of $PM_{2.5}$ were below 30 $\mu g/m^3$. Although the annual average concentrations of $PM_{2.5}$ in Mexico City in recent years have been under this value, our estimates of early 1990s annual average $PM_{2.5}$ concentrations indicate that they were not uncommonly in the range of 50 to 60 $\mu g/m^3$.

This same issue has been faced by the scientists responsible for the 2010 and 2013 Global Burden of Disease analyses. In China, for example, current annual averages in certain regions of the country are well above 100 $\mu g/m^3$ and on a population average basis the mean annual average exposure to $PM_{2.5}$ is on the order of 70 $\mu g/m^3$. If the concentration-response functions seen in the cohort studies are extrapolated linearly to these levels of concentration, they produce estimates of relative risk which are implausibly large.

The GBD addressed this issue using a novel approach, known as the “integrated exposure response (IER) function”. They used meta-analysis to pool estimates of risk from eight cohort studies of ambient air pollution with results from studies of mortality risk among people exposed to fine particles through active smoking, passive smoking, and use of dirty fuels (coal, dung, wood) indoors for cooking and heating (Lim et al., 2010; Burnett et al., 2014; Forouzanfar et al., 2015).

Figure 5.6. Hypothetical data fitting two shapes of exposure-response functions: Linear (red line) and integrated exposure-response (blue line).

Left graph fits low and right graph fits high $PM_{2.5}$ concentrations



Source: Shin et al., 2015.

As seen in Figure 5.6 (panels a “low concentration” and b “high concentration”), by using the IER model they were able to avoid the implausibly large estimates of risk that would otherwise result with a linear model at high concentrations – constraining them to be no larger than the relative risks seen in active smokers, in persons exposed to passive smoking, and in people exposed indoors to high levels of smoke from cooking and heating.

Because the concentrations of $PM_{2.5}$ in Mexico City in the early 1990s were well above the levels seen in the Six Cities and ACS studies, and because these early values provide the anchor against which benefits from subsequent improvement of air quality are measured, our $PM_{2.5}$ risk assessment relies on the GBD’s integrated exposure response function (IER).

The IER function estimates the relative risk, RR, as a function of ambient $PM_{2.5}$ concentrations, C, using a four parameter model:

$$RR = 1 + \alpha *(1 – \exp (- \beta * (C – Xo)^{\delta}))$$

The parameter estimates were derived, as mentioned above, from a meta-analysis which included 8 cohort studies of ambient $PM_{2.5}$ and studies of active smoking, passive smoking and exposure to smoke from indoor cooking and heating using dirty fuels (e.g., coal, dung and wood). The GBD analysis of the IER coefficients was conducted separately for four classes of disease in adults – ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, lung cancer – and one for young children - lower respiratory infections. Because of this, 20 parameters have been estimated, four for each of five disease categories.

The GBD research group used a novel approach to characterize the uncertainty in the parameters. Rather than providing a mean and standard deviation for each parameter, or a set of means and a variance-covariance matrix for the full set of parameters, they used a non-parametric approach to produce a matrix of 1000 equally likely set of values of the parameters. The result provides a sound characterization of the uncertainty in estimates of relative risk derived using the IER. It reflects both within study parameter uncertainty and between study variability.

Table 5.4. Uncertainty in slope of the IER at two $PM_{2.5}$ concentrations

Disease	Average Annual $PM_{2.5}$ Concentration			
	20 (µg/m³)		50 (µg/m³)	
	Slope Estimate (% increase in RR per (µg/m³) $PM_{2.5}$)			
	Median	95% CI	Median	95% CI
IHD	0.55	(0.41, 1.89)	0.23	(0.18, 1.05)
STK	2.04	(0.34, 6.09)	1.12	(<0.01, 1.82)
COPD	0.51	(0.21, 0.84)	0.34	(0.17, 0.53)
LC	0.71	(0.18, 1.13)	0.52	(0.18, 0.74)
ALRI	1.06	(0.66, 1.51)	1.14	(0.49, 2.05)
All*	0.69	(0.37, 2.06)	0.43	(0.19, 1.09)

IHD = ischemic heart disease; STK = cerebrovascular stroke; COPD = chronic obstructive pulmonary disease; LC = lung cancer; ALRI = acute lower respiratory infections.

Note: These summary coefficients were derived by weighting the 20 µg/m^3 disease-specific slopes by the fraction of deaths from each disease in 2015 (when levels were ~ 20 µg/m^3). In 2015 there were 9,851 IHD deaths (70.9%); 1,195 STK deaths (8.6%); 2,012 COPD deaths (14.5%); 667 LC deaths (4.8%); and 168 ALRI deaths (1.2%). The summary coefficient appropriate for 50 µg/m^3 was derived by weighting the disease-specific slopes by the fraction of deaths from each of these diseases in 1990 (when levels were ~ 50 µg/m^3). In 1990 there were 5,180 IHD deaths (57.2%); 875 STK deaths (9.7%); 1,671 COPD deaths (18.5%); 561 LC deaths (6.2%); and 763 ALRI deaths (8.4%).

Table 5.4 provides estimates of the marginal slope (% per µg/m^3), the percentage change in relative risk per unit change in $PM_{2.5}$ concentrations, for each of the five diseases considered by the integrated exposure-response function. Because the IER function is curved, the slopes are not constant but depend on the level of $PM_{2.5}$ concentration of interest. For that reason, the table provides estimates of the slopes at two levels of $PM_{2.5}$ concentrations – 20 µg/m^3 and 50 µg/m^3 – to represent the range of values relevant to risk assessment in Mexico City. At each concentration, for each disease, the table provides the median estimate (50%), the 2.5% lower estimate, and the 97.5% upper estimate of the slope. These values were estimated numerically and rely on the non-age-specific IER coefficients.

Both the slopes and the uncertainty in estimates of the slope vary substantially from disease to disease and depend on the $PM_{2.5}$ concentration of interest. At 20 μg/m^3, the slopes (median estimates) vary from 0.51% per μg/m^3 for chronic obstructive pulmonary disease to 2.04% per μg/m^3 for cerebrovascular stroke. At this low level of $PM_{2.5}$, the least uncertain slope (lower respiratory infections) varies by only a factor of 2.3, from 0.66 % to 1.51% per μg/m^3. In contrast, the most uncertain slope (cerebrovascular stroke) varies by a factor of almost 18, from 0.34% to 6.09% per μg/m^3.

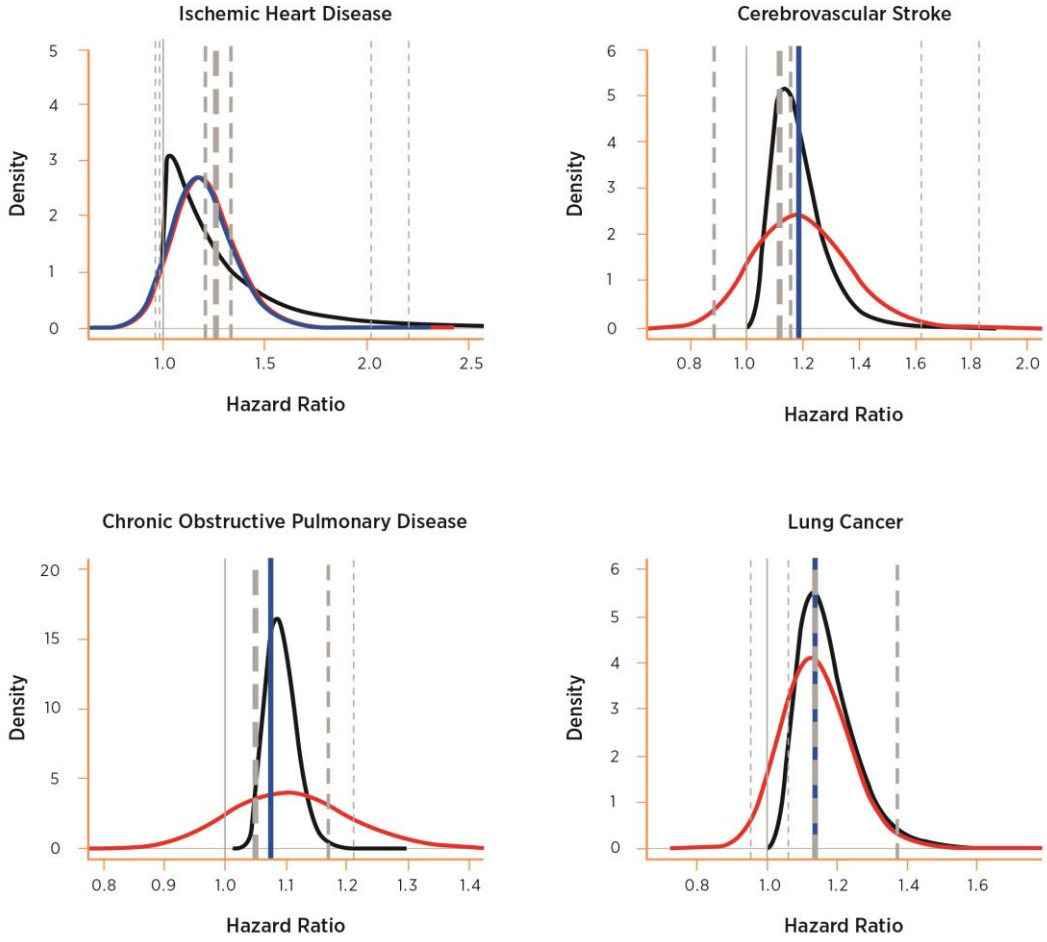
At 50 μg/m^3, the slopes (median estimates) for all diseases except ALRI are smaller and vary from 0.23% per μg/m^3 for ischemic heart disease to 1.12 % per μg/m^3 for cerebrovascular stroke. At this high level of $PM_{2.5}$, the least uncertain slope (chronic obstructive pulmonary disease) varies by a factor of ~ 3, from 0.17% to 0.53% per μg/m^3. The most uncertain slope (cerebrovascular stroke) varies by a factor of more than 200 from < 0.01 % to 1.82% per μg/m^3.

The final row of Table 5.4 provides a single summary slope, obtained by weighting the disease-specific slopes by estimates of the numbers of deaths from each disease in Mexico City during the period of interest. At a concentration of 20 μg/m^3, the summary slope (median estimate) is 0.69 % per μg/m^3 – varying by a factor of more than 5 from 0.37% to 2.06% per μg/m^3. At a higher concentration of 50 μg/m^3, the summary slope (median estimate) is lower – 0.43% per μg/m^3 – varying by a factor of roughly almost 6 from 0.19% to 1.09% per μg/m^3.

These central estimates of the summary slope from the IER are generally consistent with the values from Höek's meta-analysis. Höek's central estimate of 0.6% increase in all-cause mortality per μg/m^3 lies between our central estimates of the IER summary slope of 0.43% per μg/m^3 (at 50 μg/m^3) and 0.69% per μg/m^3 (at 20 μg/m^3).[58]

[58] Note also that our summary slopes apply to a subset of all-cause mortality – i.e., mortality from ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, lung cancer and acute lower respiratory infections.

Figure 5.7. The influence of within study parameter uncertainty and between study parameter variability on the uncertainty in estimates of relative risk of ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, and lung cancer deaths from exposure to fine particles under three approaches for parameter estimation: (i) Gamma distribution with hybrid Bayesian-frequentist approach (black line); (ii) Normal distribution with non-informative priors (red line); and (iii) Normal risk distribution with Q-stat moment estimates (blue line). The vertical dashed lines are reported cohort risk estimates with thickness inversely proportional to the standard error of cohort risk estimates.



Source: Shin et al., 2015.

Figure 5.7 (taken from a recent paper by Shin et al., 2015), gives a sense -- for ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, and lung cancer – of the overall uncertainty in estimates of relative risk and in the contribution of between study variability to this uncertainty. Note that for all four diseases, regardless of the approach chosen for parameter estimation (blue, red or black lines), the dominant source of uncertainty is between-study variability in parameter estimates (gray lines).

ii. Ozone

We follow the approach used by the 2010 and 2013 GBD analyses for estimating mortality risks from population exposure to ozone and rely on Jerrett's analysis of ozone-related mortality in the ACS study (Jerrett, 2009). Jerrett relied on the seasonal average (from 1 April through 30 September) of daily 1-hour maximum ozone values as an exposure metric. Exposures in the ACS study varied from 33.3 ppb to 104 ppb.

Jerrett estimated the relative risk, RR, as a function of ambient ozone concentrations, C, using a two parameter model:

$$RR = \exp (\beta * (C – Xo))$$

in which β is the slope, reflecting the change in risk per unit of exposure, and Xo is the counterfactual, the lowest level of exposure known to lead to increases in risk. Jerrett's central estimate of β was 0.00385 per ppb ozone with a standard deviation of 0.00143 per ppb ozone. The GBD analyses coupled these values of β with the counterfactual value, Xo, treated as uniformly distributed between 33.3 and 41.9 ppb.

One issue not faced by the GBD team was what to assume about chronic obstructive pulmonary disease mortality risk at ozone levels well above those seen in the ACS study. In Mexico City, from 1990 until the early 2000's, seasonal averages of daily 1-hour maxima values were well above 100 ppb. Throughout the early 1990's

seasonal averages of daily 1-hour maxima were commonly in the range of 120 to 180 ppb, occasionally reaching 200 ppb.

To address this issue we relied on the approach followed by Lelieveld et al. (2015) in their recent analysis of mortality attributable to air pollution throughout the world. Lelieveld used a model, first suggested for analysis of mortality from PM by Ostro (2004), in which relative risk is given by:

$$RR = ((C + 1) / (Xo + 1))^{\delta}$$

The two parameters, δ and Xo, are chosen to mimic the behavior of Jerrett's model in the region informed by data from the ACS study and then to constrain relative risks to plausible values in the high concentration region. In this way our approach is not unlike the IER for $PM_{2.5}$.

In summary, our ***base analysis*** for ozone relies on an 'Ostro-like' concentration response function -- with δ parameterized as normal (0.25, 0.075) and Xo parameterized as uniform (33.3 ppbv, 41.9 ppbv). We also conduct a ***sensitivity analysis*** in which we use the Jerrett model – with β parameterized as normal (0.00385 per ppb ozone, 0.00143 per ppb ozone) and Xo unchanged – without any constraint of relative risk estimates at high levels of exposure.

6. Main Findings: Benefits in Mexico City from Air Quality Improvements, 1990 – 2014

By reducing average ambient $PM_{2.5}$ concentrations from 45 µg/m^3 in 1990 to 20 µg/m^3 in 2014 and simultaneously reducing ambient ozone concentrations from over 130 ppb in 1990 to close to 80 ppb in 2014,[59] Mexico City has been able to reduce the number of deaths attributable to air pollution during this 25-year period by more than 22 thousand (95% CI: 17.9 to 28.0 thousand). Roughly 80% of the benefits are due to improvements in $PM_{2.5}$.

Our estimates of the impact of improvements in air quality on mortality are presented in Table 6.1. Central estimates and 95% confidence intervals are given for the reduction in the number of deaths attributable to air pollution over the entire 25-year period and in each of five 5-year periods.

Table 6.1. Premature avoided deaths (Thousands) due to reduction of ambient $PM_{2.5}$ and ozone concentrations in Mexico City – overall (1990 through 2014) and in each 5-year period

	1990-94	1995-99	2000-04	2005-09	2010-14	TOTAL*
Expected Value	0.7	2.3	4.3	6.7	8.6	22.5
95% Confidence Interval	(0.4 - 1.1)	(1.7 - 2.9)	(3.4 - 5.3)	(5.3 - 8.2)	(6.8 - 10.8)	(17.9 - 28.0)

*Totals may not exactly equal the sums of period-specific estimates due to rounding errors.

The greatest reductions in $PM_{2.5}$ and ozone were observed between 1990 and 2004, with an average reduction of 20 µg/m^3 for fine particles and of 45 ppb for ozone. Since 2004 concentrations decreased, but only marginally – i.e., by 5 µg/m^3 for $PM_{2.5}$

[59] Annual concentrations are estimated with averages from fixed-site monitoring stations for 1990 and 2014. For $PM_{2.5}$ annual average estimates were computed from annual averages from four fixed-site monitoring stations in 1990 and from five in 2014 -which were in turn estimated from predicted 24-h concentrations from the GAM developed for the project. For ozone, seasonal (February to July) 1-hour maxima levels were computed from measurements from 6 and 27 monitoring stations, respectively, for 1990 and 2014.

and by 5 ppb for ozone. Starting in 2010 the downward trend for fine particles stopped, and a very slight increase is noticeable which at the moment is of less than 10% (See section **Exposure Assessment for $PM_{2.5}$ and Ozone in Mexico City)**.

In terms of health benefits, the largest changes are projected to occur in recent years. This is because health benefits are related to the differences between actual levels of air pollution and the levels that would have existed if controls had not been implemented – and these differences are the largest in the most recent years. It is important to understand that benefit estimates are influenced by changes in levels of pollution and also by changes in the size and age structure of the population –as these influence the total number of deaths in the population. The absolute size of the population of Mexico City has not increased substantially over the past 25 years, but it has aged and, as a result, the number of deaths has increased from 45 thousand in 1990 to 59 thousand in 2014. In 1990 only 36% of the population was 30 years of age or older but, by 2014, this age-group represented well over half of the population (55%). This growth in the middle aged and elderly segments of the population has increased the size of the "pool" of inhabitants in the age-groups most susceptible to experience mortality due to chronic exposure to air pollution and has contributed to the increase in the number of premature deaths avoided in recent years.

Table 6.2. Contributions of specific causes of death to the expected number of premature deaths avoided by reduction of $PM_{2.5}$ and ozone exposures in Mexico City, 1990 – 2014

Cause of Death	Premature Deaths Avoided* (Thousands)	Percentage*
Ischemic Heart Disease	10.0	44 %
Chronic Obstructive Pulmonary Disease	6.5	29 %
Cerebrovascular Stroke	4.3	19 %
Trachea, Bronchus and Lung Cancers	1.0	4 %
Acute Lower Respiratory Infections	0.7	3 %
Total	**22.5**	**100%**

*Totals may not exactly equal the sums of disease-specific estimates due to rounding errors.

The largest part of the impact is due to reduction of mortality from ischemic heart disease and chronic obstructive pulmonary disease (Table 6.2). Cerebrovascular stroke and lung cancer among adults, and lower respiratory infections among young children also contribute, but together they account for only about 1/4th of the mortality benefits of air pollution improvements.

As expected, we find that the impact of air pollution on mortality is concentrated among the elderly because air pollution primarily affects chronic diseases (Table 6.3). However, it is important to note that, when viewed from the perspective of their impact on longevity, deaths among young children from acute lower respiratory infections become much more important. Each of these deaths involves a loss of life expectancy of many decades. In contrast, deaths among adults due to ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, or lung cancer typically involve losses of life expectancy of perhaps one or two decades.

These estimates are not precise, with a 95% confidence interval that ranges from almost 18 to 28 thousand deaths attributable to air pollution. This uncertainty is not the result of careless analysis, but arises from the fundamental scientific uncertainty about the **true** concentration-response functions for $PM_{2.5}$ and ozone, and from uncertainties in the estimates of population exposures, including the actual measurement at monitoring sites of $PM_{2.5}$ and ozone, the scarcity of monitoring sites in the earlier years of the study period, the spatial interpolation of concentrations from these sites to the *delegaciones*, and the need to estimate concentrations of $PM_{2.5}$ for the period before 2004 when $PM_{2.5}$ was not measured.

Table 6.3. Contributions of impacts of deaths in various age-groups to the expected number of premature deaths avoided by reduction of $PM_{2.5}$ and ozone exposures in Mexico City, 1990 – 2014

Age-Group	Premature Deaths Avoided* (Thousands)	Percentage*
0 – 19	0.7	3 %
20 – 39	0.9	4 %
40 – 59	4.2	19 %
60 – 79	9.4	42 %
≥ 80	7.3	32 %
Total	**22.5**	**100%**

*Totals may not exactly equal the sums of age-specific estimates due to rounding errors.

The Analytica software used to conduct this analysis provides information useful for evaluating the relative importance of each of the variables as individual sources of uncertainty to the overall uncertainty in results. The measure of importance of each variable is the absolute value of the rank correlation (i.e., Spearman correlation) between the values taken by that variable and the resulting estimates of the reduction in the number of deaths attributable to air pollution.

Table 6.4 below summarizes the results of the importance analysis. Results are provided under two assumptions about the nature of the errors in estimates of $PM_{2.5}$ and ozone concentrations – first, that the year-and-*delegación*-specific errors are independent; and second, that they are perfectly correlated. The case of correlated errors is included to reflect the possibility that the sources of uncertainty are not independent, but instead are common across years and *delegación* – so that if one value is underestimated, all values are underestimated … and vice versa.

Table 6.4. Importance of key parameters to overall uncertainty under independent error and correlated error assumptions

Parameter	Importance	
	Independent Errors	Independent Errors
$PM_{2.5}$ Integrated Exposure Response Function	0.60	0.64
$PM_{2.5}$ Population Exposure	0.03	0.07
Ozone Concentration-Response Function	0.27	0.28
Ozone Population Exposure	0.03	0.02

The dominant role of $PM_{2.5}$ is evident. The single largest source of uncertainty in our results is that about the $PM_{2.5}$ concentration-response function (r = 0.60 to 0.64). Uncertainty about the ozone concentration-response function (r = 0.27 to 0.28) is the second largest source of uncertainty in our estimates. Our analysis suggests that uncertainty about PM and ozone concentrations is not an important determinant of results. The absolute value of the Spearman correlation between final results and estimates of pollutant concentrations appears to be small (r = 0.02 to 0.07). The largest of these values only occurs under the somewhat implausible assumption that errors in *alcadia*-and-year-specific estimates of $PM_{2.5}$ are perfectly correlated.

The reasons for this are quite clear. Deaths avoided from $PM_{2.5}$ exposure account for 80% of the total effect and, as has been noted previously, the uncertainty in the slope of the concentration-response function is uncertain – because of (i) the differences in the quantitative effect estimates from the major cohort studies, and (ii) the need to rely on evidence from studies of second-hand smoke, indoor air pollution from cooking and heating, and active smoking, to constrain the IER function at high levels of exposure. Uncertainty in estimates of $PM_{2.5}$ exposures in Mexico City results from – (i) the need to estimate the $PM_{2.5}$ levels for the period 1990 to 2003, when $PM_{2.5}$ was not routinely measured by fixed-site monitoring stations administered by environmental authorities in the city, and (ii) because the network of fixed-site $PM_{2.5}$ monitors that have consistently used particulate matter High Volume Air Samplers (Federal Reference Method) is relatively sparse.

Estimates of the health benefits that have been realized as a result of the improvements in air pollution have been developed using the approach described in the methods sections of this report. The integrated exposure-response functions for fine particulate matter from the 2010 and 2013 Global Burden of Disease analyses have been applied to estimates of the reductions in levels of fine particulate matter that have been realized in each *delegación* since 1990 to estimate the fraction of mortality attributable to $PM_{2.5}$ exposure from five causes of death: ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, trachea, bronchus and lung cancers, in adults (30 years of age and older), and lower respiratory infections in children (under 5 years old). The exposure-response function for ozone from Jerrett's analysis of the ACS study, modified to limit the increases in relative risk seen at levels of ozone above those observed in the ACS study, has been applied to estimates of the reductions in levels of ozone since 1990 to calculate the fraction of chronic obstructive pulmonary disease mortality in adults attributable to ozone exposure.

These estimates of the fractions of disease-specific mortality, which for fine particles were also estimated for each age group, attributable to air pollution exposures have been multiplied by data on the number of deaths in each age group from each cause in each *delegación* to estimate the number of deaths in each age group from each cause in each *delegación* attributable to air pollution. The resulting estimates have been aggregated across age groups, diseases and *delegaciones* to obtain the number of deaths avoided as a result of the improvements in air pollution in each year.

The primary results presented above have come from a ***base case analysis*** which:

i. Uses 1990 as the reference year;

ii. Uses the age-specific coefficients from the GBD's integrated-exposure response function for $PM_{2.5}$;

iii. Relies on a concentration-response function for ozone modified from the GBD to constrain risks at levels of exposure above those observed in the ACS study;

iv. Treats the *delegación*-and-year specific errors in PM and ozone concentrations as independent; and

v. Assigns a probability of 2/3 to the proposition that the influence of PM measurements from the Xalostoc monitoring site should be down-weighted in the derivation of population exposures.

The results of five 'one-way' sensitivity analyses which explore the impact of each of these assumptions on our results are presented below (Table 6.5).

Table 6.5. Impact of key assumptions on estimates of the number of premature deaths avoided by reduction of $PM_{2.5}$ and ozone exposures in Mexico City, 1990 – 2014

	Premature deaths avoided (Thousands)	
Scenario	**Expected Value**	**95% Confidence Interval**
Base Case	**22.5**	**(17.9 - 28.0)**
Sensitivity Analyses		
Ozone CRF: Unconstrained	27.0	(21.1 - 33.4)
IER PM Coefficients: Non-Age-Specific	23.4	(14.5 - 42.0)
Xalostoc PM Measurements: No Down-Weighting	22.8	(18.0 - 27.6)
PM and Ozone Errors: Correlated	22.4	(18.2 – 28.4)
Reference Year: 1993*	17.8	(13.9 - 22.4)

*For the case in which the reference year is 1993, the period of analysis of effects is 1993 – 2014.

The choices made about several of these assumptions have impacts on our central estimates of health benefits. For instance, if we had not constrained the ozone concentration response function, our central effect estimate would have been ~ 20% larger. Likewise, if we had used 1993 instead of 1990 as the reference year, our central estimate would have been ~ 20% lower. In contrast, the decision about whether to down-weight pollution concentration estimates from Xalostoc appears to have had only an infinitesimal impact on our results. These choices also have impacts on our characterization of uncertainty in the results. If we had used the non-age-specific coefficients for PM mortality the uncertainty in our estimates would have been much larger – the 95% CI would have ranged from 14.5 thousand to 42.0 thousand deaths (instead of 17.9 to 28.0 thousand). Similarly, if we had assumed that the *delegación*-and-year specific errors in $PM_{2.5}$ and ozone were perfectly

correlated the uncertainty in our estimates would have been marginally smaller – the 95% CI would have ranged from 18.0 to 27.6 thousand deaths (instead of 17.9 to 28.0 thousand).

Having said this, it is clear that none of these choices would have dramatically changed the essential story. That is, reductions in $PM_{2.5}$ and ozone over the past twenty-five years have led to substantial improvements in health and reductions in mortality, saving on the order of 20 thousand lives over the period.

It is important to recognize that, in fact, lives cannot be saved by air pollution controls or any other public policy intervention. They can merely be extended. This report uses the measure of 'premature deaths avoided' as a *proxy* for the increases in life expectancy achieved by improvements of air pollution. Reducing air pollution levels leads to increases in life expectancy. Future analyses within this project will provide estimates of the benefits of air pollution improvements, expressed in terms of their impact on life expectancy as well as their impact on the number of deaths attributable to air pollution.

Finally, this analysis is based on the assumption that, without the rigorous air pollution controls put in place since 1990, air quality would have remained as it was then. In reality with the growth of the population, the size increase of the pool of middle aged and elderly segments of the population which are most susceptible to experience mortality due to chronic exposure to air pollution, plus growth in economic activity that has occurred in Mexico City and the surrounding urbanized area, it is virtually certain that without substantial regulation, air pollution levels would have increased. Thus, our estimates of the mortality benefits of these controls almost certainly underestimate the true benefits of government regulations and programs.

III. Concluding Remarks for Phase I and II

The essential story of this risk assessment conducted for Mexico City from 1990 to 2014 is that reductions in $PM_{2.5}$ and ozone over the past twenty-five years have led to substantial improvements in health and reductions in mortality – saving on the order of 20 thousand lives over the period (Table III.1.). More specifically, as measured in monitoring sties, by reducing citywide average ambient $PM_{2.5}$ concentrations from 45 $\mu g/m^3$ in 1990 to 20 $\mu g/m^3$ in 2014 and simultaneously reducing ambient ozone concentrations from over 130 ppb in 1990 to close to 80 ppb in 2014,[60] Mexico City has been able to reduce the number of deaths attributable to fine particles and ozone during this 25-year period by 22.5 thousand (95% CI: 17.9 to 28.0 thousand). Roughly 18.0 thousand of these avoided deaths are due to improvements in $PM_{2.5}$ (95% CI: 14.0 to 23.5 thousand), and over 4.0 thousand to ozone (95% CI: 2.7 to 5.6 thousand).

The ***base case analysis*** to estimate the benefits associated with air quality improvements in Mexico City has the following characteristics:

(i) Uses 1990 as the reference year;

(ii) Applies the concentration-response functions from the GBD, for $PM_{2.5}$ it uses the age-specific coefficients from the integrated-exposure response (IER) function, and for ozone relies on a concentration-response function modified from the GBD to constrain risks at levels of exposure above those observed in the ACS study;

(iii) Treats the *delegación*-and-year specific errors in PM and ozone concentrations as independent; and

(iv) Assigns a probability of 2/3 to the proposition that the influence of PM estimated concentrations from the Xalostoc monitoring site should be down-weighted in the derivation of population exposures for the *delegaciones*.

[60] Annual concentrations are estimated with averages from fixed-site monitoring stations for 1990 and 2014. For $PM_{2.5}$ annual average estimates were derived from the GAM developed for the project based on data from four fixed-site monitoring stations in 1990, and five in 2014. For ozone, seasonal (February to July) 1-hour maximum average concentrations were computed from measurements from 6 and 27 fixed-site monitoring stations, respectively, for 1990 and 2014.

Table III.1. Expected number of premature deaths avoided by reductions of $PM_{2.5}$ and ozone exposures, contributions of specific causes of death and sensitivity analysis for Mexico City, 1990 – 2014

	Premature deaths avoided (Thousands)*	95% Confidence Interval*
Base Case	**22.5**	**(17.9 - 28.0)**
Cause of death		**Percentage***
Ischemic Heart Disease	10.0	44 %
Chronic Obstructive Pulmonary Disease	6.5	29 %
Cerebrovascular Stroke	4.3	19 %
Trachea, Bronchus and Lung Cancers	1.0	4 %
Acute Lower Respiratory Infections	0.7	3 %
Sensitivity Analyses: impact of key assumptions		**95% CI**
Ozone CRF: Unconstrained	27.0	(21.1 - 33.4)
IER PM Coefficients: Non-Age-Specific	23.4	(14.5 - 42.0)
Xalostoc PM Measurements: No Down-Weighting	22.8	(18.0 - 27.6)
PM and Ozone Errors: Correlated	22.4	(18.2 – 28.4)
Reference Year: 1993*	17.8	(13.9 - 22.4)

*For the case in which the reference year is 1993, the period of analysis of effects is 1993 – 2014.

The numbers of avoided deaths are not precise, as our 95% confidence interval ranges from almost 18 to 28 thousand avoided deaths. This uncertainty arises from the fundamental scientific uncertainty about the **true** concentration-response functions for $PM_{2.5}$ and ozone, and from uncertainties in the estimates of population exposures. The evaluation of the relative importance of each of the variables as individual sources of uncertainty to the overall uncertainty in results shows that the single largest source of uncertainty is the one derived from the $PM_{2.5}$ concentration-response function. The ozone concentration-response function is the second largest source of uncertainty. Uncertainty in the estimates of population exposures, that stem from the actual measurements at monitoring sites, the scarcity of monitoring sites in the earlier years of the study period, the spatial extrapolation of concentrations from these sites to the *delegaciones*, and the need to estimate concentrations of $PM_{2.5}$ for the period before 2004 when $PM_{2.5}$ was not measured, were not as important determinants of uncertainty in our results.

This analysis includes the causes of death associated with chronic exposures to ambient fine particles and ozone that were analysed on the GBD studies. These

causes of death were selected because the scientific evidence points to a causal relationship between chronic exposures to fine particles, with more limited evidence for chronic exposures to ozone, and mortality due to specific diseases. The analysis was conducted separately for four classes of disease in adults, ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, lung cancer, and one for young children, lower respiratory infections.

Our results show that the vast majority of the impact is due to reduction of mortality from ischemic heart disease (44%), followed by chronic obstructive pulmonary disease (almost 30%), and then by cerebrovascular stroke (close to 20%) (Table III.1). The contribution of the remaining two causes of death, lung cancer among adults, and acute lower respiratory infections among young children, account together for less than 10% of the mortality benefits of air pollution improvements.

As would be expected, we find that the benefits from air pollution reductions are concentrated among the elderly (>70% of total avoided deaths in the age-group of over 60 years of age), because air pollution primarily affects chronic diseases. However, it is important to note that, when viewed from the perspective of their impact on longevity, premature deaths avoided among young children from acute lower respiratory infections (about 3% of total avoided deaths) become much more important. Each such death would involve a loss of life expectancy of many decades. In contrast, avoided deaths among adults due to ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, or lung cancer would typically involve losses of life expectancy of perhaps one or two decades.

The results that were discussed come from a ***base case analysis*** which derives from choices that impact our central estimates of health benefits. We conducted a sensitivity analysis to explore the influence of such choices on our results (Table III.1.). Our central effect estimate would have been larger (~ 20%) if we had not constrained the ozone concentration response function to reduce the risk estimate at the elevated concentrations found in Mexico City in the early 90s. Likewise, our central estimate would have been lower (~ 20%) if we had used as the reference

year 1993 instead of 1990, because between 1990 and 1993 we observe high peaks in pollutant concentrations. In contrast, the decision about whether to down-weight $PM_{2.5}$ concentrations from Xalostoc, given that the site is located in a "hot spot" that captures local emission sources unlikely to represent exposures of populations that are not in the immediate vicinity of the station, appears to have had only an infinitesimal impact on our results.

Our choices also impact our characterization of uncertainty in the results. The stronger effect is seen by changing the age-specific IER coefficients to the non-age-specific coefficients for PM mortality. The uncertainty in our estimates would have been much larger, with a 95% CI ranging from 14.5 thousand to 42.0 thousand avoided deaths (instead of 17.9 to 28.0 thousand). The opposite effect results if assuming a perfect correlation between *delegación*-and-year specific errors in PM and ozone. Marginally smaller uncertainty in our estimates would have resulted, the 95% CI would have ranged from 18.0 to 27.6 thousand avoided deaths (instead of 17.9 to 28.0 thousand).

Public policies that aim at improving air quality benefit public health because premature mortality risk is reduced in large populations. However, the individuals that are affected are not known. Thus, our risk assessment uses 'premature deaths avoided' as the metric to evaluate the health benefits that result from air pollution improvements. In reality, this metric is a *proxy* for "life expectancy increases", since reductions in air pollution levels lead to increases in life expectancy. In a future stage of this project life expectancy changes will be a metric to quantify the benefits of air quality improvements.

The rigorous air pollution controls put in place in Mexico City have allowed air quality improvements. It is clear that without the implementation of public policy strategies, air pollution levels would have increased as a result of the population and vehicular fleet growth, augmented economic activity, and the expansion of the urban sprawl that have occurred in Mexico City and the surrounding urbanized area.

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Methodological Appendices

Appendix I. Generalized Additive Model (GAM) to predict $PM_{2.5}$ daily concentrations (In Spanish)

Appendix II. Sufficiency criteria and data management for air pollutants and meteorological variables (In Spanish)

Appendix III. Appendix III. Causes of death. Data management and quality control procedures (In Spanish)

Appendix I. Generalized Additive Model (GAM) to predict $PM_{2.5}$ daily concentrations

Apéndice I. Modelo aditivo generalizado (GAM) desarrollado para predecir las concentraciones diarias de $PM_{2.5}$

Modelo predictivo – Modelo aditivo generalizado (GAM)

$$PM_{2.5_{observada}} = \alpha + \beta_1 PM_{10_{observada}} + \beta_{2i}\,\text{estación} + \beta_{6i}\,\text{mes} + s(\text{año}) + s(\text{ws}) + (\text{temp}) + s(\text{rh}) + \varepsilon_{ij}$$

donde:
α = intercepto de la regresión
β = coeficientes de la regresión
estación (β_{2i}, i=2-5), Pedregal es la categoría de referencia
mes (β_{6i}, i=6-16), agosto es la categoría de referencia
s(x) = función "spline" suavizada para año, velocidad del viento, temperatura y humedad relativa

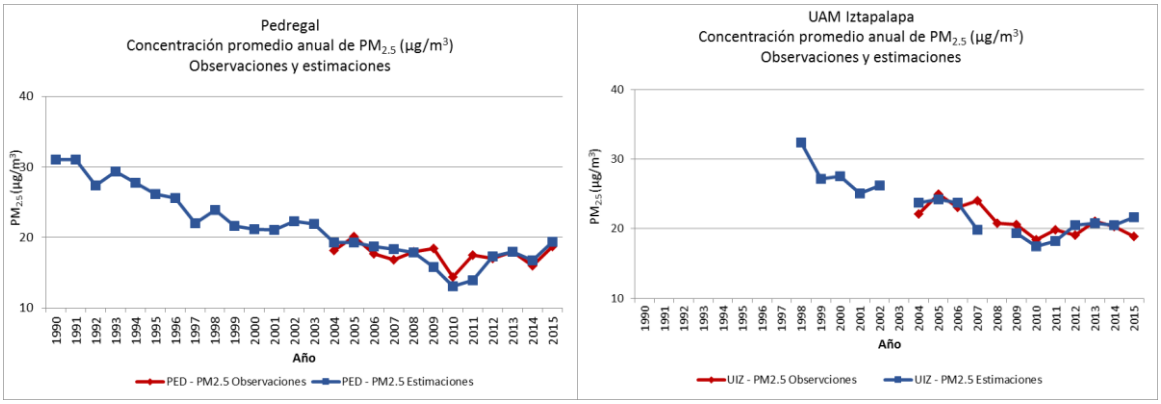
Descripción de las variables

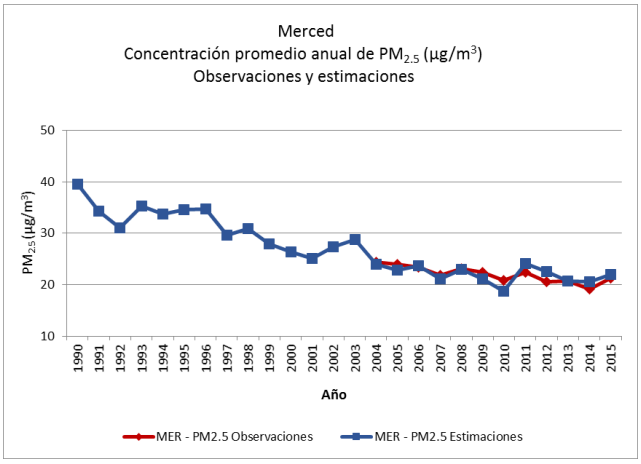
- $PM_{2.5}$ y PM_{10} – Datos de la red manual de monitoreo atmosférico (SEDEMA - REDMA)
- Temperatura (temp), velocidad del viento (wsp), humedad relativa (rh) – Datos de la red meteorológica (SEDEMA - REDMET). Excepción: en 2007 se imputaron datos de la red de monitoreo de la UNAM a falta de suficiencia de datos de la REDMET.
- Mes y estación - Variables categóricas. Se seleccionaron como categorías de referencia a la estación y al mes con los promedios anuales de menor valor, Pedregal y agosto, respectivamente.
    - Mes: agosto = 1, sep = 2, oct = 3…, julio = 12
    - Estación: PED = 1, UIZ = 2, TLA = 3, MER = 4, XAL = 5

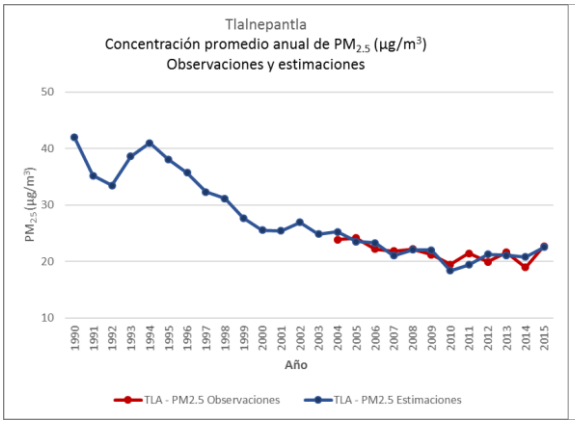
Cuadro I. Concentración promedio anual de PM2.5 para las variables categóricas, estación y mes, 2004-2015

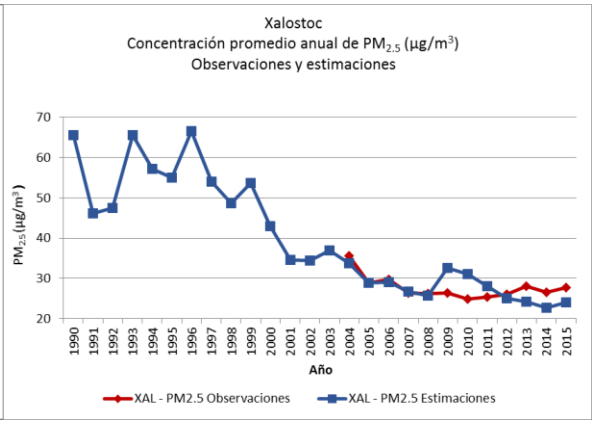
Estación de monitoreo	Concentración promedio anual $PM_{2.5}$ ($\mu g/m^3$)	Codificación
PED	17.5	Referencia
UIZ	21.1	2
TLA	21.6	3
MER	22.0	4
XAL	27.6	5
Mes	**Concentración promedio anual $PM_{2.5}$ ($\mu g/m^3$)**	**Codificación**
Agosto	15.0	Referencia
Septiembre	16.0	2
Octubre	17.2	3
Noviembre	24.6	4
Diciembre	33.7	5
Enero	27.1	6
Febrero	23.2	7
Marzo	20.5	8
Abril	25.3	9
Mayo	26.5	10
Junio	20.6	11
Julio	16.5	12

Concentración promedio anual de $PM_{2.5}$ para las cinco estaciones de monitoreo. Datos observados con monitoreo utilizando método manual (2004-2015) y estimados a través del GAM (1990-2015)









Resultados del modelo predictivo GAM

Parametric coefficients:				
	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	5.307595	0.457585	11.599	< 2e-16
pm10	0.291810	0.006057	48.173	< 2e-16
station_reco2	0.144804	0.338236	0.428	0.66860
station_reco3	-0.466448	0.330962	-1.409	0.15883
station_reco4	-0.465159	0.335767	-1.385	0.16605
station_reco5	-1.946478	0.408377	-4.766	1.97e-06
mes_reco2	1.385585	0.483855	2.864	0.00422
mes_reco3	0.374621	0.494266	0.758	0.44855
mes_reco4	1.788563	0.583552	3.065	0.00220
mes_reco5	4.090909	0.660399	6.195	6.65e-10
mes_reco6	1.152854	0.659464	1.748	0.08054
mes_reco7	1.023974	0.619021	1.654	0.09820
mes_reco8	1.264956	0.576154	2.196	0.02820
mes_reco9	4.057287	0.557929	7.272	4.50e-13
mes_reco10	5.739892	0.525899	10.914	< 2e-16
mes_reco11	2.481734	0.499185	4.972	7.02e-07
mes_reco12	0.180569	0.476466	0.379	0.70473

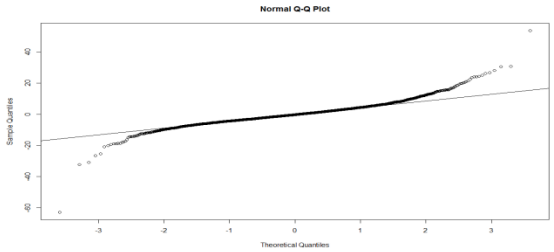
Approximate significance of smooth terms:				
	edf	Ref.df	F	p-value
s(year)	8.018	8.747	65.81	<2e-16
s(hr)	4.828	5.951	40.45	<2e-16
s(temp)	5.758	6.981	15.75	<2e-16
s(wsp)	3.987	5.007	128.68	<2e-16

R^2	Akaike Information Criterion	Residual Maximum Likelihood (REML)
0.735	18979.51	9495

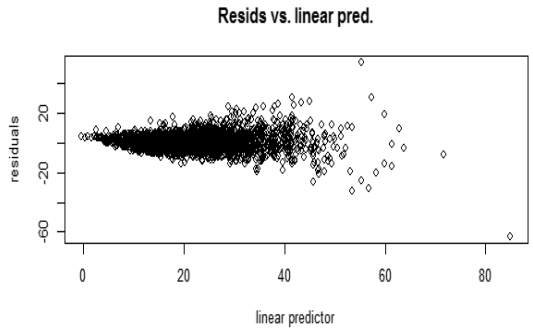
Coeficiente de Determinación (R^2) - porcentaje de variabilidad que explica el modelo
Criterio de Información de Akaike (AIC) - indica el beneficio de tener más información en el modelo en términos de la reducción de la varianza, con el costo de ese beneficio en términos de la pérdida de grados de libertad (un mejor ajuste del modelo a menor valor de AIC)
REML - máxima verosimilitud restringida, estima los parámetros de covarianza

Diagnóstico del modelo mediante el análisis de residuos.

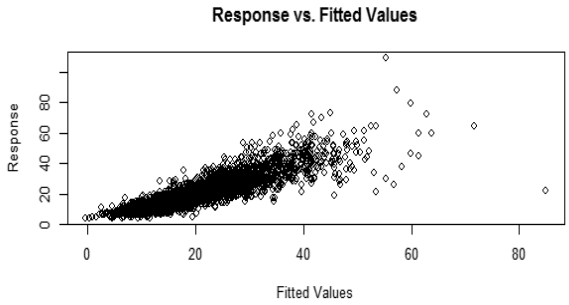
Linealidad



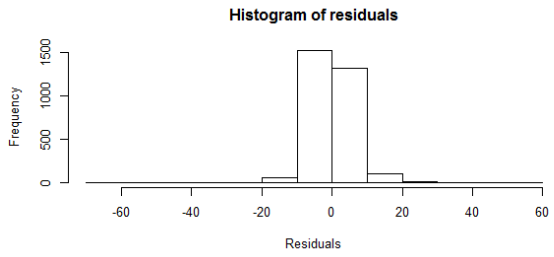
Homocedasticidad



Relación entre valores observados vs. estimados

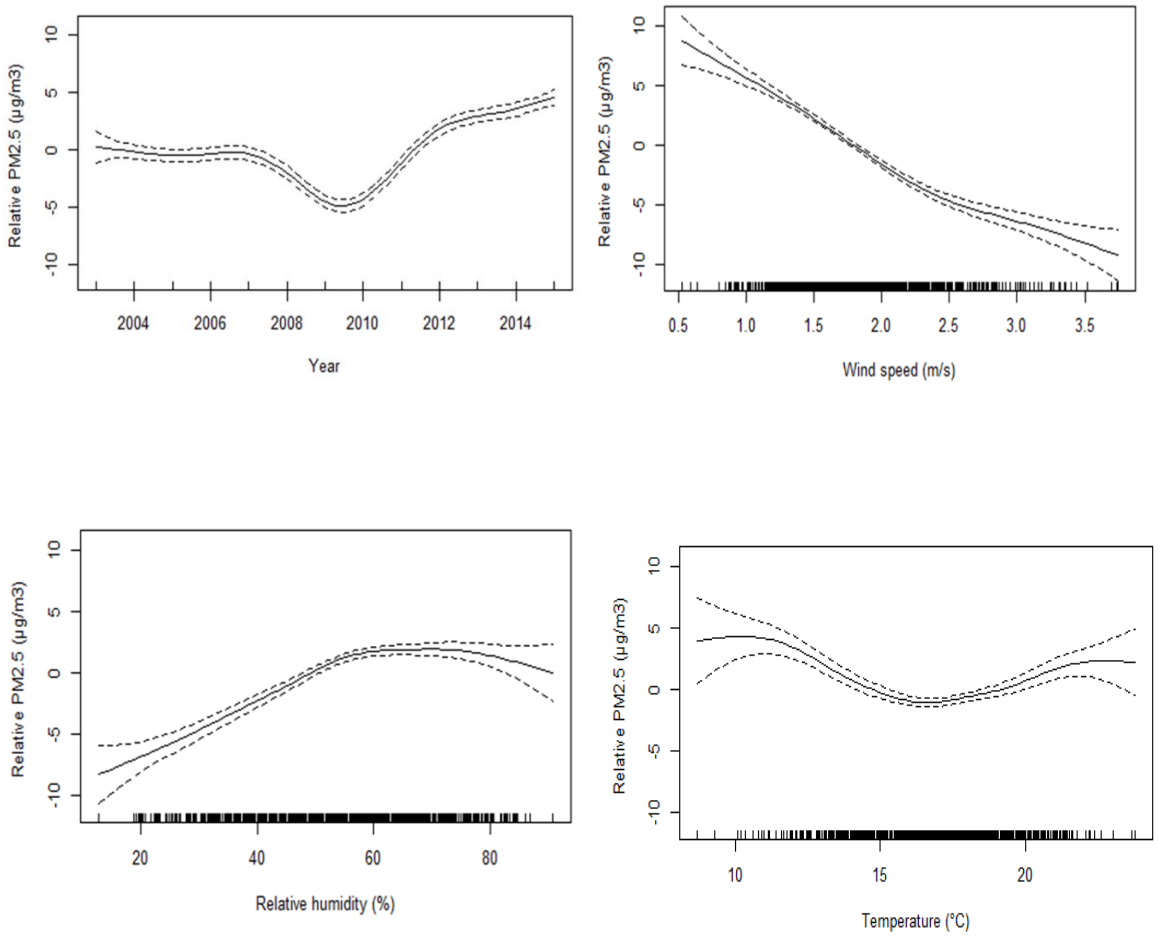


Histograma de frecuencias de los residuos



Resultados del modelo predictivo GAM

Tendencias anuales ajustadas por variables meteorológicas, estación de monitoreo, año (gráfica superior izquierda). Relaciones no lineales entre concentraciones de $PM_{2.5}$ y velocidad del viento (gráfica superior derecha), humedad relativa (gráfica inferior izquierda) y temperatura (gráfica inferior derecha).



Appendix II. Sufficiency criteria and data management for air pollutants and meteorological variables

Apéndice II. Criterios de suficiencia y manejo de datos para contaminantes de aire y variables meteorológicas

Criterio de suficiencia: al menos 75% de los datos válidos

$PM_{2.5}$ y PM_{10}
Métrica de exposición: promedio anual de las concentraciones de 24 horas

Mediciones integradas de 24 horas cada 6 días
Observaciones por año, $n_{año}$ = 61-62 días
Observaciones por trimestre, n_{tri}= 15 días

- Trimestre válido: se cuenta con al menos 75% de las observaciones diarias en el trimestre ($n_{día}$ > 11)
- Año válido: se cuenta con al menos 2 trimestres válidos

Variables meteorológicas, NO_2 y SO_2 [61]
Mediciones horarias

- Día válido: se cuenta con al menos el 75% de los valores de las concentraciones horarias de cada día (n_{hora} > 18)

Se seleccionaron los datos de ozono, NO_2, SO_2, temperatura, humedad relativa y velocidad del viento, de los días en los que hubiera mediciones de PM. De esta forma se construyó una base con datos cada 6 días. Para el resto del procesamiento se utilizaron los siguientes criterios.

Observaciones por año, $n_{año}$ = 61-62 días
Observaciones por trimestre, n_{tri}= 15 días

- Trimestre válido: se cuenta con al menos 75% de las observaciones diarias en el trimestre ($n_{día}$ > 11)
- Año válido: se cuenta con 2 trimestres válidos

Ozono

Métrica de exposición: promedio estacional (febrero a julio) de las concentraciones horarias máximas

Mediciones horarias
Observaciones por año, $n_{año}$ = 365 días

- Hora válida: se cuenta con al menos 75% de los registros minutarios en 1 hora (este procesamiento es a cargo de SEDEMA, CDMX)
- Día válido: se cuenta con al menos el 75% de los valores de las concentraciones horarias de cada día (n_{hora} < 18)
- Trimestre válido: se cuenta con al menos 75% de las observaciones diarias en el trimestre ($n_{día}$ > 68)
- Año válido: 2 trimestres válidos (para nuestra métrica de exposición deben ser válidos el 1er y 2° trimestre)

[61] Se usaron como variables predictivas y se valoró su inclusión en el modelo GAM. Sin embargo, tenían muchos datos faltantes y se reducía el número de predicciones en alrededor de 7 años. Se optó por un modelo sin estos dos gases.

- Definición de trimestres:
  - 1er trimestre: febrero, marzo y abril
  - 2º trimestre: mayo, junio y julio
  - 3er trimestre: agosto, septiembre y octubre
  - 4º trimestre: noviembre, diciembre y enero

Estimación de la métrica de exposición

Se utiliza la base de datos (que proporciona SEDEMA) con las concentraciones horarias

Para cada año y para cada estación de monitoreo se sigue el siguiente procesamiento:

- Se determinan los valores máximos de 1 hora de cada día
- Se promedian los valores máximos de 1 hora de cada día en cada trimestre
- Se promedian los promedios de los valores máximos de 1 hora de cada día del 1er trimestre (febrero-abril) y del 2° trimestre (mayo-julio)

Appendix III. Causes of death. Data management and quality control procedures

Apéndice III. Causas de mortalidad. Manejo y control de calidad de los datos

Fuentes de información

La Secretaría de Salud del gobierno de la Ciudad de México (SEDESA) proporcionó los datos de mortalidad de la Ciudad de México para el periodo 1990 - 2014 que se incluyen en este reporte. SEDESA, a su vez, obtiene estos datos de los registros anuales del Instituto Nacional de Estadística, Geografía e Informática (INEGI), en donde se incluye el número de decesos ocurridos por día para cada año. Para el periodo 1990 – 1997 se utilizan los tabuladores de la Clasificación Internacional de las Enfermedades (CIE) en su novena versión (CIE- 9) y a partir de 1998 se utilizan aquellos de la décima versión (CIE-10).

Causas de muerte y grupos etarios analizados

Las causas de muerte y grupos de edad incluidos en nuestros análisis corresponden a los reportadas en el cuadro I.

Cuadro I. Causas de mortalidad asociadas con la exposición crónica a partículas finas y ozono

Causas de muerte		Códigos CIE9 (1990-1997)	Códigos CIE10 (1998-2014)	Grupo Etario (años)
Enfermedades respiratorias inferiores agudas		487, 481, 482.2, 480.1, 466, 480.0-480.9, 482.0- 4829, 483-486, 513, 770	J09-J11, J13, J14 J12.1, J12, J15-J22, J85, P23	<5
Cáncer de tráquea, bronquios y pulmón		162-162.9 231.1, 231.2, 231.8, 235.7	C33-C34, D02.1-D02.2, D38.1	≥25
Enfermedad isquémica del corazón		410-414	I20-I25	≥25-30 >30-35 >35-40 >40-45 >45-50 >50-55 >55-60 >60-65 >65-70 >70-75 >75-80 >80
Enfermedad cerebrovascular	Infarto hemorrágico	430-431	I60-I62	
	Infarto isquémico	432	163	
Enfermedad pulmonar obstructiva crónica		490-492.8, 494, 496	J40-J44 J47	≥25

Notas: 1) Las cinco causas de mortalidad se asocian con la exposición crónica a partículas finas. La exposición crónica a ozono se asocia solamente con mortalidad por EPOC. 2) El análisis para enfermedad isquémica del corazón y enfermedad cerebrovascular se realiza con parámetros de riesgo para cada grupo etario quinquenal.

Fuente: IHME, 2015. Mapping revisions and variants of the ICD. Web Table 3 - List of International Classification of Diseases codes mapped to the Global Burden of Disease cause list. http://www.healthdata.org/sites/default/files/files/data_for_download/2012/IHME_GBD2010_CauseListandICD.pdf.

Manejo de datos

Paso 1. Selección de variables relevantes para cálculo de total de muertes anuales

Los registros anuales de INEGI se depuraron para asegurar que las muertes en la base de datos cumplieran con las características que requiere este proyecto: delegaciones de la Ciudad de México, causas de mortalidad (cuadro I), estratificación por género y grupo de edad, y año del deceso [3-4].

- **Entidad de residencia:** entidad federativa en donde la persona tiene su domicilio particular, principal o permanente.
- **Municipio de residencia:** municipio o delegación (en el caso de la Ciudad de México) en donde la persona tiene su domicilio particular, principal o permanente.
- **Causa de la defunción (lista detallada):** causa básica de la defunción de acuerdo con la Clasificación Internacional de Enfermedades, su información identifica la enfermedad o lesión que inició la cadena de acontecimientos patológicos que condujeron directamente a la muerte, o las circunstancias del accidente o violencia que produjo la lesión fatal.
- **Sexo:** Condición biológica que distingue a las personas en hombres y mujeres.
- **Edad:** Tiempo transcurrido entre la fecha de nacimiento de la persona y la del momento en que ocurre o se registra el hecho vital.
- **Año de ocurrencia:** Año en que ocurrió el hecho vital.

Paso 2. Limpieza de la información

Entidad y municipio de residencia. Se verificó que las muertes fueran de individuos que hubieran residido en la Ciudad de México (Clave de Entidad Federativa = 09). De forma similar se verificó que las claves para la delegación de residencia correspondieran con que establece el INEGI (cuadro II).

En los casos en los que no se especificó la delegación de residencia esta variable se codificó como no especificada (NE) en nuestras bases de datos y dichas observaciones sólo se incluyeron en la estimación de muertes totales ocurridas en la Ciudad de México.

Cuadro II. Claves y nombres de las delegaciones de la Ciudad de México

Clave	Nombre
002	Azcapotzalco
003	Coyoacán
004	Cuajimalpa de Morelos
005	Gustavo A. Madero
006	Iztacalco
007	Iztapalapa
008	La Magdalena Contreras
009	Milpa Alta
010	Álvaro Obregón
011	Tláhuac
012	Tlalpan
013	Xochimilco
014	Benito Juárez
015	Cuauhtémoc
016	Miguel Hidalgo
017	Venustiano Carranza

Causa de defunción. Se confirmó que cada grupo de causa de muerte incluyera solamente aquellos códigos especificados en el cuadro I. En caso contrario se descartaron de las bases de datos.
También se confirmó que las muertes se hubieran codificado con base en la CIE-9 o en la CIE-10 de 1990 a 1997 para la primera, y, a partir de 1998, para la segunda. Cuando el registro de la fecha de defunción no coincidió con el período de aplicación de la CIE que correspondía, éste se re-codificó de acuerdo con el código equivalente de la versión de la CIE correcta.

Sexo. Se confirmó que no hubiera errores en el rango de valores establecidos por INEGI: hombre (1), mujer (2) y no especificado (9). Para las enfermedades isquémicas únicamente cinco individuos fueron codificados con sexo "no especificado" durante el periodo 1990-2014.
El sexo es relevante en nuestro análisis para el grupo de Enfermedades Isquémicas del Corazón ya que las muertes anuales por estas causas se ajustaron por sexo para corregir errores en la clasificación del diagnóstico de muerte.

Edad. Se trata de una variable importante para nuestro análisis debido a que los resultados se presentan por grupos etarios por rango quinquenal.

Se verificó que la edad de los individuos en cada grupo de causas de muerte se encontrara dentro de los rangos que se presentan en el cuadro I. Para los registros que indicaban edad "no especificada" se intentó estimar su edad a partir de su fecha de nacimiento; se descartaron los registros para los que no fue posible estimar la edad. El cuadro III muestra el total de registros con edad "no especificada" que se registraron.

Cuadro II. Número de muertes con edad no especificada por causa en la Ciudad de México, 1990-2014

Causas de muerte	Muertes (#)
Enfermedades respiratorias inferiores agudas	0/8,455
Cáncer de tráquea, bronquios y pulmón	1/15,347
Enfermedad isquémica del corazón	25/167,075
Enfermedad cerebrovascular	15/ 51,390
Enfermedad pulmonar obstructiva crónica	19/45,749

Para el grupo de menores de cinco años sólo se incluyeron las muertes post-neonatales (mayores a 28 días) por estar más influenciada por el ambiente externo del infante. La evidencia científica señala que las muertes durante el primer mes de edad se deben principalmente a causas intrínsecas como anomalías congénitas y que, en cambio, es poco probable que estén influenciadas por la exposición a contaminantes del aire [5-6].

Año de ocurrencia. Se corroboró que las muertes se encontraran dentro del periodo 1990-2014, de forma contraria se excluyeron de la base de datos. Se contó con información para todas las alcadías y años. La excepción fue la delegación Álvaro Obregón para la que no se obtuvieron datos de mortalidad para ninguna de las causas en 1992.

Se hizo el ajuste de los fallecimientos que ocurren durante noviembre y diciembre y que frecuentemente se registran hasta los primeros días del año subsecuente. Así, se contabilizaron de acuerdo con el año de ocurrencia y no de registro.

Paso 3. Corrección por mala clasificación en el diagnóstico de muerte

Actualmente se reconoce que la calidad de la información acerca de las causas de mortalidad que recogen los registros de mortalidad es diferente para cada país y puede variar en el tiempo. Esta situación es resultado de factores que incluyen los cambios en la codificación de la Clasificación Internacional de Enfermedades y muertes atribuidas a causas que no pueden o no deben ser consideradas como tales. Estas causas se conocen como "códigos basura" (CB) y deben reasignarse a ciertos "códigos objetivo" (CO), que constituyen las causas probables de los decesos. La redistribución de CB a CO se ha determinado con base en prácticas de certificación, conocimiento experto de la etiología o fisiopatología de las enfermedades o mediante la combinación de ambas y del desarrollo de algoritmos. Las estimaciones de la Carga Global de la Enfermedad del *Institute for Health Metrics and Evaluation* (IHME) y de análisis similares de la Organización Mundial de la Salud han empleado diversos enfoques para dicha tarea, incluyendo la redistribución proporcional de CB a CO para diferentes grupos etarios por sexo [7-8].

A nivel mundial los CB correspondientes a insuficiencia cardiaca[62] (códigos CIE-9: 428 y CIE-10: I50) son altamente prevalentes en los datos de mortalidad, y se considera que su principal CO son las enfermedades isquémicas del corazón [9]. Si bien México se ha reconocido como unos de los países latinoamericanos con buenos sistemas de registro de muertes y relativamente baja proporción de CB, la práctica de reasignar CB a CO es una tarea necesaria [8].

Así, para mejorar el registro de los datos de mortalidad por enfermedades isquémicas del corazón se realizó la corrección de CB utilizando las proporciones de Naghavi et al. (2010) [8]. El cuadro IV muestra los CB y las proporciones que se emplearon para la reasignación de muertes mal clasificadas a sus potenciales CO.

El método que se utilizó consta de dos pasos. Primero, se agruparon los CB por sexo y grupo etario en las bases de datos originales para cada una de las delegaciones de la Ciudad de México para cada año. Posteriormente, se llevó a cabo la redistribución proporcional de los CB a los CO, sumándose las estimaciones corregidas para cada sexo para contar con el total anual de casos de muertes por enfermedades isquémicas del corazón para cada delegación.

Finalmente, para el periodo 1990-1997 durante el cual estuvo vigente en México la codificación de enfermedades con base en la CIE-9, se consideraron las proporciones de redistribución correspondientes a las de un país en desarrollo de acuerdo con lo señalado

[62] La insuficiencia cardíaca corresponde a la etapa final de muchos procesos patológicos tanto cardíacos y como no cardíacos, que van desde cardiopatía isquémica y miocardiopatías hasta enfermedades respiratorias y anemia. Como tal la insuficiencia cardíaca no corresponde como tal a una causa subyacente de muerte de acuerdo con la Organización Mundial de la Salud, sino a causa intermedia de la muerte con una amplia gama de posibles causas subyacentes de la muerte.

por Naghavi et al. (2010), debido a que una vez que entró en vigor la codificación con base en CIE-10 a partir de 1998, hubo mejorías sustantivas en la codificación de enfermedades, lo cual representó una disminución en la proporción de CB utilizados.

Cuadro IV. Proporción de *códigos basura* por sexo y grupo etarios para su reasignación a enfermedades isquémicas del corazón

Códigos basura (CB)		**Códigos objetivo (CO)**		**Versión CIE**	**Proporciones**			
					Hombres (grupos etarios)		**Mujeres (grupos etarios)**	
				Periodo	**25-49**	**>50**	**25-49**	**>50**
Insuficiencia cardíaca **Insuficiencia cardíaca congestiva** **Insuficiencia ventricular izquierda** **Insuficiencia cardíaca sin especificar**	428 428.0 428.1 428.9	Enfermedades Isquémicas Complicaciones siguientes a infarto agudo de miocardio Otras enfermedades isquémicas agudas del corazón	410-414	CIE-9 1990-1997	0.8	0.955	0.71	0.88
	I50 I50.0 I50.1 I50.9		I21-I25	CIE-10 1998-2014	0.718	0.74	0.474	0.755

Fuente: Modificado de Naghavi *et al.,* 2010. Annex 3. Details of redistribution packages for exposure to unspecified factor X59, female genital organ malignant neoplasm, unspecified site C57.9, heart failure I50, peritonitis K65, and septicemia A40, A41.

El cuadro V muestra el incremento en el número de muertes por enfermedad isquémica del corazón antes y después de la corrección de las muertes potencialmente mal clasificadas como insuficiencia cardiaca, por delegación y para toda la Ciudad de México para el periodo 1990-2014. Se observa un incremento del 7.5% (12,512) en las muertes como resultado de la reasignación de CB correspondientes a insuficiencia cardiaca.

Cuadro V. Muertes por enfermedad isquémica del corazón antes y después de la corrección de los códigos asignados a insuficiencia cardiaca por delegación en la CDMX, 1990-2014		
Delegación	**Muertes sin corrección (#)**	**Muertes con corrección (#)**
Azcapotzalco	10,096	10,828
Coyoacán	13,183	14,080
Cuajimalpa de Morelos	2,499	2,721
Gustavo A. Madero	25,331	27,224
Iztacalco	8,221	8,767
Iztapalapa	20,444	22,105
La Magdalena Contreras	4,108	4,354
Milpa Alta	1,135	1,283
Álvaro Obregón	12,133	12,996
Tláhuac	3,017	3,338
Tlalpan	9,000	9,595
Xochimilco	5,202	5,666
Benito Juárez	13,472	14,379
Cuauhtémoc	16,298	17,540
Miguel Hidalgo	10,515	11,370
Venustiano Carranza	12,181	13,101
No especificado	240	242
CDMX	167,075	179,587

HISTORICAL ANALYSIS OF AIR QUALITY-RELATED HEALTH BENEFITS IN THE POPULATION IN MEXICO CITY FROM 1990 TO 2012

Phase III. Verification of health benefits due to improved air quality in Mexico City (Epidemiological Analysis)

Final Report

2018

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Layout: Adrenalina Cebrián y Jorge Del Cueto

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EXECUTIVE SUMMARY

Community air pollution concentrations are associated with increased mortality in adults and in children, and air quality improvements reported in multiple cities in the United States and Europe have been associated with increased life expectancy in the population.

As we show in this report, in Mexico City air quality improvements have had public health benefits. In Phase II of this project (*Estimation of the Health Benefits of Air Pollution Improvements in Mexico City*), applying risk assessment approaches, we estimated that more than 20 thousand premature deaths were avoided due to reductions in $PM_{2.5}$ (ambient particulate matter with an aerodynamic diameter ≤ 2.5 μm, also known as fine particles) and ozone community concentrations, from 1990 to 2014. Phase III (*Verification of health benefits due to improved air quality in Mexico City (Epidemiological Analysis)*) now reveals that by using epidemiological methods air quality improvements, that is reductions in $PM_{2.5}$ and ozone levels, have had simultaneous and independent beneficial health impacts, measured as increased life expectancy and of life of years gained.

The broad objective of Phase III is to analyze the public health benefits of air quality improvements in terms of several general measures of population health, including life expectancy, in Mexico City from 1990 to 2015[1]. Our work uses epidemiological methods with direct information on *alcaldía*-specific health outcomes, air pollution, and additional risk factors from Mexico City.

This report includes the evaluation of *alcaldía*-specific health benefits associated with air pollution improvements, adjusted for socioeconomic position indicators, proxy[2] indicators for the prevalence of cigarette smoking, among other risk factors from 1990 to 2015. Public health benefit indicators included in our cross-sectional and longitudinal[3] modeling are *alcaldía*-specific life expectancy at birth, temporary life expectancy for children (0 to 4 years old)and for adults (25 to 74 years old), years of life lost in children and adults, and years of life lost from five specific causes of death determined to be causally associated with chronic air pollution exposures in the Global Burden of Disease analyses and included in our Phase II work of this project --ischemic heart disease, cerebrovascular stroke (hemorrhagic and ischemic), chronic obstructive pulmonary disease (COPD), and lung cancer, in adults, and acute lower respiratory infections (ALRI) in children.

We briefly describe time trends and spatial distribution of indicators for health outcomes, air pollution and socioeconomic position, as well as for other explanatory variables included in the analyses. Also

[1] Our study was extended to 2015 because variables included in our analyses were available for that year --although the project's official title refers to a shorter period, 1990-2012.

[2] A proxy variable is not relevant in and on itself but serves in place of some other variable that has not been observed, included or measured. An adequate proxy variable is correlated with the variable of primary interest.

[3] Cross-sectional analyses evaluate the relationship between variables at a given time; in this case, the analysis is for each census and intercensal years. Longitudinal analyses evaluate the relationship across time, which in our study translates into incorporating all census and intercensal years of *alcaldía*-specific data for population and socioeconomic position indicators.

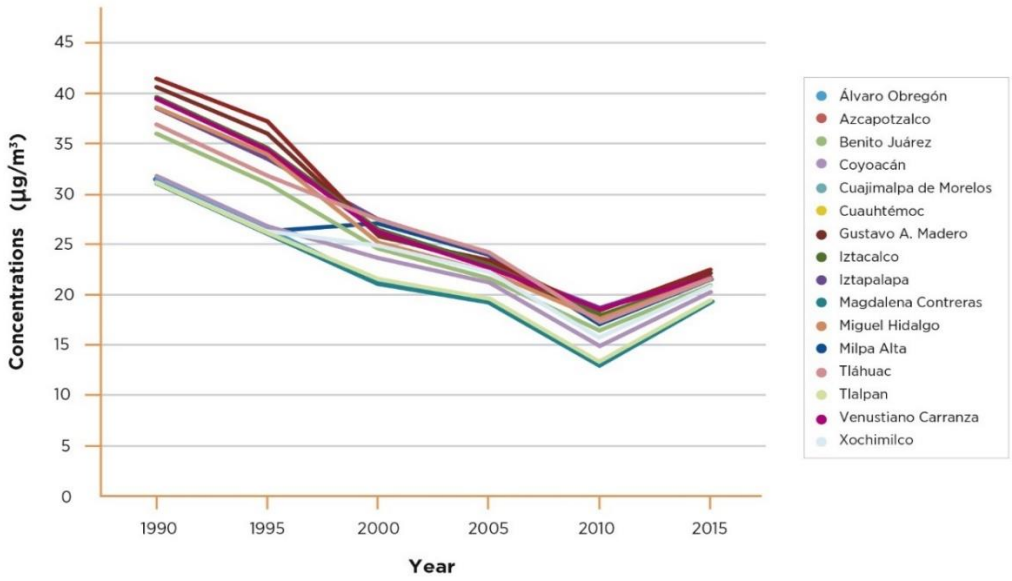
presented are our results from the cross-sectional correlations and longitudinal modeling for the relationship between health outcomes and air pollution.

Air Quality

Data from SEDEMA's air quality network was integrated and analyzed. In our work for Phase II, we computed fine particles ($PM_{2.5}$) and ozone exposure metrics from 1990 to 2015 and used such estimates for the present analyses. $PM_{2.5}$ concentrations were predicted via a generalized additive model for years prior to official and continuous air quality fixed-site monitoring efforts (1990 to 2003). For ozone, official data from the monitoring network is available for the entire study period. The indicators of exposure for each pollutant are those used in cohort epidemiological studies and have been associated with adverse health impacts. For $PM_{2.5}$ we estimated annual average concentrations, and for ozone seasonal (6-month) 1-hour maximum daily concentrations. Lastly, spatial interpolation methods were applied to determine $PM_{2.5}$ and ozone from monitoring site to *alcaldía* level, which is the spatial resolution level of health outcomes and the other risk factors.

In the past 25 years ambient $PM_{2.5}$ and ozone levels have significantly declined in Mexico City. In the early 1990s annual average $PM_{2.5}$ concentrations exceeded 40 µg/m^3 in some *alcaldías* and average concentration in the City was close to 36 µg/m^3. By 2015 levels in all *alcaldías* were below 23 µg/m^3 and city-wide average levels equaled 21 µg/m^3 (Fig. I). Interestingly, in 2010 concentrations reached their lowest levels in the City, likely related with unusually heavy rains during typically high particle level months (January and February) washing out ambient particles.

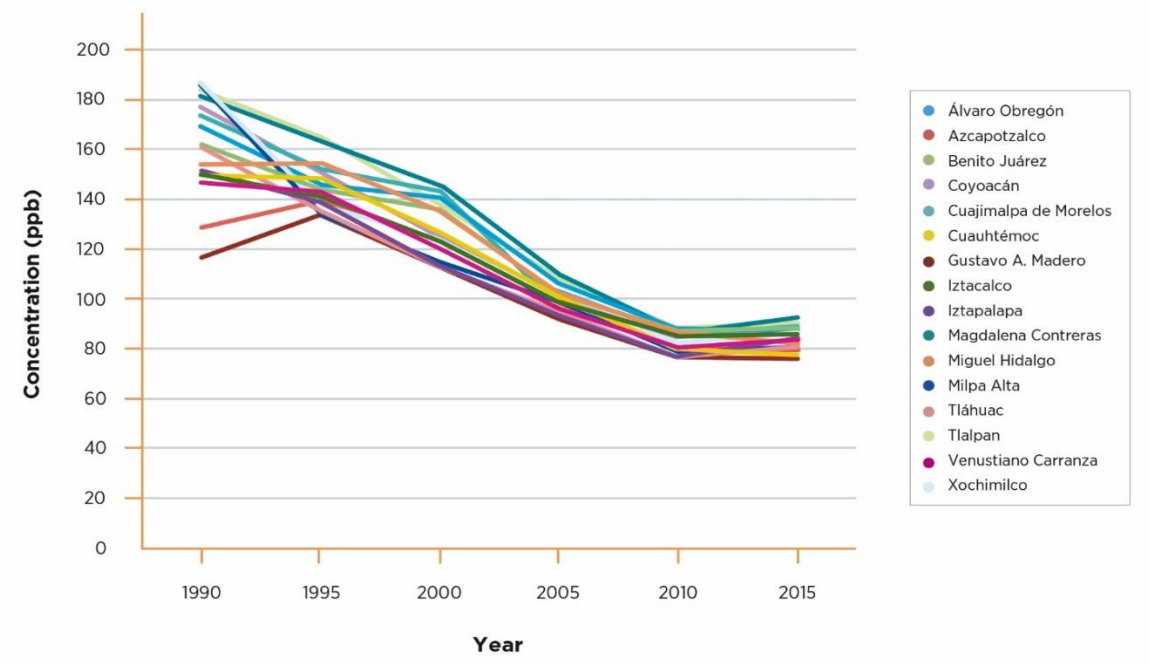
Figure I. Time-trends of alcaldía*-specific annual average $PM_{2.5}$ concentrations (µg/m^3)*



Ozone has also shown very significant improvements in Mexico City (Fig. II). City-wide average levels, estimated as seasonal (6 month) 1-hour daily maximum concentrations, in 1990 were above 160 ppb, and

ranged between 117 and 185 ppb among City *alcaldías*. The steady decline in ozone concentrations through the City led to 2015 mean levels of 84 ppb, and values below 91 ppb prevailing in all City *alcaldías*.

Figure II. Time-trends of alcaldía*-specific seasonal (6 month) 1-hour maximum ozone concentrations (ppb)*

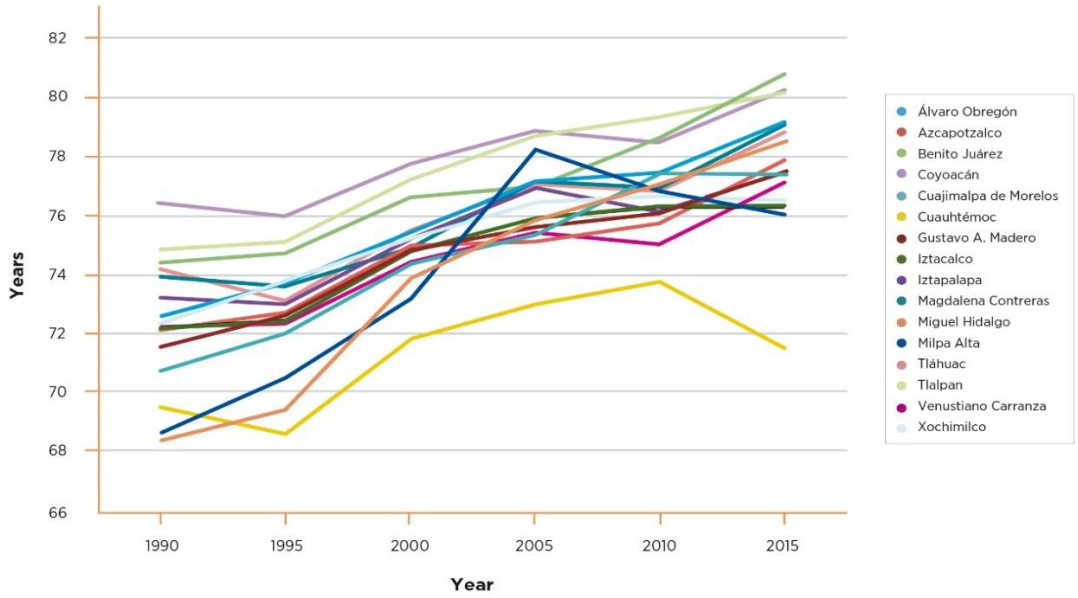


Life Expectancy and Temporary Life Expectancy

Health outcome indicators included in our analyses were computed from official death counts for the *alcaldías* of Mexico City. To illustrate how life expectancy has changed through time and by *alcaldía*, we will describe life expectancy at birth, and temporary life expectancy for children and for adults.

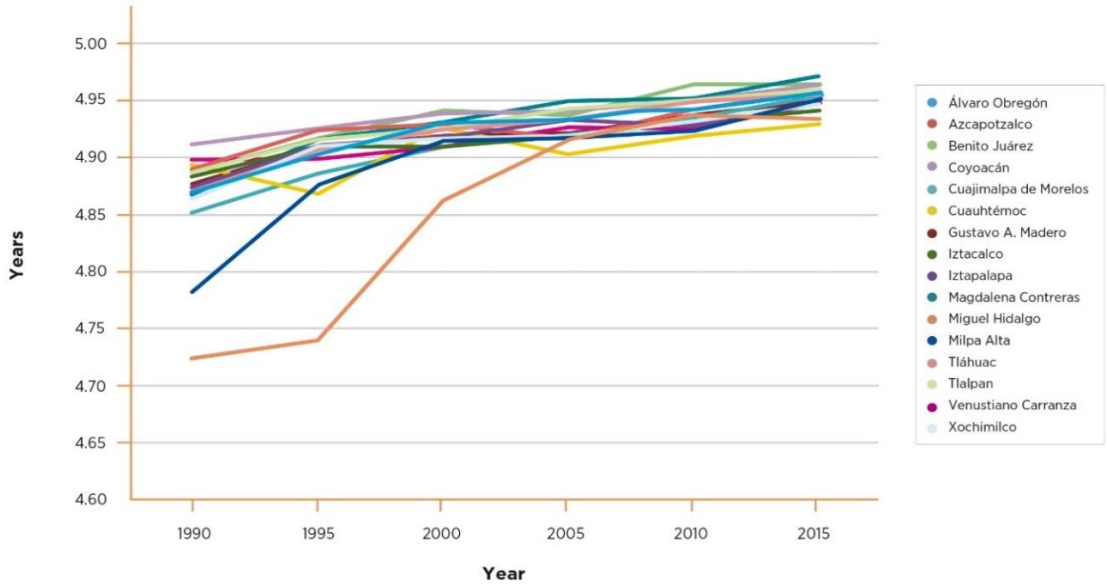
In Mexico City life expectancy at birth for total population increased by almost 8% from 1990 to 2015, going from close to 72 years to almost 78 years. Males and females presented a similar relative increase for the same period, with life expectancy in men going from 69 to close to 75 years, and in women from 75 to slightly over 80 years. Most *alcaldías* in Mexico City showed a steady increasing trend in life expectancy throughout the study period, the most notable exceptions are Milpa Alta and Cuauhtémoc, with declining life expectancy from 2005 and 2010, respectively (Fig. III).

Figure III. Time trends of alcaldía-*specific life expectancy at birth (years)*



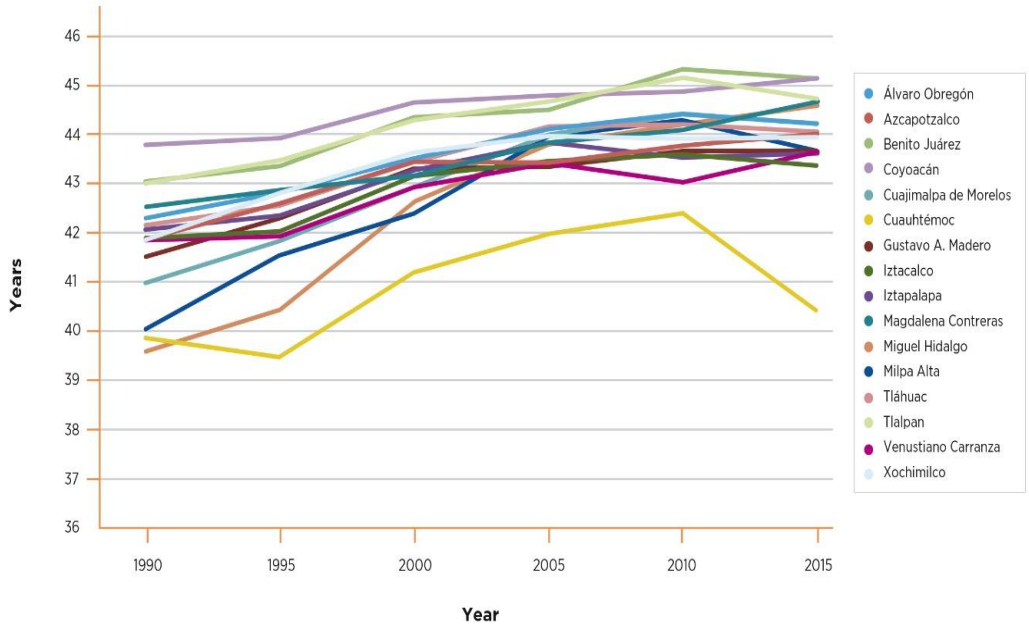
For children 0-4 years, temporary life expectancy shows a steady and light increase in most *alcaldías*, with average temporary life expectancy in 1990 of 4.87, compared to a possible of 5 years, and of 4.95 years by 2015 (Fig. IV). Variability in temporary life expectancy was evident among *alcaldías* from 1990 to 2000, but by 2015 very tight values prevailed among them.

Figure IV. Time trends of alcaldía-*specific temporary life expectancy (years) for population between 0 to 4 years old*



For adults aged 25 to 74, the temporary life expectancy in 1990 was close to 42 years, compared to a total possible of 50 years, and it rose to almost 44 years by 2015 (Fig. V). Temporary life expectancy between 25-74 years shows a very similar time-trend pattern as it did for total life expectancy, with a steady and increasing trend in most *alcaldías*, but in Cuauhtémoc, where temporary life expectancy decreased from 2010 to 2015.

Figure V. Time trends of alcaldía*-specific temporary life expectancy (years) for population between 25 to 74 years old*



Smoking Related Diseases

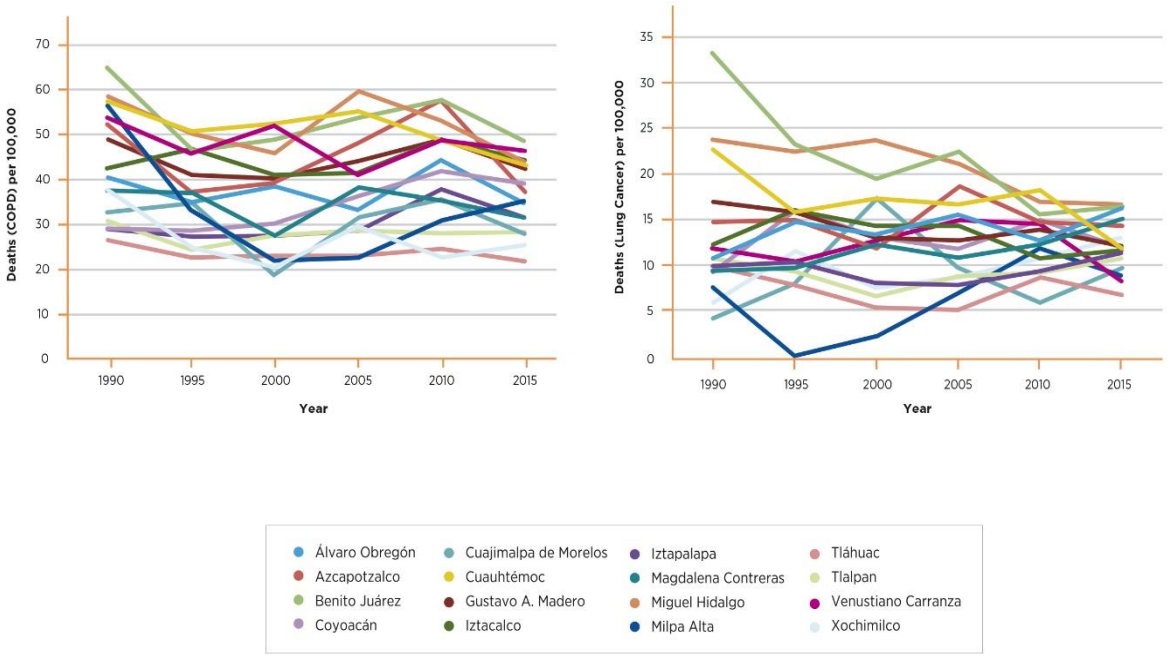
Due to smoking prevalence data constraints,[4] our analyses used death rates for COPD and lung cancer as indicators of accumulated exposure to smoking to adjust for prevalence of smoking at *alcaldía* level. By using COPD and lung cancer death rates—which are causally associated with smoking-- in our analyses we're able to provide a conservative estimate of the effect of air pollution adjusted for smoking.

[4] Information on addictions has been collected with national surveys, The National Health Survey (ENSA, 2000), the National Health and Nutrition Survey (ENSANUT, 2006 and 2012), and the National Addictions Survey (ENA, 2011 and 2016). Such surveys' results were not used because they are not representative at the municipal level (or *alcaldía* for Mexico City) as needed for this study --interviews for some *alcaldías* had very small (or null) sample sizes--, and the definition of a smoker differed between surveys --in one it refers more likely to an occasional smoker, and in the other, to a habitual smoker.

The COPD mortality rate in Mexico City dropped during the study period, from 44 deaths per 100,000 in 1990 to 36 deaths per 100,000 in 2015. There was barely no between-*alcaldía* variability through the period (Fig. VI).

Lung cancer mortality rates, which include trachea, bronchus and lung cancers, in Mexico City have barely changed over the 25-year study period. Mortality rates have gone from 13 to 12 deaths per 100,000 from 1990 to 2015 (fig. VI). Notable improvements in *alcaldía*-specific lung cancer rates were present in Benito Juárez, Cuauhtémoc, and Miguel Hidalgo.

Figure VI. Time trends of alcaldía*-specific COPD and lung cancer mortality rates (deaths per 100,000*)



Socioeconomic Position Indicators

Life expectancy may be affected by air pollution and other risk factors, that encompass socioeconomic position characteristics. This study relies on CONAPO's socioeconomic position indicators constructed with data from the General Census of Population and Households (1990, 2000 and 2010), and from the Counts of Population and Households (1995, 2005 y 2015) (Table I).

Table I shows the behavior over time of the overall fraction (%) of the population of Mexico City reporting each of CONAPO's socioeconomic position indicators for 1990 to 2015.

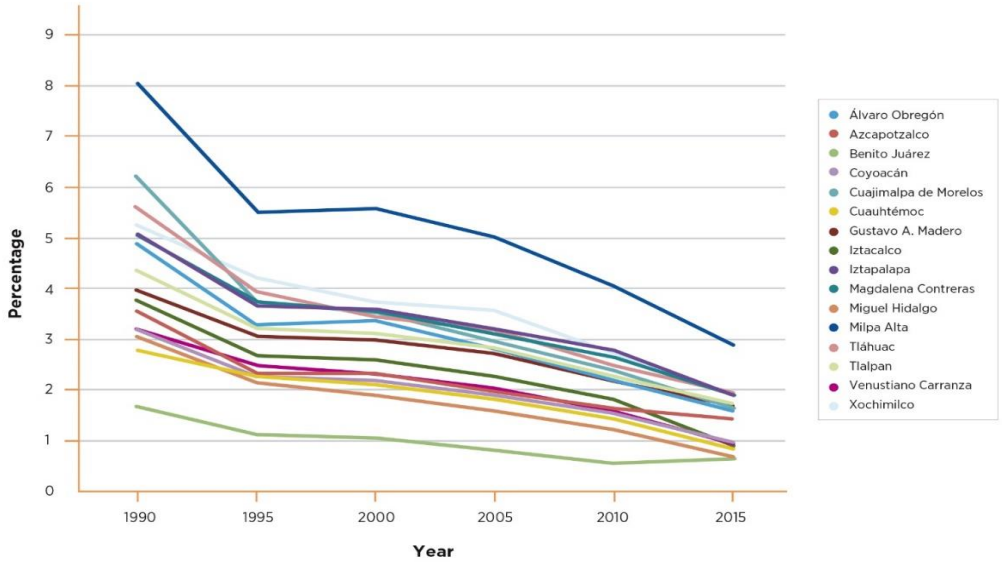
Table I Behavior of Socioeconomic Position Indicators for Mexico City (%), 1990-2015

Socioeconomic Position Indicators	Illiteracy (ANALF)	Low Education (SPRIM)	No Sewer nor Toilet (OVSDE)	No Electricity (OVSEE)	No Running Water (OVSAE)	Overcrowding (VHAC)	Soil Floor (OVPT)	Small Villages (PL<5000)	Low Income (PO2SM)
1990	4.36	17.5	2.94	1.12	4.30	48.0	3.55	1.32	61.7
1995	3.11	12.0	2.56	0.11	2.87	46.7	1.11	1.29	47.4
2000	2.97	12.2	0.60	0.21	1.78	35.5	1.75	1.08	42.3
2005	2.61	9.6	0.19	0.16	2.04	29.8	1.40	1.53	32.7
2010	2.09	8.5	0.11	0.10	2.37	26.3	1.22	1.83	28.4
2015	1.48	6.5	0.05	0.06	1.70	19.6	0.58	1.83	28.0

Source: CONAPO, 2018

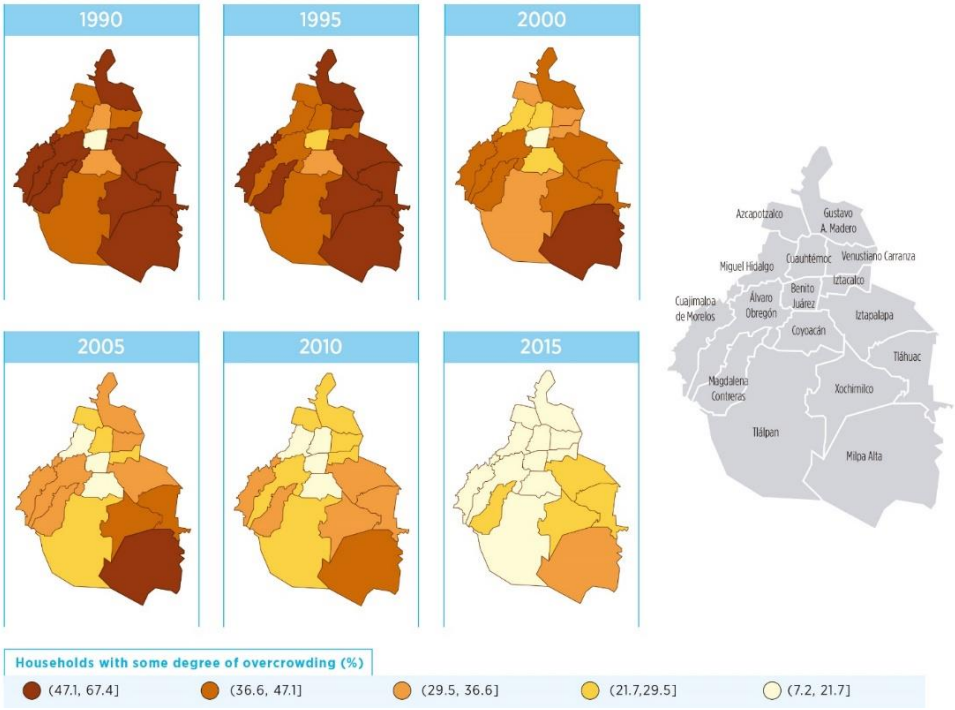
There have been substantial improvements in these socioeconomic position indicators between 1990 and 2015 in Mexico City. Just to illustrate, we name two of the indicators and their corresponding temporal trends and spatial distribution. For illiteracy among those 15 years or older in Mexico City, the percentage dropped from close to 4.4% in 1990 to almost 1.5% in 2015 with all *alcaldías* following this trend towards a smaller fraction of illiterate population (Fig. VII).

Figure VII. Time trends of alcaldía*-specific illiterate population aged 15 years and above (%)*



Similarly, significant improvements for the indicator of households with some degree of overcrowding were evident for all *alcaldías*. In the city the fraction of houses with some degree of overcrowding dropped to less than half from 1990 to 2015, going from close to 48% to 20% (Fig. VIII).

Figure VIII. Spatial distribution of alcaldía*-specific households with some degree of overcrowding (%) by year*



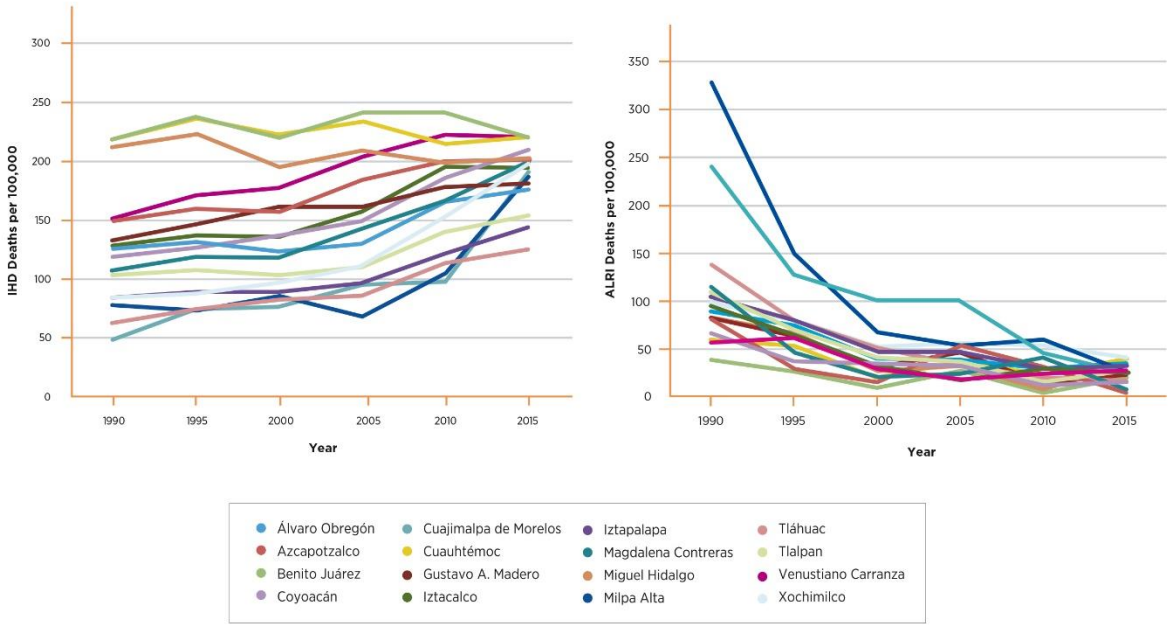
For both socioeconomic position indicators, we find that Milpa Alta consistently lagged with a higher fraction of illiterate population and more households with some degree of overcrowding. The opposite holds true for Benito Juárez, with lower fractions for both indicators.

Air Pollution Related Diseases

Our analysis focuses in the health benefits associated with air quality improvements. Health effects were measured with several health indicators, including years of life lost from the five selected causes of death that are causally associated with exposures to $PM_{2.5}$ or ozone. Time trends and spatial distribution for ischemic heart disease (IHD) and cerebrovascular stroke in adults, and ALRI in children are briefly presented below --COPD and lung cancer were described in the section of Smoking Related Diseases.

IHD is one of the leading causes of death in Mexico City, and the overall rate has increased, going from 126 deaths per 100,000 in 1990 to 189 deaths per 100,000 in 2015 (Fig. IX). Most *alcaldía*-specific rates of IHD mortality show this rising trend from 1990 to 2015.

Figure IX. Time trends of alcaldía*-specific IHD and ALRI mortality rates (deaths per 100,000)*



Cerebrovascular stroke mortality rates in Mexico City have been pretty stable over the 25-year study period and have remained in the range of 22 deaths per 100,000 since 1990. Some between-year variability has been present, but there has not been much between-*alcaldía* variability (See Appendix VI).

The ALRI mortality rate among children decreased dramatically in the City. These mortality rates decreased from 110 deaths per 100,000 in 1990 to 24 per 100,000 in 2015. The *alcaldías* with the largest decrease in ALRI mortality rates are Milpa Alta and Cuajimalpa de Morelos (Figure IX).

Reference Diseases

Changes in life expectancy or in years of life lost are likely to be associated with factors not captured by the socioeconomic position indicators described above. Such risk factors may include individual behavioral factors like nutrition, or institutional factors such as access to health care. To provide some insight into these unmeasured risk factors, we include "reference" causes of death, which are common causes of death not expected to be associated or only weakly associated with air pollution. Reference causes of death are: diabetes, hypertension, colon cancer, stomach cancer, and external causes (assault). (See Appendix VII for figures and maps with time trends and spatial distribution of Reference Diseases.)

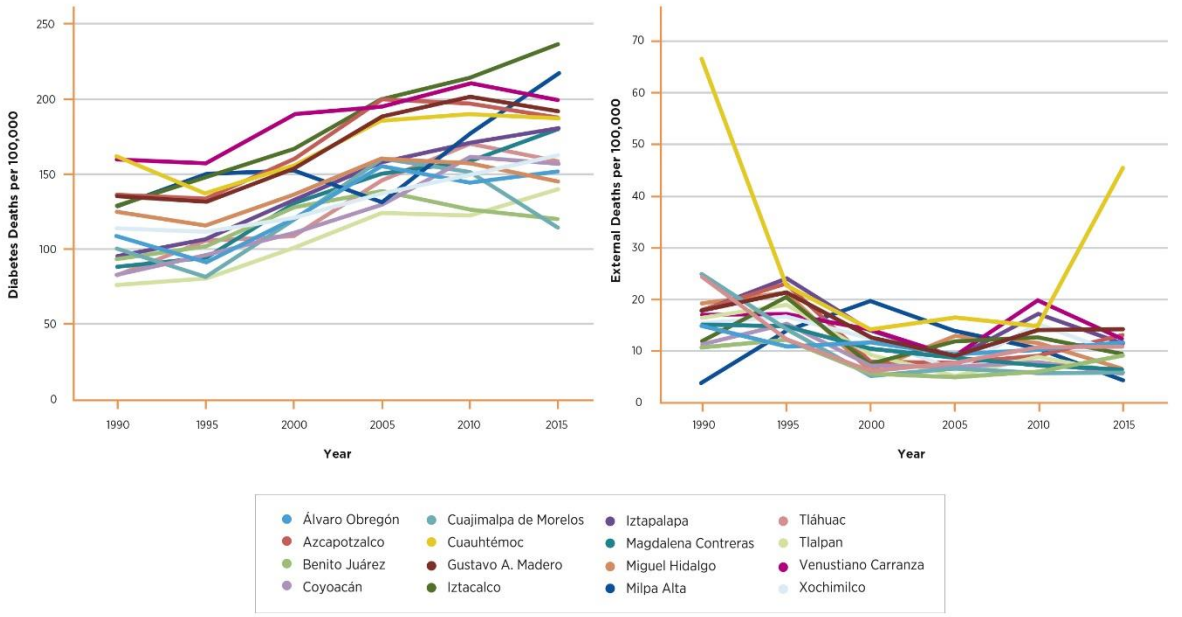
Significant increases were found for diabetes, hypertension, and colon cancer mortality rates in Mexico City. Diabetes mortality rates increased from 117 per 100,000 in 1990 to 172 in 2015. The *alcaldía*-specific rates are consistent in showing a similar increase from 1990 to 2015 -with some starting to show a reversal trend since 2010.

In the city, hypertension mortality rates increased by about 30% (25 per 100,000 in 1990 to 33 per 100,00 in 2015), and those for colon cancer have grown in over 80% doubled (6.6 per 100,000 in 1990 to 12 per 100,000 in 2015).

Mortality rates of stomach cancer in Mexico City have shown little change from 1990 to 2015, remaining close to 11 per 100,000 throughout the period.

External causes include deaths by assault –by homicide and injuries inflicted by another person with intent to injure or kill, by any means. There is not much heterogeneity in the *alcaldía*-specific rates of mortality due to external causes; the exception to this pattern is Cuauhtémoc, with higher rates than the rest of the *alcaldías* in 1990 and 2015.

Figure X. Time trends of alcaldía-specific diabetes and external causes' mortality rates (deaths per 100,000)



Results

Correlations

The primary focus of our analyses is to assess the relationship between the different measures of life expectancy and air pollution ($PM_{2.5}$ and ozone) over time within each of the 16 *alcaldías* of Mexico City. In a first cut to the analysis, we find consistent negative correlations between *alcaldía*- and year-specific $PM_{2.5}$ and O_3 and each of the measures of life expectancy (Table II). We also found positive correlations for measures of years of life lost versus corresponding measures of air pollution (results not shown). This means that higher *alcaldía*- and year-specific air pollution levels are correlated with lower life expectancy or with an increase in years of life lost.

There are also correlations between many of the socioeconomic position indicators and life expectancy measures (negative correlations) (Table II) or years of life lost measures (positive correlations) (results not shown), which strongly indicate the importance of controlling for socioeconomic position and other *alcaldía*- and year-specific risk factors as alternative explanations for the apparent associations between life expectancy measures or life years lost measures and air pollution.

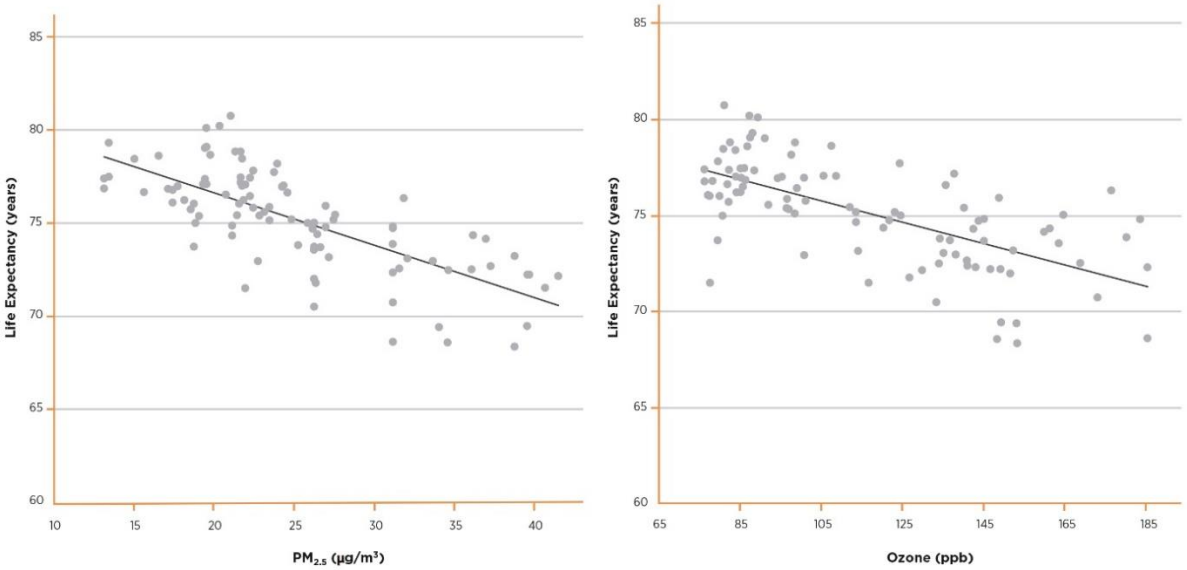
Table II. Correlations between alcaldía- *and year-specific life expectancy and temporary life expectancy with air pollution and socioeconomic position indicators*

Air Pollution and Socioeconomic Position Indicators		Life Expectancy			Temporary Life Expectancy	
		Total	Men	Women	0-4 Years	25-74 Years
Fine Particles	$PM_{2.5}$	-0.73	-0.68	-0.75	-0.65	-0.72
Ozone	O_3	-0.66	-0.59	-0.70	-0.67	-0.59
Low Income	PO2SM	-0.68	-0.56	-0.77	-0.62	-0.65
Overcrowding	VHAC	-0.64	-0.50	-0.75	-0.56	-0.57
Low Education	SPRIM	-0.57	-0.43	-0.66	-0.57	-0.52
Illiteracy	ANALF	-0.50	-0.36	-0.59	-0.53	-0.45
No Sewer nor Toilet	OVSDE	-0.44	-0.36	-0.47	-0.46	-0.40
No Electricity	OVSEE	-0.35	-0.30	-0.34	-0.48	-0.36
Soil Floor	OVPT	-0.26	-0.19	-0.30	-0.37	-0.25
No Running Water	OVSAE	-0.09	0.00	-0.15	-0.18	-0.06
Small Villages	PL<5000	-0.04	0.03	-0.09	-0.06	-0.03
Total Population	POB_TOT	0.07	0.08	0.06	0.11	0.03

Bi-Variate Associations

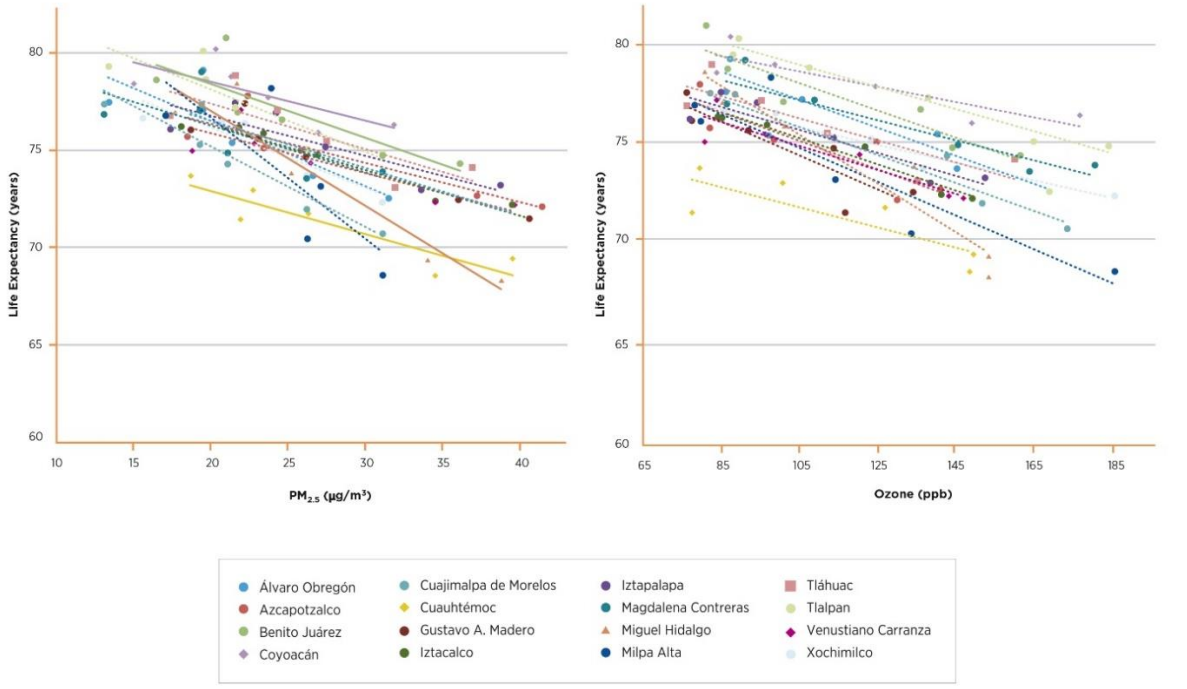
Life expectancies plotted against $PM_{2.5}$ or ozone concentrations for 1990 through 2015, with the cross-sectional (spatial) relations across *alcaldías* and the longitudinal (temporal) relations across years, clearly suggest negative associations between *alcaldía*- and year-specific life expectancy and $PM_{2.5}$ or ozone (Fig. XI). The estimated coefficients of such correlations for $PM_{2.5}$ and for ozone are -0.73 and -0.66, respectively (Table II).

Figure XI. Scatterplots and fitted lines of alcaldía- *and year-specific life expectancy versus corresponding measures of annual average $PM_{2.5}$ concentrations (left pane) and seasonal (6 month) 1-hour maximum ozone concentrations (ppb) (right pane)*



The following figures present the same data showing the longitudinal relations by *alcaldía* between life expectancy and $PM_{2.5}$ or ozone (left and right panes, respectively, in Fig. XII). Negative longitudinal (within each *alcaldía*) associations between life expectancy and $PM_{2.5}$ or ozone are observed in both scatter plots.

Figure XII. Scatterplots and fitted lines of year-specific life expectancy versus annual average $PM_{2.5}$ concentrations (right pane) and seasonal (6 month) 1-hour maximum ozone concentrations (ppb) (right pane) by alcaldía



Regression Analyses: Methods and Results

As a general measure of population health, life expectancy reflects the net effects of the full range of risk factors including genetics, behaviors, the environment, community context, and efficacy of medical treatment and care. Thus, *alcaldía* air pollution levels are only one of a myriad of individual, population, and community factors affecting life expectancy. How can we control for possible confounding by these known and unknown factors?

We built mixed models for the health outcome indicators in a stepwise approach. We assumed that each *alcaldía* has a unique set of characteristics that define life expectancy compared to the other *alcaldías* (random effect), and that life expectancy across all the *alcaldías* changed year-to-year (random effect). We estimated a regression coefficient common to all *alcaldías* for each pollutant, $PM_{2.5}$ and O_3 (fixed effect for each). As population size between *alcaldías* and years shows significant variability, we gave more weight to the points with larger populations, that is weighting by the square root of the population.

Our approach was to build-up from an agnostic model that simply includes the above set of core variables (Simple Model) for total life expectancy versus $PM_{2.5}$ and O_3, to a Parsimonious Model that efficiently adjusted for *alcaldías*, year, indicators of socioeconomic position, proxy indicators for smoking, and general health status, weighted by population. We examined this model for total life expectancy of the whole population and for men and women separately.

We applied the Parsimonious Model to the years of life lost for children (0-4 years) and adults (25-74 years), and to the estimated years of life lost due to five specific mortality causes that have been determined to be related with long-term air pollution exposures in the Global Burden of Disease and in our Phase II work – ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, lung cancer, in adults, and acute lower respiratory infections in children.

Results from our Parsimonious Model on total life expectancy showed independent and significant impacts of both, $PM_{2.5}$ and O_3: each 10 $\mu g/m^3$ decrease in $PM_{2.5}$ was significantly associated with an increase in life expectancy of 0.89 years (95% CI 0.14 to 1.65 years), and each 10 ppb improvement in O_3 with a life expectancy increase of 0.24 years (95% CI 0.08 to 0.40 years) (Table III). Our analyses of life expectancy for men and women indicates the same effect of $PM_{2.5}$ for men (0.79 years, 95% CI -.14 to 1.72 years) and women (0.79 years, 95% CI 0.33 to 1.45 years). For O_3 women had much smaller effect estimates (0.13 years, 95% CI -.03 to 0.28) compared to men (0.36 years, 95% CI 0.15 to 0.57), although these sex-specific effect estimates have overlapping confidence intervals.

Our results for gains in life of years between ages 25 and 74 years indicate a highly significant association with $PM_{2.5}$ (0.56 years, 95% CI 0.28 to 0.83) and with O_3 (0.10 years, 95% CI 0.03 to 0.17). For this age-group there was a significant increase in years of life attributable to ischemic heart disease and to COPD associated with $PM_{2.5}$ improvements. For years of life gained between ages 0 and 4 years there was a modest, statistically non-significant association with $PM_{2.5}$ (0.0070 years or 2.5 days) and a small significant association with O_3 (0.0037 years or 1.3 days). We found no positive association with years of life gained from acute lower respiratory infections.

Table III. Weighted regressions of total life expectancy and life expectancy for men and for women versus fixed effects of air pollution, adjusting for fixed effects of socio-economic position, reference death rates and proxy indicators for prevalence of smoking, and for random effects of alcaldías *and census years*

Variable	Total Life Expectancy			Life Expectancy Men			Life Expectancy Women		
	Beta	SE	p value	Beta	SE	p value	Beta	SE	p value
Intercept	90.43	1.73	<0.0001	90.29	2.23	<0.0001	89.62	1.47	<0.0001
$PM_{2.5}$ (10µg/m³)	-0.89	0.38	0.028	-0.79	0.47	0.12	-0.79	0.33	0.026
O_3 (10 ppb)	-0.24	0.08	0.004	-0.36	0.11	0.0011	-0.13	0.08	0.108
Overcrowding	-0.11	0.02	<0.0001	-0.11	0.03	0.0015	-0.11	0.02	<0.0001
Diabetes Death Rate	-0.02	0.01	0.002	-0.02	0.01	0.0059	-0.01	0.01	0.024
Colon Cancer Colon Cancer Death	-0.08	0.05	0.107	-0.15	0.06	0.022	0.01	0.05	0.83
External Causes (Assault) Death Rate	-0.02	0.01	0.094	-0.05	0.02	0.014	-0.01	0.01	0.56
COPD Death Rate	-0.05	0.02	0.012	-0.06	0.02	0.021	-0.05	0.02	0.009
Lung Cancer Death Rate	-0.03	0.03	0.37	-0.03	0.04	0.52	-0.05	0.03	0.095
Alcaldía (Random)			<0.0001			<0.0001			0.0026
Census Year (Random)			0.078			0.30			0.080

Closing Remarks

Life expectancy of Mexico City inhabitants is affected by exposures to air pollution. We found that over the past 25 years air quality improvements in Mexico City have been associated with increased life expectancy. Improvements in each 10 µg/m³ annual average $PM_{2.5}$ are associated with 0.89 years (95% CI -0.14 to 1.65 years) longer life expectancy at birth. We also found a simultaneous and independent effect for O_3, improvements in each 10 ppb seasonal average peak O_3 are associated with 0.24 years (95% CI 0.08 to 0.40 years) longer life expectancy.

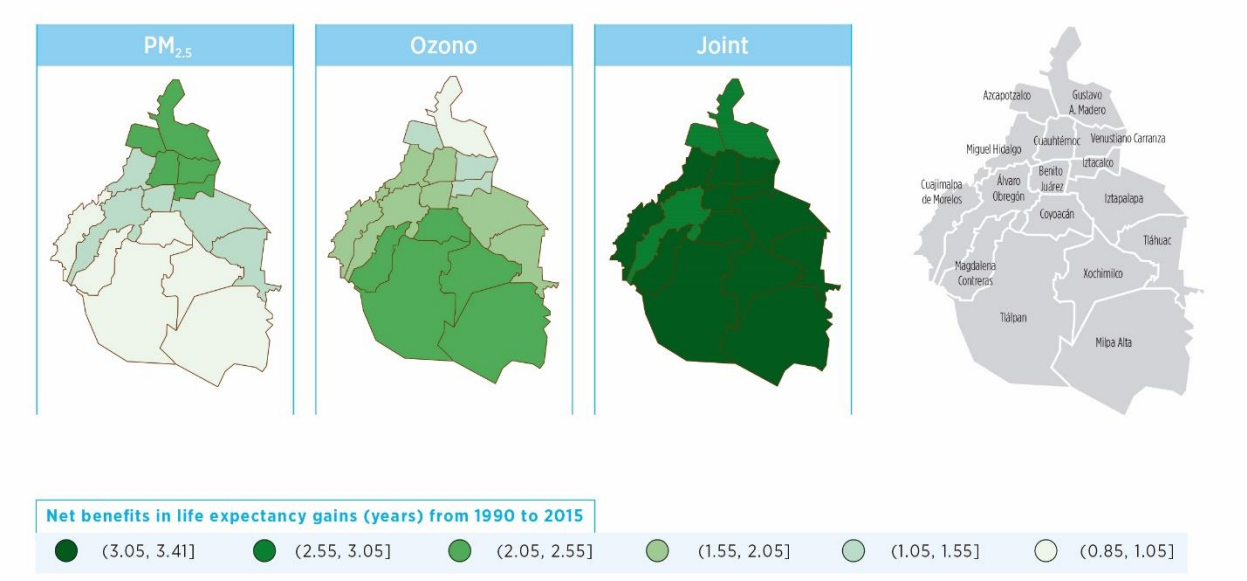
Our results show strong associations with years of life lost with $PM_{2.5}$ and O_3 among adults 25 to 74 years of age: Each 10 µg/m³ improvement in $PM_{2.5}$ was associated with 0.56 years (95% CI 0.28 to 0.83)

reduction in life years lost, and each 10 ppb improvement in O_3 with 0.10 (95%CI 0.03 to 0.17) reduction in life years lost. This is consistent with the cohort studies, such as the Harvard Six Cites study (Dockery et al., 1993), which found that dirtier $PM_{2.5}$ cities had lower survival and higher mortality rates than the cleanest city.

The evidence for shorter life expectancy in Mexico City associated with $PM_{2.5}$ is very consistent with similar studies conducted in county-specific life expectancy changes in the United States. Pope et al. (2009) and Correia et al. (2017) reported, respectively, that life expectancy increased by 0.61 years (95% CI 0.22 to 1.00) and 0.35 years (95% CI 0.04 to 0.66) associated with each 10 $\mu g/m^3$ improvement in annual average $PM_{2.5}$.

There is limited evidence that living in communities with higher O_3 is associated with increased mortality and shorter life expectancy. The differential $PM_{2.5}$ and O_3 spatial variability, together with the wide range of O_3 concentrations seen across the study period, allowed us to have the statistical power to detect a simultaneous and independent effect for $PM_{2.5}$ and for O_3. This is an important contribution to the scientific evidence of population health benefits that result from improved ozone air quality.

Figure XII. Independent and joint net benefits measured as life expectancy gains (years) from improved $PM_{2.5}$ (annual average concentrations) and ozone (seasonal maximum 1-hour daily concentrations) in Mexico City, 1990 - 2015



Our Parsimonious Model indicates that net benefits in Mexico City, associated with air quality improvements in $PM_{2.5}$ and ozone from 1990 to 2015, represent a life expectancy gain of 3.2 years (Fig. XII). This life expectancy increase incorporates the benefits due to improvements in $PM_{2.5}$ annual concentrations of almost 15 $\mu g/m^3$, and in seasonal hourly O_3 peaks of close to 80 ppb. Thus, the joint net benefit associated with improvements in both pollutants represents an increase in life expectancy of 3.2 years. As seen in figure XII, net benefits present a different spatial pattern for $PM_{2.5}$ and O_3. We found

greater improvements in $PM_{2.5}$ air quality in the north, while greater O_3 improvements are seen in the south. These air quality improvements led to larger gains in life expectancy in the northern *alcaldías* attributable to $PM_{2.5}$ (up to 1.7 years) and greater life expectancy gains in the southern *alcaldías* attributable to O_3 (up to 2.6 years). The joint effects of $PM_{2.5}$ and O_3 improvements led to substantial life expectancy gains (2.6 to 3.4 years) in all *alcaldías* (Figure XII).

Apte et al. (2018) have recently estimated that current (2016) $PM_{2.5}$ exposures reduce life expectancy globally by 1.03 years, and O_3 exposures by 0.05 years. They suggest that if air quality improved globally to meet the Air Quality Guideline for $PM_{2.5}$ of 10 $\mu g/m^3$ from the World Health Organization median life expectancy could increase by 0.6 year (interquartile range of 0.2−1.0 year). This benefit is equivalent to that of eradicating lung and breast cancer together. These authors report that average exposures to $PM_{2.5}$ for our country of 18.3 $\mu g/m^3$ imply an average of 0.48 years life lost due to $PM_{2.5}$.

Our results, based on analyses using direct *alcaldía*-specific information on air quality and health-related outcomes, adjusting for socioeconomic position, general health indicators and proxy indicators of accumulated exposure to smoking, are consistent with world-wide most recent findings that indicate that air quality improvements have beneficial public health effects, measured as increased life expectancy and reduced life years lost. Public policies aimed at further improving air quality will continue to benefit public health.

INTRODUCTION

The Secretaries of Environment (SEDEMA) and Health (SEDESA) of the government of Mexico City initiated a program of collaboration with the Harvard T.H. Chan School of Public Health in 2015. The focus of the program is on Mexico City's air quality, and its general objective is to analyze the health benefits for the inhabitants of Mexico City associated with the improvements in air quality achieved over the last 20 years through implementation of air quality policies. These benefits are being assessed through the interpretation and application of international and national scientific information in the fields of epidemiology and air quality, as well as with the implementation of risk assessment and policy analyses methodologies.

The program consisted of four phases, three of which have been completed. Phase I (*State of Knowledge and Relevance to Mexico City*) focused in a literature review on the state of knowledge and the scientific evidence was described through examination of epidemiological studies carried out to date. These studies constitute the basis for interpreting the relationship between exposure to atmospheric pollutants and adverse health impacts. In Phase II (*Estimation of the Health Benefits of Air Pollution Improvements*), also completed in 2016, risk assessment methods were used to estimate health benefits attributable to reductions in $PM_{2.5}$ and ozone concentrations achieved in Mexico City between 1990 and 2014.

In 2017, during Phase IV (*Public Policy and Economic Valuation of the Health Benefits of Air Quality Improvements*), a cost-effectiveness analysis was conducted to assess the benefits associated with the reduction in emissions of fine suspended particles ($PM_{2.5}$) from heavy diesel vehicles, considering that this pollutant has been associated with adverse health impacts. For such purposes, the Mexico City 2014 Emissions Inventory information was used, the health benefits in the Mexico City population were modeled, the costs associated with the emissions control alternatives were estimated, and health benefits were quantified in monetary terms (economic valuation).

Phase III, *Verification of health benefits due to improved air quality in Mexico City (Epidemiological Analysis)*, seeks to assess the relationship between population health benefits, including increased life expectancy, and improvements in air quality in Mexico City from 1990 to 2015. We use epidemiological methods with direct information on multiple factors that may influence the health-air pollution relationship. In 2017, during Phase III a), we constructed a rich database that includes information on air pollution, health outcomes --measured as cause-specific mortality--, population, and socioeconomic position indicators. Pursuant to the scope of this multiphase project, this document is the final report of Phase III b).

This document includes the analyses of the association between public health benefits and risk factors of the community, with focus on air quality, in the population of Mexico City. We include the results of cross-

sectional and longitudinal analyses[5] of the relationship between *alcaldía*[6]-specific life expectancy at birth and *alcaldía*-specific air quality ($PM_{2.5}$ and ozone), for census and intercensal years.[7]

The report includes, as well, the results of the extension of the *alcaldía*-specific repeated cross-sectional and longitudinal analyses for years of lost life expectancy (temporary life expectancy) for children (0 to 4 years old) and for adults (25 to 74 years old) –causes of death have been determined to be causally associated with air pollution exposures for children and adults over 25 years old. Also contained are results of the *alcaldía*-specific cross-sectional and longitudinal analyses for life lost (years of life lost) in children and adults, and from specific causes of death determined to be causally associated with chronic air pollution exposures in the Global Burden of Disease analyses and included in our Phase II work of this project --ischemic heart disease, cerebrovascular stroke (hemorrhagic and ischemic), chronic obstructive pulmonary disease (COPD), and lung cancer, in adults, and acute lower respiratory infections (ALRI) in children.

This document presents a brief description of time trends and of spatial distribution of air pollution ($PM_{2.5}$ and ozone), population health-related outcome indicators, and socioeconomic position indicators. Results of the cross-sectional and longitudinal analyses follow, and it ends with a discussion of main findings.

[5] Cross-sectional analyses evaluate the relationship between variables at a given time; in this case, the analysis is for each of the census and intercensal years. Longitudinal analyses evaluate the relationship between variables over time and, in this case, the analysis is for the period 1990-2015.

[6] Since 2018 geopolitical and administrative entities are called *alcaldías* in Mexico City (*delegaciones* until then). In the rest of the county these are called *municipios*.

[7] Our unit of analysis is the *alcaldía*, thus our analyses were conducted for the years 1990, 1995, 2000, 2005, 2010 and 2015, in agreement with available official information on population and socioeconomic position indicators derived from population and household census and intercensal counts.

AIR POLLUTION, HEALTH OUTCOMES AND OTHER RISK FACTORS

Air pollutants, health outcomes and other risk factors that are comprised in the analyses are defined and are described in terms of time trends (1990-2015) specific by *alcaldía* (n=16), and of spatial distribution among *alcaldías*. Appendices II to VII present time trends and spatial distribution of the whole suite of indicators that are included in our analyses.

The description and behavior of variables that may explain health outcomes encompass factors of the community, and those related to lifestyle. Among the first ones we include our main predictor, focus of our study, air pollution. Air pollution indicators refer to chronic exposures to $PM_{2.5}$ and ozone.

A section that describes public health outcome indicators in our analyses follows. These include total life expectancy at birth, life expectancy for men and women, and, temporary life expectancy in children and adults.

Additionally, we incorporate socioeconomic position indicators from official sources (*Consejo Nacional de Población*, CONAPO), that contain measures for level of education, household specific conditions –having sewer and toilet, electricity, running water, overcrowding, soil floor--, and income.

Two groups of causes of death are important variables in our work and are also characterized in the next sections.[8] The first group consists of the five causes of death that have been determined to be causally associated with long-term air pollution exposures: ischemic heart disease, cerebrovascular stroke (hemorrhagic and ischemic), chronic obstructive pulmonary disease (COPD), and lung cancer, in adults, and acute lower respiratory infections (ALRI) in children. COPD and lung cancer are also used as proxy variables to control for smoking prevalence (See Smoking Related Diseases) in models in which outcome variables are life expectancy, temporary life expectancy or years of life lost for each of the five specific causes of death that have been associated with air pollution exposures.

The second group of causes of death refers to reference diseases --diabetes, hypertension, colon cancer, stomach cancer and external causes (assault). These causes have not been linked or have been weakly linked with air pollution exposures. However, they are likely related to life expectancy or years of life lost and serve as proxy variables for risk factors not directly accounted for, such as community or individual behavioral factors.

[8] The Secretary of Health of the government of Mexico City (SEDESA) provided the mortality data of Mexico City that were used for this project. SEDESA, in turn, receives the data from the annual registries of INEGI (*Instituto Nacional de Estadística, Geografía e Informática*). Death certificates record death causes based on the codes of the International Classification of Diseases (ICD) --published by the World Health Organization which stipulates that Member States use the most current ICD revision for mortality and morbidity statistics. In Mexico the ICD ninth edition (ICD-9) were used until 1997, and ICD-10 codes since 1998.

AIR POLLUTION

Fine Particles and Ozone in CDMX, 1990-2015

Air quality has significantly improved in Mexico City. Systematic monitoring of air pollutants has been in place for several decades in the Metropolitan Area, so such improvements have been documented. The data from monitoring sites were provided by SEDEMA to construct the exposure indicators for this study.

Exposure indicators were built in agreement with those from the cohort studies that have yielded the most robust relative risk estimates for the relationship between chronic exposure to $PM_{2.5}$ and ozone and mortality. The exposure metrics are annual average $PM_{2.5}$ concentrations, and seasonal (six month)[9] daily 1-hour maximum ozone levels. These exposure metrics were first calculated using data from fixed-site monitoring stations and then were spatially interpolated to each *alcaldía*, to match the spatial resolution of official mortality data –to be used for the epidemiological analyses.

Air pollutants have been routinely monitored by environmental authorities, $PM_{2.5}$ since the end of 2003 and ozone before 1990. During our work for Phase II, fine particle concentrations were estimated for 1990 to 2003, which include the earlier years of our current analysis (1990, 1995, 2000), by means of a Generalized Additive Model (GAM). The GAM included predictive variables such as PM_{10} (PM_{10} has been routinely monitored since the late 1980s), meteorological variables (wind speed, relative humidity and temperature), monitoring station, year and month (as a proxy for season).[10] We used $PM_{2.5}$ and PM_{10} data from the five fixed-site monitoring stations that have consistently used High Volume Air Samplers (Federal Reference Method). To compute our ozone exposure metric all available daily data from fixed-site monitoring stations were used. The number of monitoring sites with available data varied yearly, starting with less than six sites in the early nineties, then increasing to over a dozen, and finally exceeding 20 since 2012.

Fine particle and ozone exposure metrics (average values and their corresponding standard error of the mean) estimated for each monitoring station were interpolated spatially to the *alcaldía* level. The analysis was first conducted at the basic geostatistical area (known as an AGEB) within an *alcaldía*. The values assigned to each AGEB were determined by how close the AGEB was to one or more of the $PM_{2.5}$ or ozone monitoring sites. More specifically, the methods applied were Proximity Analysis (nearest monitor) and Inverse Distance Weighting (IDW), using weights proportional to $1/distance^2$. For $PM_{2.5}$ we restrained the

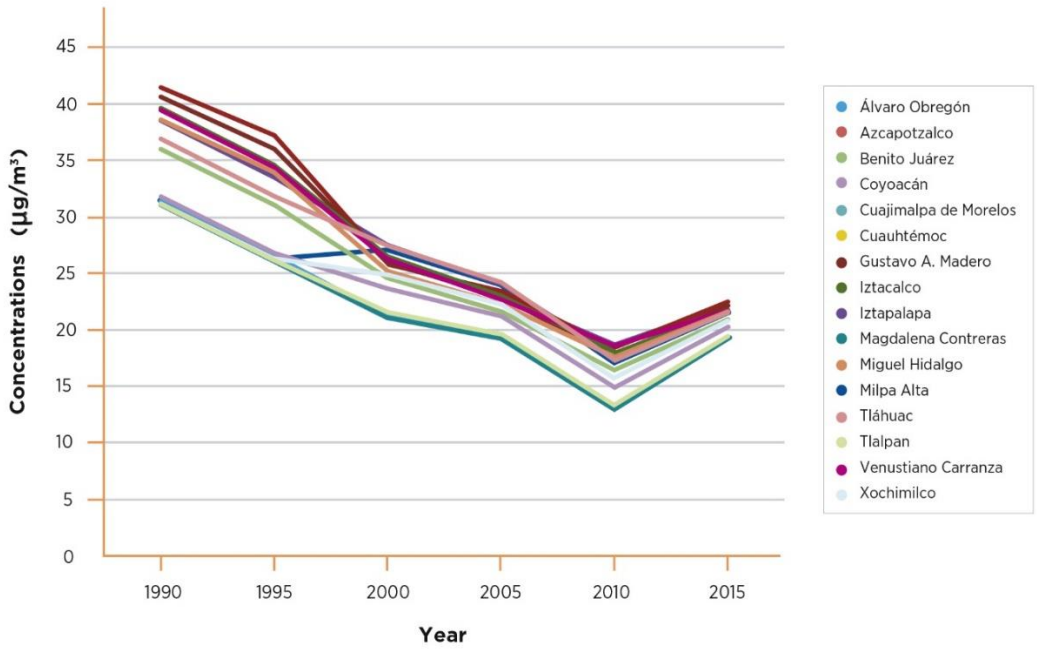
[9] "Seasonal" is defined as closely as possible to the definition used in the ACS cohort study. For this project "seasonal" comprises the period between February 1st and July 31st, to include the highest ozone months (March-June), adding one month before and one after, and exclude the months that have had historically the lowest ozone concentrations (September-December). This is in line with the definition of the "ozone season" by the Environmental authorities from Mexico City, which runs from the second week in February through June, when the rainy season begins. For reference, the ACS study defined "seasonal" as the warmer months that tend to have higher ozone concentrations (April-September) (Jerrett et al., 2009).

[10] The adjusted R-square of the GAM, used to predict $PM_{2.5}$ 24-hour concentrations, was equal to 0.73. Available data allowed to predict 6761 24-h $PM_{2.5}$ concentrations (equivalent to an average of 54 daily concentrations per station on an annual basis).

area of influence of the Xalostoc monitoring site on surrounding *alcaldías*, since it has a micro-scale spatial representativeness (according to the definition of the US-EPA) which implies that the very high concentrations monitored at this station are impacted by local sources and are unlikely to represent the exposure of populations that are not in the immediate vicinity. For that purpose, Xalostoc $PM_{2.5}$ concentrations were estimated using weights proportional to 1/distance^3.

Air quality has substantially improved in the past 25 years in Mexico City. In the early 1990s annual average $PM_{2.5}$ concentrations exceeded 40 µg/m^3 in some *alcaldías*, and the city-wide average exceeded 35 µg/m^3. By 2015 significant reductions were observed as levels in all *alcaldías* were below 23 µg/m^3 and the average for the city equaled 21 µg/m^3 (Figure 1). Interestingly, in 2010 concentrations reached their lowest levels in the City, possibly related with unusually heavy rains that likely washed out ambient particles during months with typically high particle concentrations (January and February).[11]

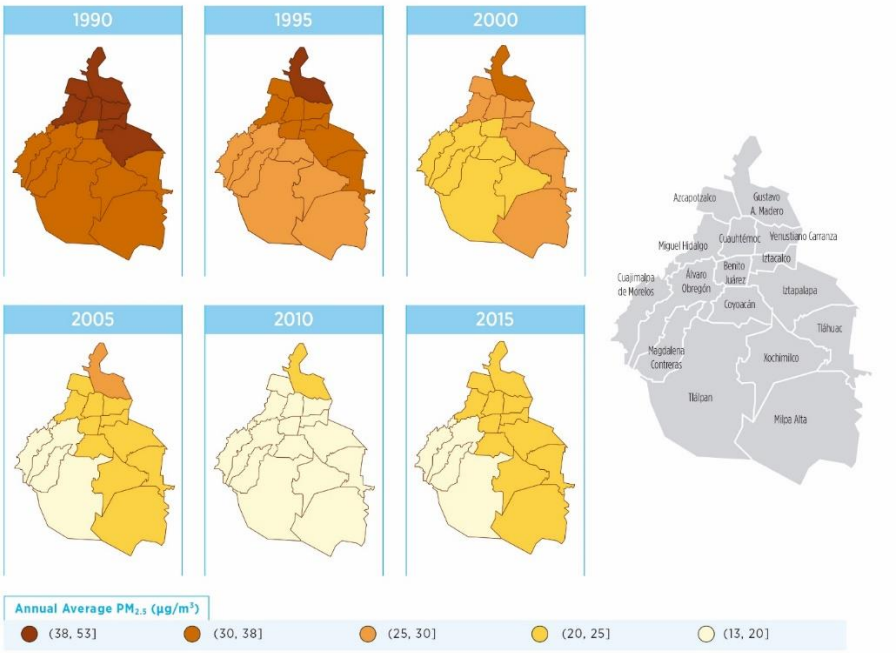
Figure 1. Time trends of alcaldía*-specific annual average $PM_{2.5}$ concentrations (µg/m^3)*



The figure below maps the spatial distribution of *alcaldía*-specific annual mean $PM_{2.5}$ concentrations by year.

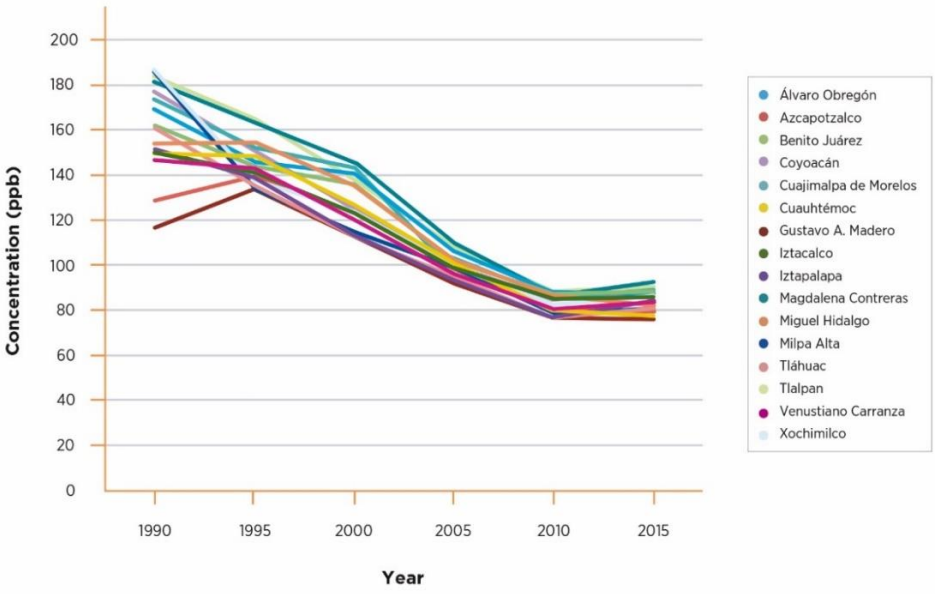
[11] In 2010 average annual rainfall was higher than for 2005 and 2015 (697.9 mm vs. 645.3 and 491.8, respectively). Rainfall in January and February reached close to 26 and 57 mm, respectively, in contrast with less than 6 and 5 mm registered for those two months in 2005 and 2015 (http://smn.cna.gob.mx/es/climatologia/temperaturas-y-lluvias/resumenes-mensuales-de-temperaturas-y-lluvias).

Figure 2. Spatial distribution of alcaldía*-specific annual average $PM_{2.5}$ concentrations ($\mu g/m^3$) by year*



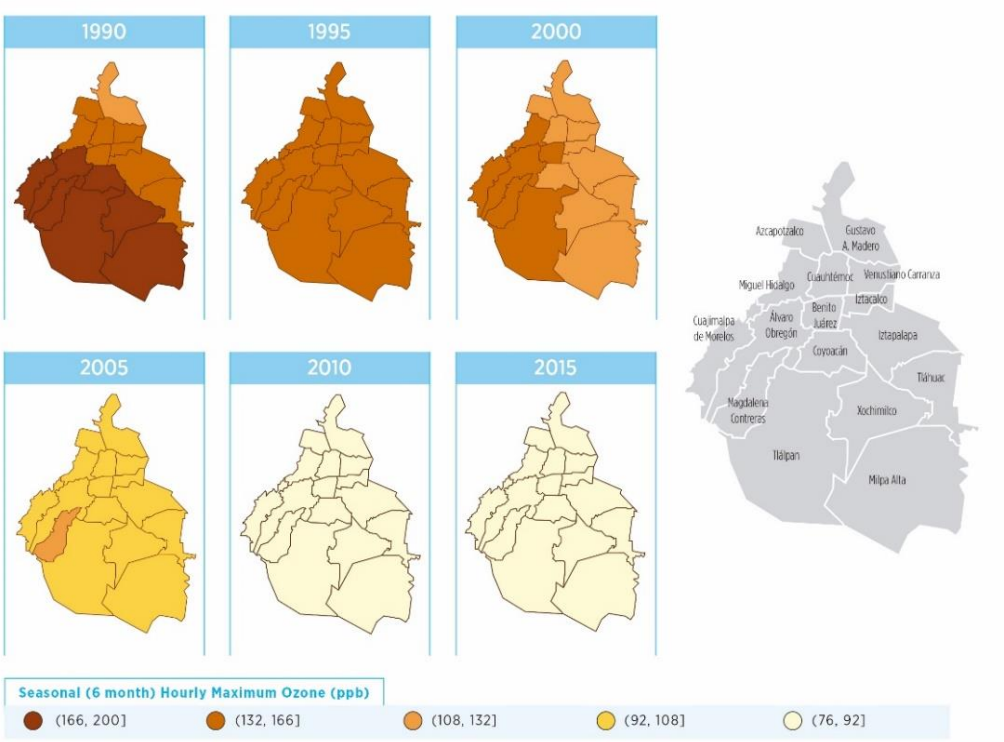
Ozone concentrations have declined significantly in Mexico City as well. In the 1990s estimated seasonal (six month) 1-hour maximum levels could range between 117 and over 185 ppb in the *alcaldías* of Mexico City. By 2010 the ozone scenario had improved; the steady decline in ozone concentrations through the City led to 2015 mean seasonal 1-hour maximum levels of 84 ppb, and values below 91 ppb prevailing in all City *alcaldías* (Figure 3).

Figure 3. Time trends of alcaldía*-specific seasonal (6 month) 1-hour maximum ozone concentrations (ppb)*



The figure below maps the spatial distribution of *alcaldía*-specific seasonal (six month) 1-hour maximum ozone concentrations by year.

Figure 4. Spatial distribution of alcaldía-*specific seasonal (6 month) 1-hour maximum ozone concentrations (ppb) by year*



LIFE EXPECTANCY

Life expectancy is a statistical measure of the average time someone is expected to live, based on the year of their birth, current age and other demographic factors including their sex. Life expectancy is an indicator of population health, and therefore it has been used to evaluate the efficiency of public policies. Although multiple factors may affect life expectancy, it has been shown that improvements in air quality may contribute to an increase in life expectancy (Pope et al., 2009).

In these analyses we first consider life expectancy at birth as an overall indicator of population health, and examine how this indicator changes with year, *alcaldía*, air quality, socioeconomic position indicators, among other risk factors.

The Phase II of this project examined health effects associated with air pollution exposures for adults (25 years and older), and for children (0 to 4 years old). For this epidemiological analysis, we calculated the temporary life expectancy for these two age ranges. Temporary life expectancy (25-74 years) is the statistical estimate of the average number of years a person is expected to live from age 25 to age 74. Likewise, temporary life expectancy (0-4 years) is the estimated average number of years of life in the 5 years starting at age 0 (birth).

Method to Estimate Life Expectancy and Life Years Lost

Total Life Expectancy: Total Life Expectancy (or life expectancy at birth) was estimated following the methods of simple decrements, as described by Preston et al., 2001. (Appendix I includes a thorough explanation.)

Inputs to estimate Total Life Expectancy are population stratified in five-year age groups and total number of deaths. First, specific mortality rates are computed for each five-year age group; these rates are the basis to estimate the probability of death by quinquennium, which are used to calculate the values of actuarial life tables, which in turn serve to compute life expectancy at birth.

Life expectancy at birth indicates the summation of person-years lived from age 0 to the final year included in the life table, divided by the initial population of such hypothetical cohort (radix =100,000 inhabitants).

Temporary Life Expectancy: Temporary Life Expectancy was calculated based upon the formula described by Arriaga (1984). Actuarial life table values of person-years lived between 0 to 4 or 25 to 74 are divided by the total number of survivors at age 0 or survivors at age 25, respectively, to compute temporary Life Expectancy from 0 to 4 years old or from 25 to 74 years old. (See Appendix I.)

Years of Life Lost: Life Years Lost were estimated for total deaths and for specific causes of death causally associated with air pollution exposures, using the method of multiple decrements described by Preston et al. (2001) (See Appendix I.)

The rates for specific causes of death were estimated, and then the associated probabilities of death were computed to estimate the life table using multiple decrements. A second step involves using the discrete approximation to Andersen and colleagues' method (2013), which utilizes the proportion of deaths for each specific cause to distribute potential total life years lost among such causes of death.

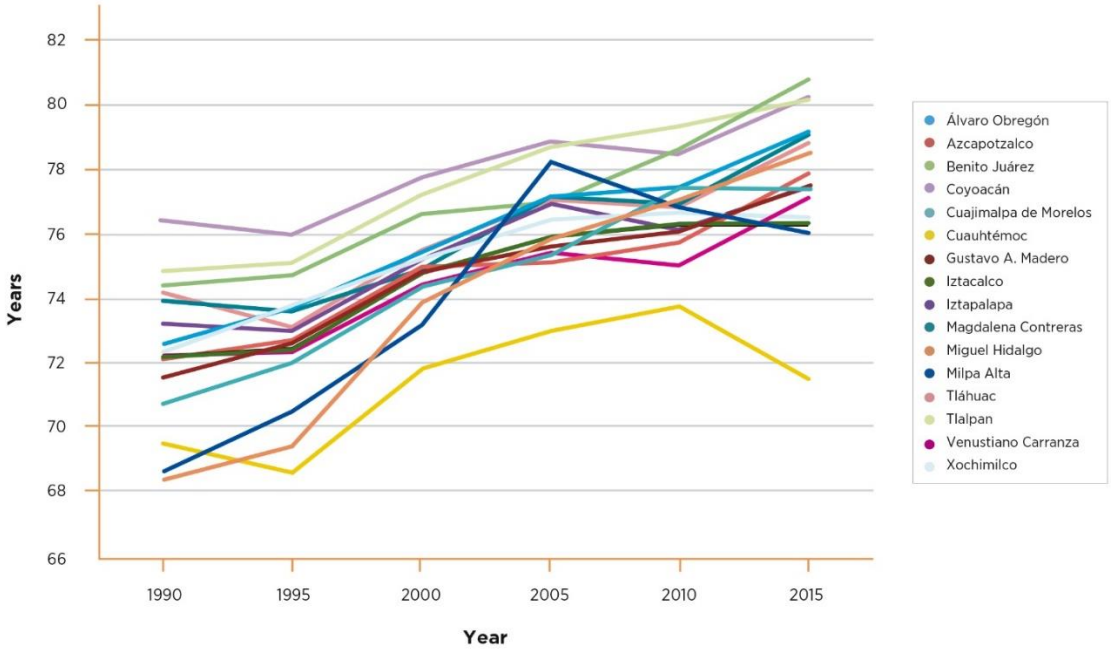
Potential life years lost result from the difference between temporary life expectancy at a certain age-group and the total number of years within the age-group range. The calculation involves accounting for mortality within the age-group range, and the hypothetical contribution of individuals who died before reaching the age of the upper limit of the age-group range.

Total Life Expectancy

In Mexico City, life expectancy at birth for total population increased by 7.7%, going from slightly over 72 years in 1990 to almost 78 years in 2015. Males and females presented a similar relative increase from 1990 to 2015, with an increment in men from 69.0 to 74.7 years, and in women from 75.2 to 80.5 years.

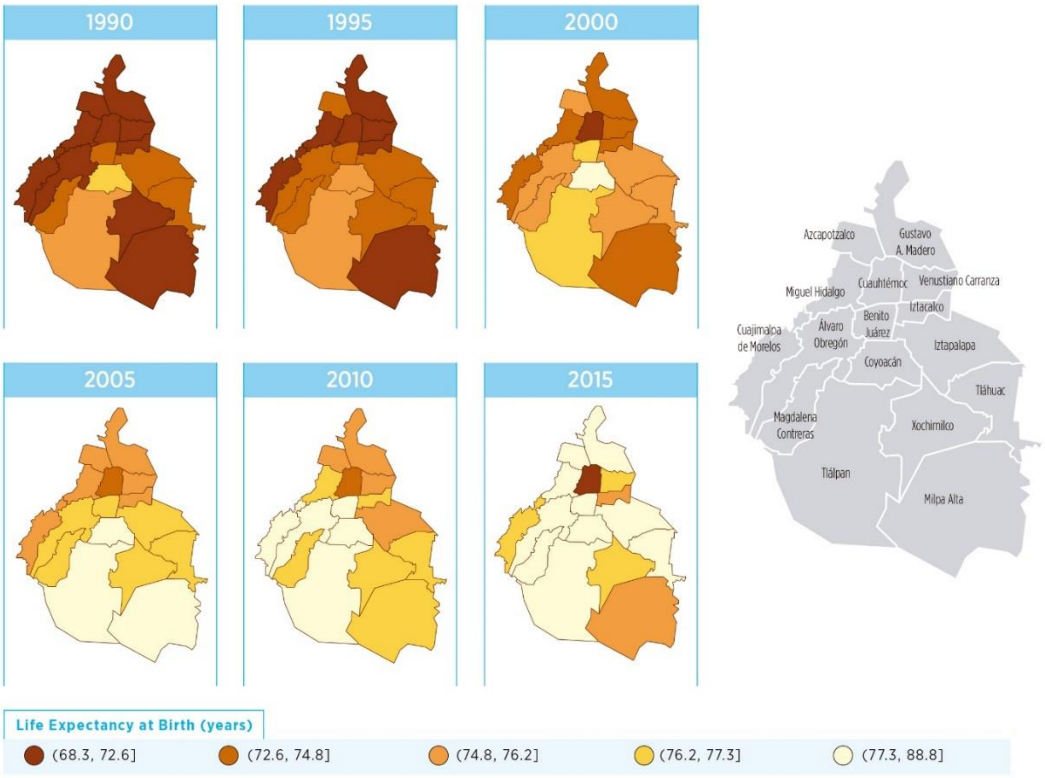
The following figure shows the life expectancy of the total population (male and female) for each *alcaldía* as a function of time. See Appendix III for life expectancy by sex *alcaldía*-specific time trends.

Figure 5. Time trends of alcaldía*-specific life expectancy at birth (years)*



Depicted below are maps with the spatial distribution of *alcaldía*–specific life expectancy for each of the 5 years between 1990 and 2015. See Appendix II for maps with *alcaldía*–specific life expectancy by sex spatial distribution.

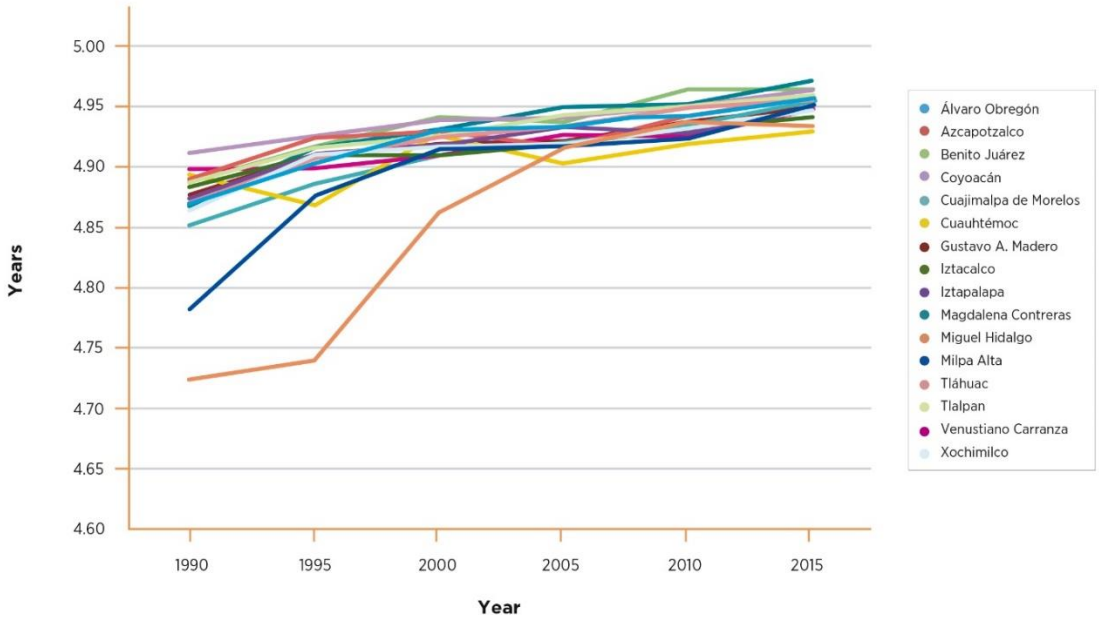
Figure 6. Spatial distribution of alcaldía*-specific life expectancy at birth (years) by year*



Temporary Life Expectancy

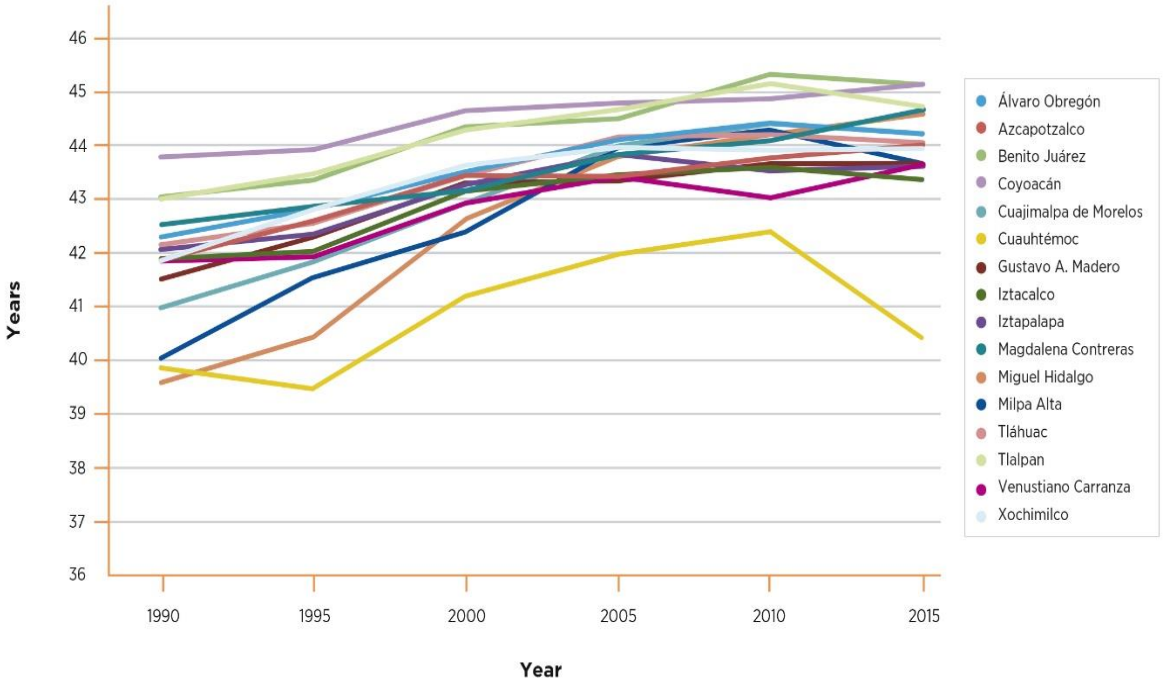
Temporary life expectancy for children 0-4 years was 4.87 in 1990, compared to a total possible of 5 years, and increased to 4.95 years in 2015. The following figure shows the *alcaldía*-specific temporary life expectancy for these children as a function of time across the study period (1990 to 2015). The earlier years presented temporary life expectancy variability between *alcaldías* but towards the end of the study period this indicator became very homogeneous between *alcaldías*. Maps with the spatial distribution of *alcaldía*–specific temporary life expectancy (0-4 year) for each of the 5 years between 1990 and 2015 are presented in Appendix III.

Figure 7. Time trends of alcaldía-*specific temporary life expectancy (years) for population between 0 to 4 years old*



For 25 to 74-year-old adults, the temporary life expectancy in 1990 was 41.8 years, compared to a total possible of 50 years between ages 25 and 74 years. In 2015 this indicator had increased to 43.8 years, that is by 4.9%. The following figure show the *alcaldía*-specific temporary life expectancy for these adults as a function of time across the study period (1990 to 2015). Most *alcaldías* presented an increasing and steady increase, except for Cuauhtémoc, where a steep decrease is shown in 2010. Maps of the spatial distribution of *alcaldía* –specific temporary life expectancy (25-74 year) for each of the 5 years between 1990 and 2015 are presented in Appendix III.

Figure 8 Time trends of alcaldía-*specific temporary life expectancy (years) for population between 25 to 74 years old*



SMOKING RELATED DISEASES

Two national surveys have collected information on smoking habits in Mexico: The National Health Survey (ENSA 2000), which later became the National Health and Nutrition Survey (ENSANUT 2006 y 2012), and the National Addictions Survey (ENA 2011 y 2016). (See Appendix IV for details on surveys description.)

Data from the surveys was analyzed to try to use smoking prevalence at *alcaldía* level and for as many years available as possible. However, the two surveys differ in terms of their methodology, sampling schemes, geographic representativeness, survey specific reagents and frequency of application. Some of these differences hinder making comparisons of smoking prevalence to assess trends over time. In addition, these surveys have no representativeness at the municipal level (or *alcaldía* for Mexico City), which became evident as unstable prevalence rates were observed when Mexico City numbers were disaggregated by *alcaldía*.[12]

Among the factors that hinder the comparativeness among the results of these surveys is the working definition of a smoker. In the survey ENA, the definition of a smoker is that of a person who has smoked in the past month (Martínez et al., 2010). In both ENSA y ENSANUT, a smoker is a person who has smoked at least 100 tobacco cigarettes throughout his/her life and who currently smokes (Guerrero-López et al., 2013). Clearly, these definitions refer to different things, with the first one referring likely to an occasional smoker, whereas the second, to a habitual smoker.

Regarding the possibility of using the data at *alcaldía* level, we estimated smoking prevalence within the *alcaldías* of Mexico City. The numbers were unstable due to the small or null sample size of individuals within some of the *alcaldías*. In ENSA 2000 and ENSANUT 2006 there were some *alcaldías* with no participants, and for other *alcaldías* the sample size was of ≤ 30 subjects. For ENSANUT 2012, the smaller sample size was of 11 individuals, and several *alcaldías* had samples of ≤ 30 subjects. Appendix IV includes official smoking prevalence --and sample size-- for Mexico City, stratified by age-group and sex.

Figures 9 and 10 show the magnitude of smoking prevalence in Mexico City and in the country, and the trends for the past 10 years. As shown in Figure 9, Mexico City has higher smoking rates than the country-wide rates. This difference remains when we stratify by sex, with the rate in the City being 1.5 to 2 times larger than nationally. Time trends show that since 2012 the smoking prevalence in the country decreased, but an inverse rate is shown for the city. The same difference in the pattern for the country vs. the city prevails for men. For women, there's a similar (and stable) trend in Mexico and in Mexico City.

[12] Only the ENA, 2011 survey was designed to have representativeness at the *alcaldía* level. However, data were not available to us.

Figure 9. Smoking prevalence (%) in adults aged 25 to 64 years in Mexico and in Mexico City for 2006, 2012 and 2016

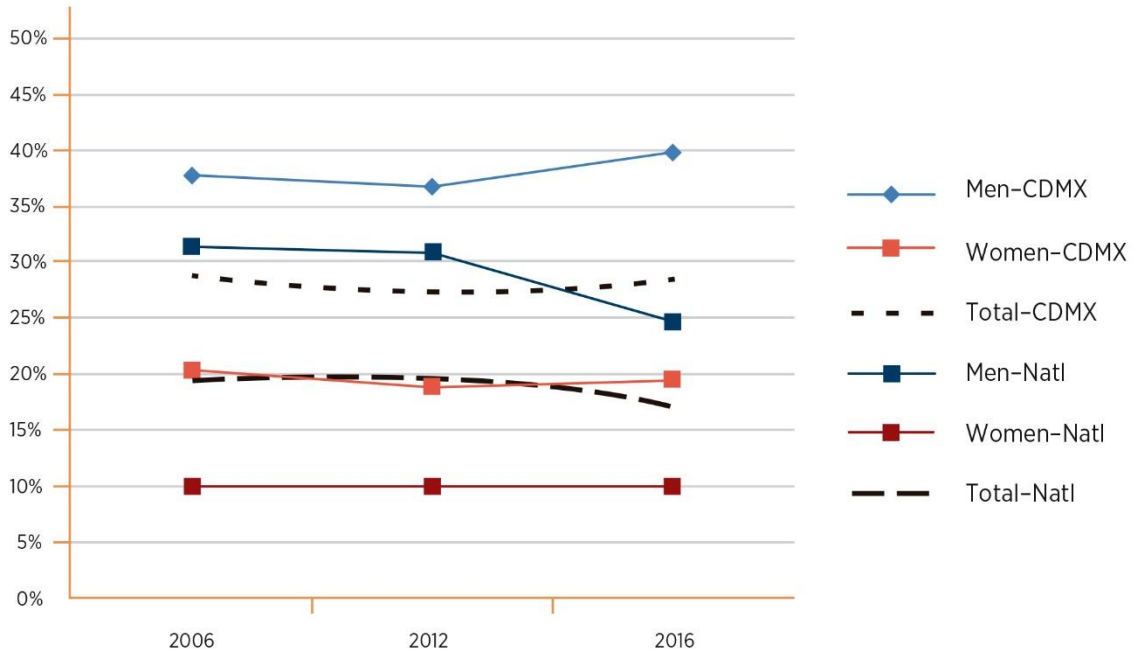
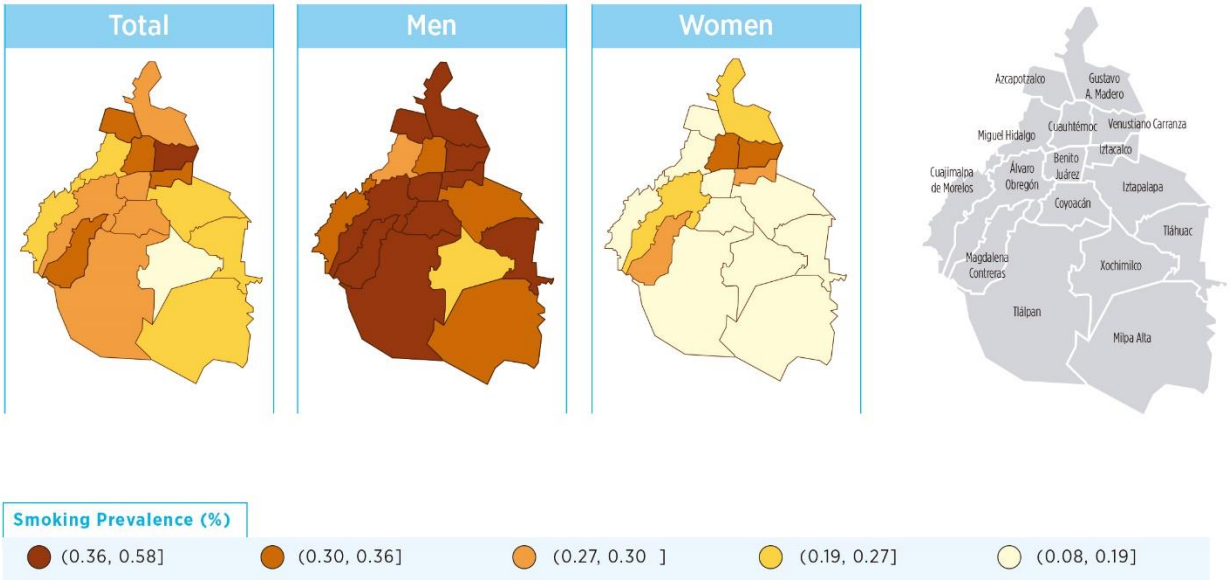


Figure 10. Spatial distribution of alcaldía*-specific smoking prevalence (%) in adults aged 25 to 64 years and stratified by sex (Aggregated 2000, 2006 and 2012 data)*

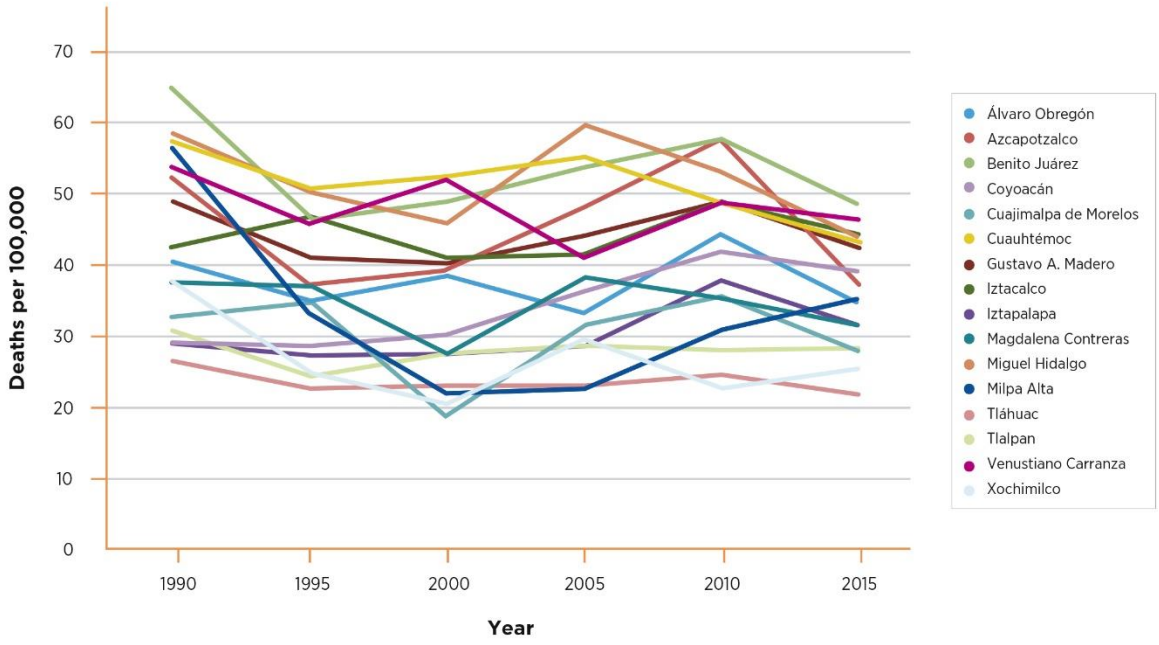


Due to smoking prevalence *alcaldía*-specific data constraints, our analyses used *alcaldía*-specific death rates for COPD and lung cancer as proxy indicators of accumulated exposure to smoking. A brief description of time trends and spatial distribution of these two causes of death follows.

Chronic Obstructive Lung Disease (COPD)

The COPD mortality rate in Mexico City dropped by 16%, from 43.6 per 100,000 in 1990 to 36.4 in 2015. There is weak evidence of improvements in the *alcaldía*-specific rates of COPD mortality (Figure 11). Maps of the changes in the spatial distribution of COPD mortality rates are found in Appendix IV.

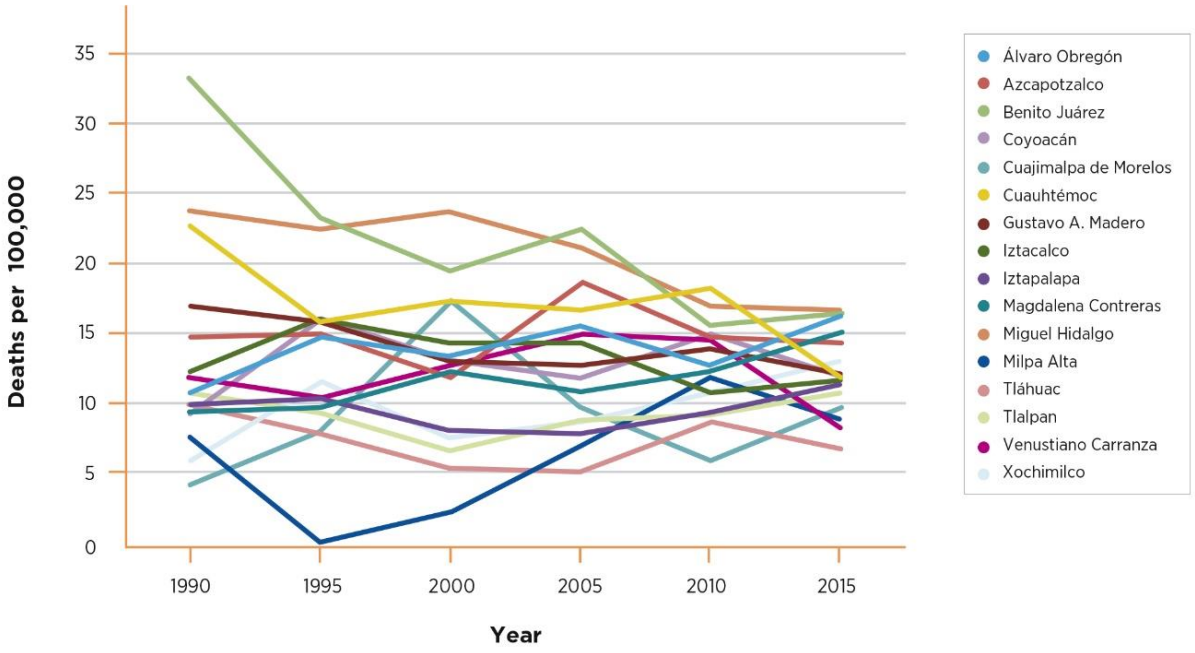
Figure 11. Time trends of alcaldía*-specific chronic obstructive pulmonary disease mortality rates (deaths per 100,000)*



Lung Cancer

Lung cancer mortality includes trachea, bronchus and lung cancers. The lung cancer mortality rate in Mexico City shows a very slight drop of approximately 9%, going from 13.3 deaths per 100,000 in 1990 to 12.2 deaths per 100,000 inhabitants in 2015. There is evidence of improvements in the *alcaldía*-specific rates of lung cancer mortality in Benito Juárez, Cuauhtémoc, and Miguel Hidalgo (Figure 12). Maps of the changes in the spatial distribution of lung cancer mortality rates are found in Appendix IV.

Figure 12. Time trends of alcaldía*-specific lung cancer mortality rates (deaths per 100,000)*



SOCIOECONOMIC POSITION INDICATORS

The relationship between air pollution and health can be affected by risk factors that include socioeconomic position indicators. CONAPO collects information from the General Census of Population and Households (1990, 2000 and 2010) and from the Counts of Population and Households (1995, 2005 y 2015) and constructs indicators that include level of education, income, and household characteristics. These indicators --life expectancy and years of life lost-- were used for this project to assess the influence of such *alcaldía*-specific population characteristics on *alcaldía*-specific health outcomes (Table 1).

Table 1. Socioeconomic Position Indicators available from census and intercensal count data, 1990-2015

Indicators	Variable Code	Available Information					
		1990	1995	2000	2005	2010	2015
1. Illiteracy Illiterate population ≥ 15 years old (%)	ANALF						
2. Low Education Population ≥ 15 years old without complete primary school (%)	SPRIM		+				
3. No Sewer nor Toilet‡ Occupants in households without sewer nor toilet (%)	OVSDE						
4. No Electricity Occupants in households without electricity (%)	OVSEE						
5. No Running Water Occupants in households without running water (%)	OVSAE						
6. Overcrowding Households with some degree of overcrowding (%)	VHAC		+				
7. Soil Floor Occupants in households with soil floor (%)	OVPT		+				
8. Small Villages Population living in locations of <5000 inhabitants (%)	PL<5000						
9. Low Income Population with a job of up to 2 minimum wages	PO2SM		+				
10. Marginalization Index*							

+ For 1995 there are missing values at *alcaldía* level, but available for CDMX.

‡ National census and national intercensal counts collected information for this indicator using three different codes (OVSDE, OVSDSE and OVSD). Two codes that refer to not having sewer nor toilet were used in all years except for 1995, when the code referred solely to not having sewer was used instead. CONAPO considers that changes in the format used to collect information did not affect importantly the definition of the indicator (Romo Viramontes, 2018). However, not having sewer nor toilet is likely to be a more stringent indicator than the one related only with not having sewer.

* The Marginalization Index for each Census or Intercensal Count is constructed using Principal Components, so should not be used to assess time trends

Source: CONAPO, 2018.

The indicators listed on Table 1 are used by CONAPO to construct Marginalization Indices since 1990, which are available at state and *alcaldía* levels. Marginalization Indices are built using Principal Components, so they are not comparable across time and can't be used to assess time trends. This is why for our analyses we use the indicators and not the index itself.

There has been substantial improvement in each of these Socioeconomic Position Indicators between 1990 and 2015. Table 2 below shows the overall fraction (%) of the sampled population for Mexico City reporting each of these socioeconomic position indicators.

Table 2. Behavior of Socioeconomic Position Indicators for Mexico City (%), 1990-2015

Socioeconomic Position Indicators	Illiteracy (ANALF)	Low Education (SPRIM)	No Sewer nor Toilet (OVSDE)	No Electricity (OVSEE)	No Running Water (OVSAE)	Overcrowding (VHAC)	Soil Floor (OVPT)	Small Villages (PL<5000)	Low Income (PO2SM)
1990	4.36	17.5	2.94	1.12	4.30	48.0	3.55	1.32	61.7
1995	3.11	12.0	2.56	0.11	2.87	46.7	1.11	1.29	47.4
2000	2.97	12.2	0.60	0.21	1.78	35.5	1.75	1.08	42.3
2005	2.61	9.6	0.19	0.16	2.04	29.8	1.40	1.53	32.7
2010	2.09	8.5	0.11	0.10	2.37	26.3	1.22	1.83	28.4
2015	1.48	6.5	0.05	0.06	1.70	19.6	0.58	1.83	28.0

Source: CONAPO, 2018.

The indicator *Percentage of population living in villages of <5000 inhabitants (PL<5000)* is excluded from the rest of the analyses because it conveys little information. In Mexico City close to 60% of the observations (54 out of 96) correspond to population living in villages with over 5,000 inhabitants; in nine out of 16 *alcaldías* there were no small villages during the six years under study.

Method to Estimate Missing Values in Socioeconomic Position Indicators

Data collected from the census and the intercensal counts allow to estimate all socioeconomic indicators presented on Table 1 for Mexico City for all years under study. At *alcaldía* level, most of the indicators are available, except for four indicators. The intercensal 1995 counts did not collect the necessary information to calculate *Percentage of population ≥15 years old w/out complete primary school (SPRIM)*, *Percentage of households with some degree of overcrowding (VHAC)*, *Percentage of occupants in households with*

ground floor (OVPT), and *Percentage of population with a job with a salary of up to 2 minimum wages (PO2SM)*.[13]

We developed a weighted linear regression for each variable to impute *alcaldía*-missing values, with the weights of the square root of the population ($\sqrt{población}$) of the corresponding *alcaldía*. The main predictor was the Mexico City value of the indicator for which no value was available at *alcaldía* level. Other explanatory variables were those for which we had 1995 *alcaldía* values: *Percentage of illiterate population ≥ 15 years old (ANALF)*, *Percentage of occupants in households without electricity (OVSEE)* and *Percentage of occupants in households without running water (OVSAE)*.[14] Finally, a categorical variable for *alcaldía* was included in the regression models.

Below is the general form of the regression equation, using as example the variable *Percentage of population with a job with a salary of up to 2 minimum wages (PO2SM)* (See Table 1 for variable coding):

$$PO2SM_{alcaldía} = a0 + a1*PO2SM_{CDMX} + a2*ANALF_{alcaldía} + a3*OVSEE_{alcaldía} + a4*OVSAE_{alcaldía} + a5*_{alcaldía}$$

Distribution of Socioeconomic Position Indicators

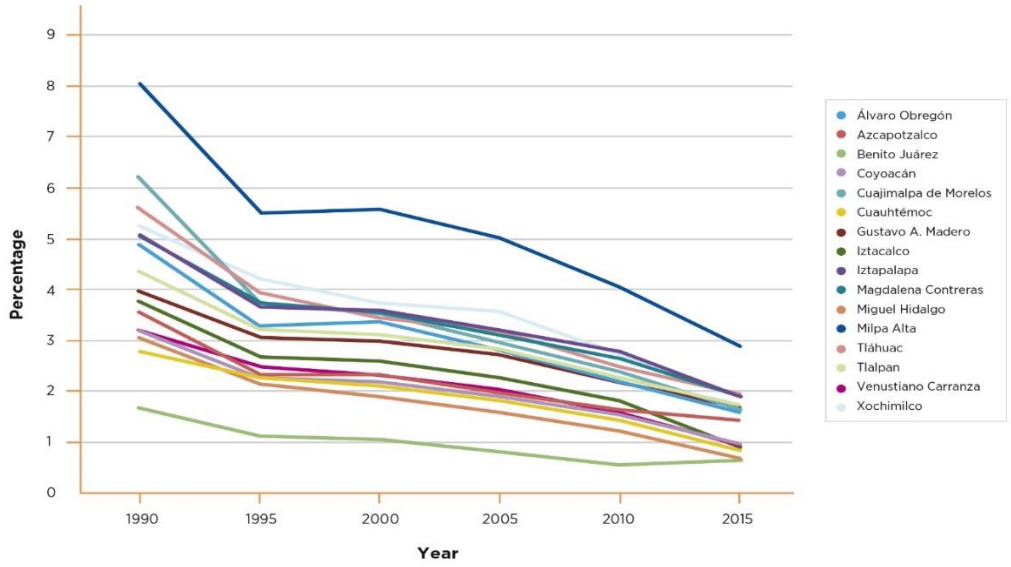
Time trends and spatial distribution by *alcaldía* of socioeconomic position indicators are presented in Appendix V. Illustrative socioeconomic position indicators with their corresponding time trends and spatial distribution are presented below.

Illiterate Population ≥ 15 years old (ANALF): Population census and population intercensal counts data for illiterate population were available for each of the study-period years (see Table 1 above). Overall the rate of illiteracy among those 15 years or older in Mexico City dropped from 4.36% in 1990 to 1.48% in 2015. The Figure below shows the trends over time of *alcaldía*-specific illiterate population.

[13] The Marginalization Index was computed for all years in spite the fact that information was not available to construct some of the indicators. CONAPO built the index for 1995 using 1990 Census figures for three indicators and excluded one of them (Romo Viramontes, 2018).

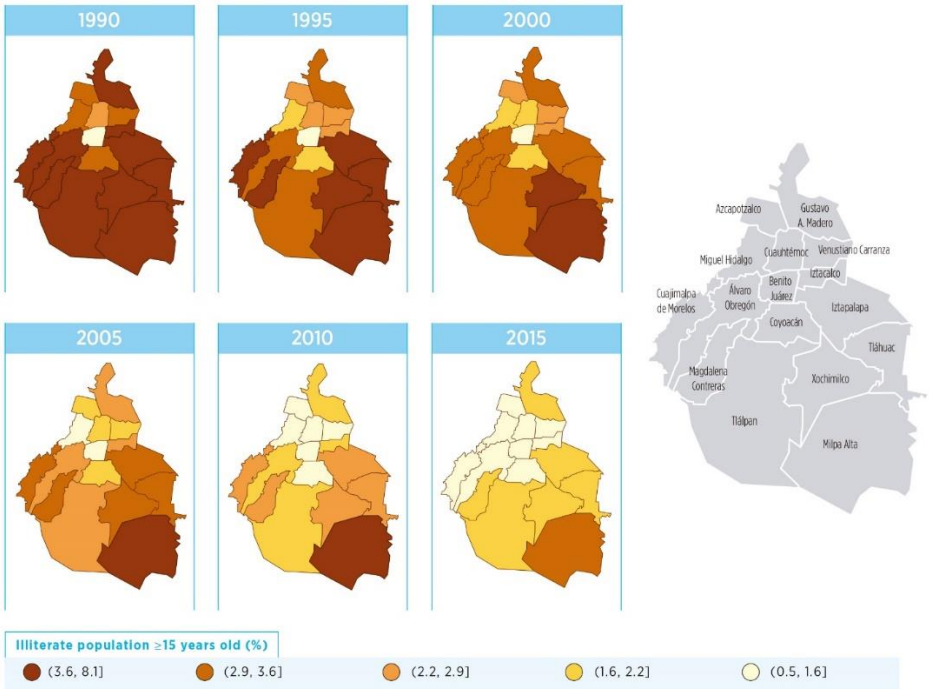
[14] *Percentage of occupants in households without sewer nor toilet (OVSDE)* was not included in the predictive model, since in 1995 information for such indicator was collected differently --*Percentage of occupants in households without sewer (OVSD)*.

Figure 13. Time trends of alcaldía*-specific illiterate population aged 15 years and above (%)*



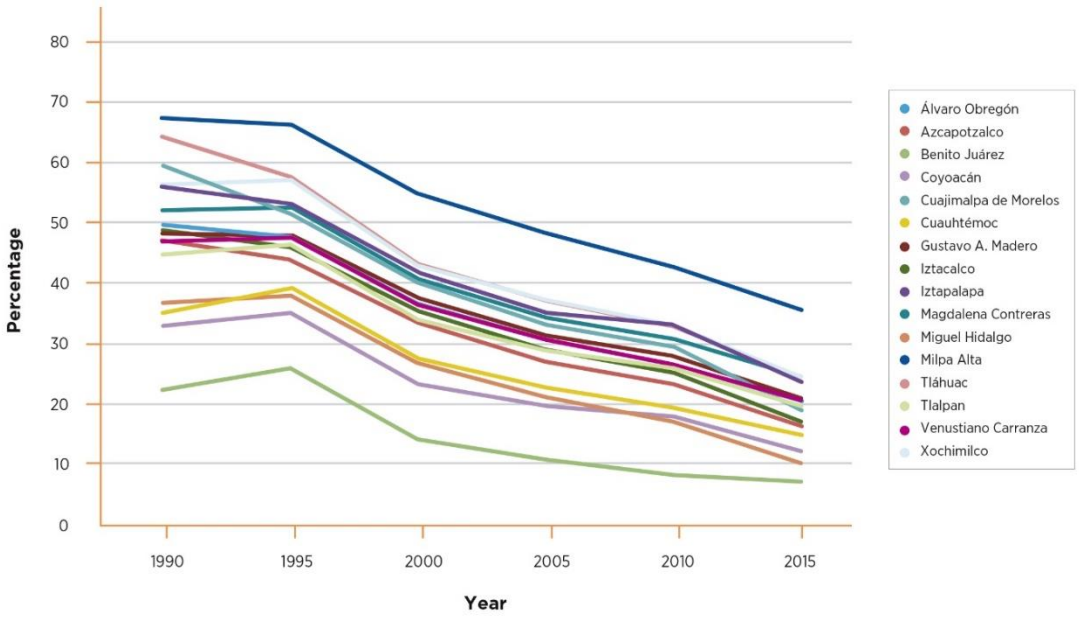
Spatial distribution of *alcaldía*-specific illiterate population ≥ 15 years old by year is mapped in the following figure. Illiteracy dropped in all *alcaldías* since 1990, with figures below 3% by 2015. Milpa Alta shows the highest and Benito Juárez the lowest numbers of illiterate population throughout the study period.

Figure 14. Spatial distribution of alcaldía*-specific illiterate population aged 15 years and above (%) by year*



Households with some degree of overcrowding (VHAC):[15] Overall the fraction of households with overcrowding in Mexico City dropped by over 50%, going from 48% in 1990 to almost 20% in 2015. The figure below shows the trends over time in *alcaldía*-specific fraction of households with overcrowding conditions.

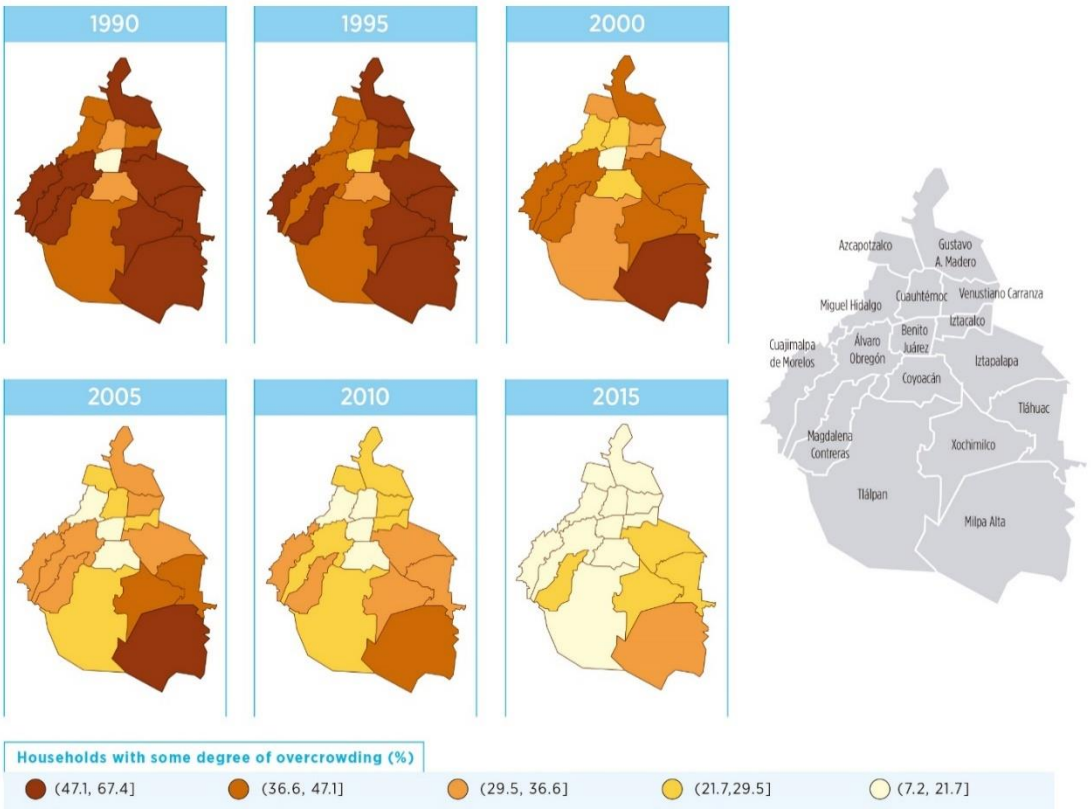
Figure 15. Time trends of alcaldía*-specific households with some degree of overcrowding (%)*



The spatial distribution of *alcaldía*-specific households with overcrowding by year is mapped in the following figure. For this indicator, Milpa Alta and Benito Juárez present the lowest and highest relative numbers of households with some degree of overcrowding for all six years.

[15] CONAPO defines households with some degree of overcrowding considering the relationship between the number of rooms or bedrooms and the number of inhabitants in the household. For example, households with one room-bedroom are considered with some degree of overcrowding if there are three or more inhabitants. For larger households, with four rooms-bedrooms are included in this category if there are nine or more inhabitants (CONAPO, 2013).

Figure 16. Spatial distribution of alcaldía*-specific households with some degree of overcrowding (%) by year*



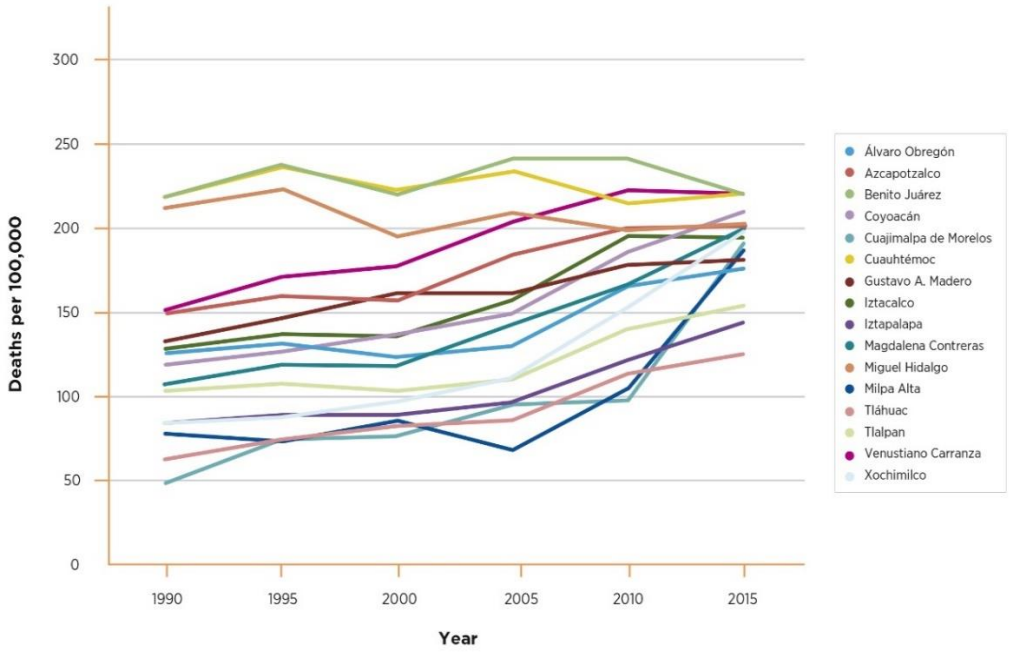
AIR POLLUTION RELATED DISEASES

Our work aims at measuring how the decline in $PM_{2.5}$ and ozone concentrations may improve life expectancy, temporary life expectancy and other health benefit indicators for specific diseases causally associated with air pollution. Based on Phase I of this project where we conducted a thorough literature review, we identified five specific causes of mortality that have been determined to be causally associated with air pollution exposures, more specifically with $PM_{2.5}$ or ozone, in the Global Burden of Disease analyses and used for our Phase II work. In adults these causes include ischemic heart disease, cerebrovascular stroke, COPD and lung cancer; in children we also identified ALRI. In the following section we describe time trends and spatial distribution of the *alcaldía*-specific mortality rates for three of such causes for the six study years –as COPD and lung cancer are very strongly associated with smoking, they are described in the section of Smoking Related Diseases. Maps with spatial distribution for these three *alcaldía*-specific causes of death by year can be found in Appendix VI.

Ischemic Heart Disease

Ischemic heart disease (IHD) is one of the most commonly reported causes of death in Mexico City. The rate of IHD death in Mexico City has increased by 50% in the past 25 years, going from 126 deaths per 100,000 in 1990 to 189 deaths per 100,000 in 2015. The figure below shows that this rising time trend is also seen in the *alcaldía*-specific IHD mortality rates. Two *alcaldías* stand aside due to higher rates since 1990: Benito Juárez and Cuauhtémoc. Benito Juárez also stood aside due to lower values in socioeconomic position indicators, such as low fraction of illiterate population and of occupants in household with soil floor.

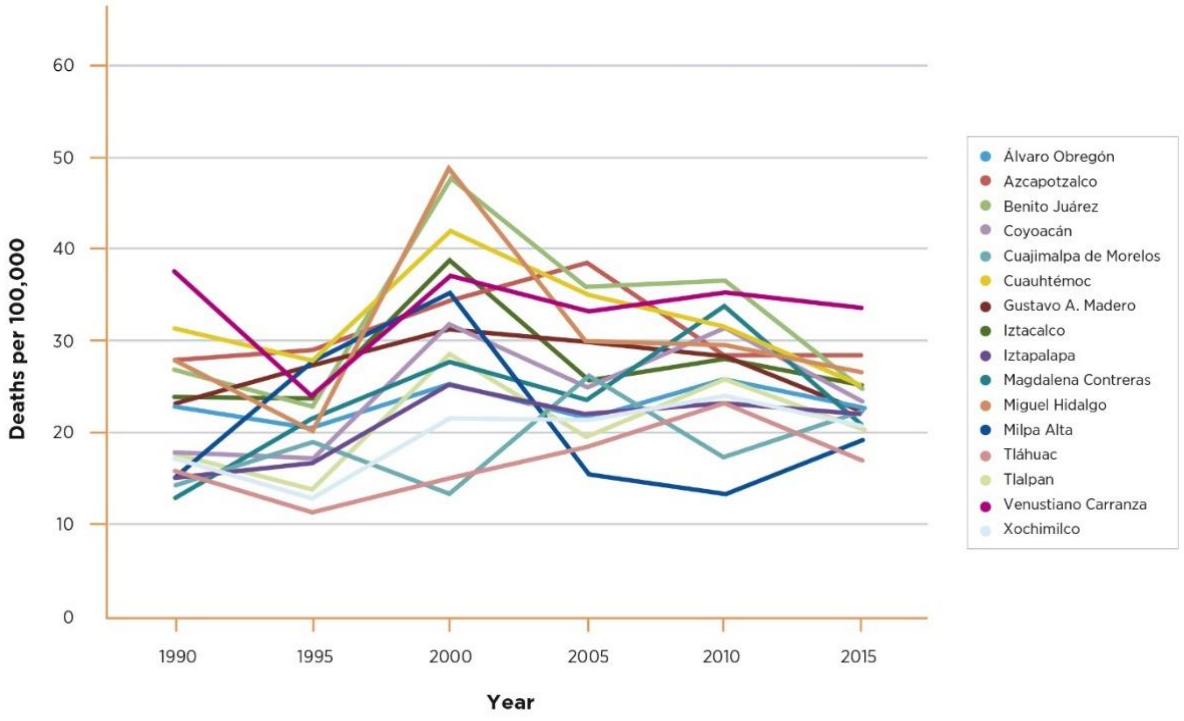
Figure 17. Time trends of alcaldía-*specific ischemic heart disease mortality rates (deaths per 100,000)*



Cerebrovascular Stroke

Cerebrovascular stroke mortality rates in Mexico City have changed little over the 25-year study period, with rates slightly above 20 deaths per 100,000 --21.7 per 100,000 in 1990 to 23.4 deaths per 100,000 in 2015. As the figure below shows, there's little evidence of change in time trends in the *alcaldía*-specific rates of stroke mortality. See Appendix VI for maps of the changes in the spatial distribution of stroke mortality rates.

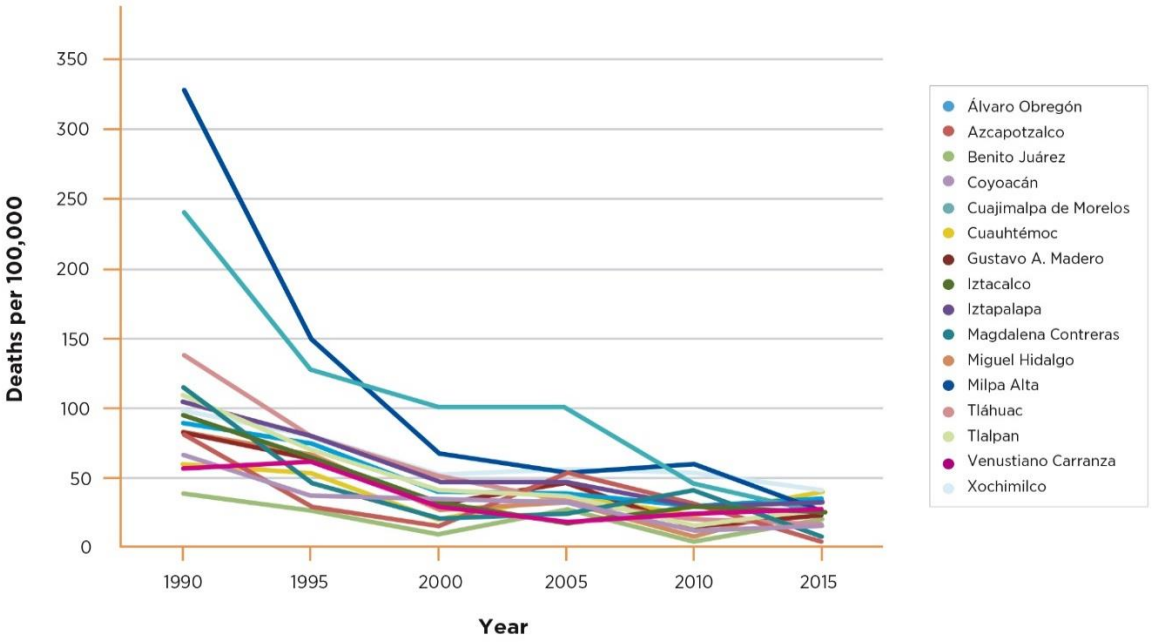
Figure 18. Time trends of alcaldía*-specific cerebrovascular stroke mortality rates (deaths per 100,000)*



Acute Lower Respiratory Infections (ALRI)

There have been dramatic improvements in ALRI mortality rates among children, with this rate showing a decrease of almost 80%. The ALRI rates in Mexico City dropped from 110.4 deaths per 100,000 in 1990 to 24.1 deaths per 100,000 in 2015. The figure below shows that the largest improvements have been in Milpa Alta and Cuajimalpa de Morelos, reaching rates that are like the rest of Mexico City's *alcaldías* by 2015. Maps of the *alcaldía*-specific ALRI mortality rates by year are found in Appendix VI.

Figure 19. Time trends of alcaldía*-specific acute lower respiratory illness mortality rates (deaths per 100,000) in children*



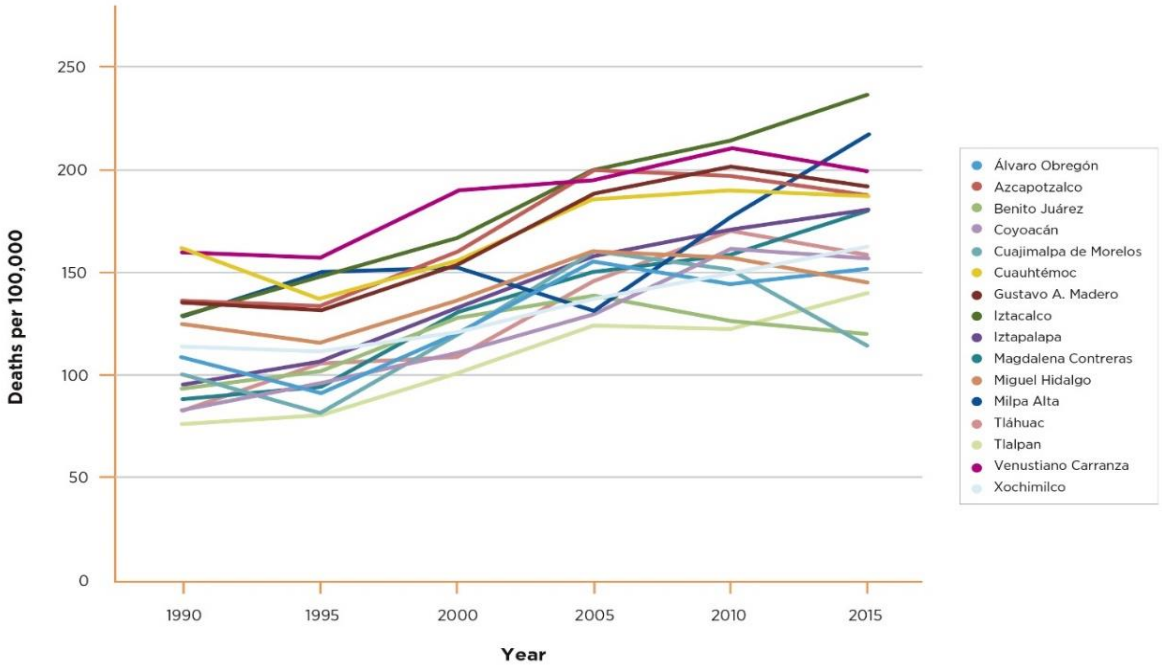
REFERENCE DISEASES

Changes in life expectancy or in years of life lost could be associated with a variety of risk factors not captured by the socioeconomic position indicators which we have compiled for the *alcaldías* over the time-period of interest. Such risk factors could include individual behavioral factors like obesity and nutrition, or institutional factors such as access to health care. To provide some insight into the influence of these unmeasured mortality risk factors, we will also include some common causes of death which would not be expected to be associated or would be weakly associated with air pollution exposures, these are: diabetes, hypertension, colon cancer, stomach cancer and external causes (assault). The next section presents a brief description of time trends of these reference causes of mortality, and the corresponding maps of the changes in the spatial distribution are shown in Appendix VII.

Diabetes

The diabetes mortality rate in Mexico City has increased significantly from 116.5 per 100,000 in 1990 to 172.0 in 2015. The *alcaldía*-specific rates of diabetes mortality are consistent in showing an increase from 1990 to 2015. Interestingly, some of the *alcaldías* started a reversal trend after 2005 or 2010 (Figure 20). Maps of the changes in the spatial distribution of diabetes mortality are found in Appendix VII.

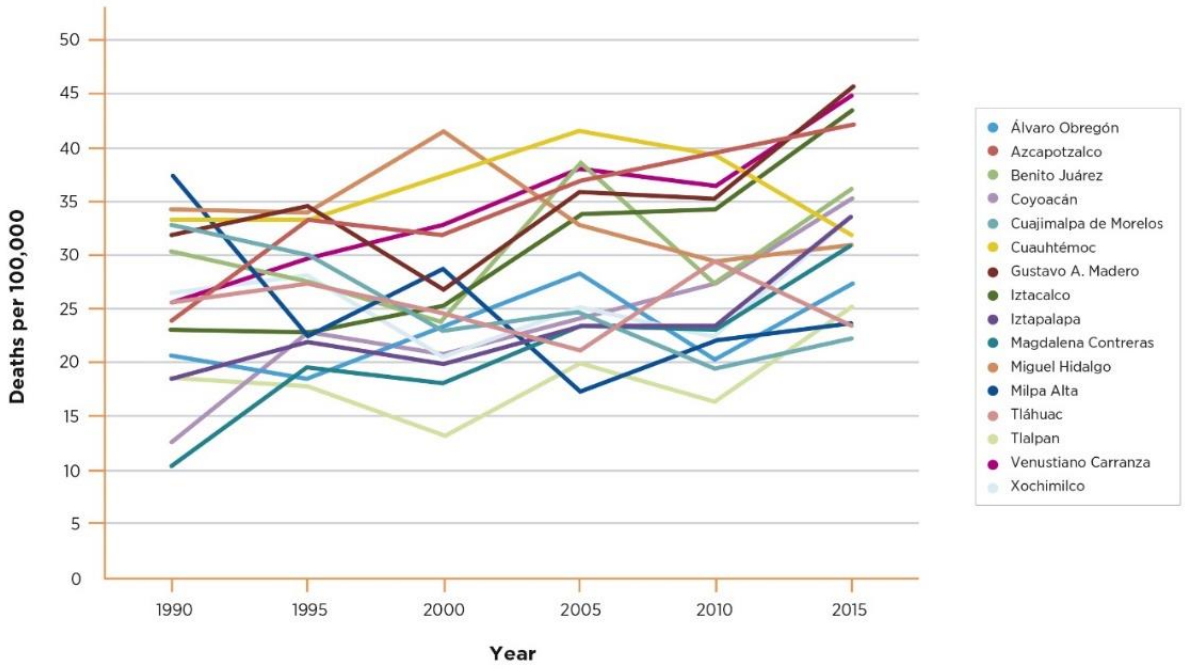
Figure 20. Time trends of alcaldía*-specific diabetes mortality rates (deaths per 100,000)*



Hypertension

The hypertension mortality rate in Mexico City has increased in more than 30%, going from 25.4 deaths per 100,000 in 1990 to 33.1 deaths per 100,000 in 2015. The *alcaldía*-specific rates of hypertension mortality are fairly unstable, but consistent with this increase over the 25-year period (Figure 21). Maps of the changes in the spatial distribution of hypertension mortality rates are found in Appendix VII.

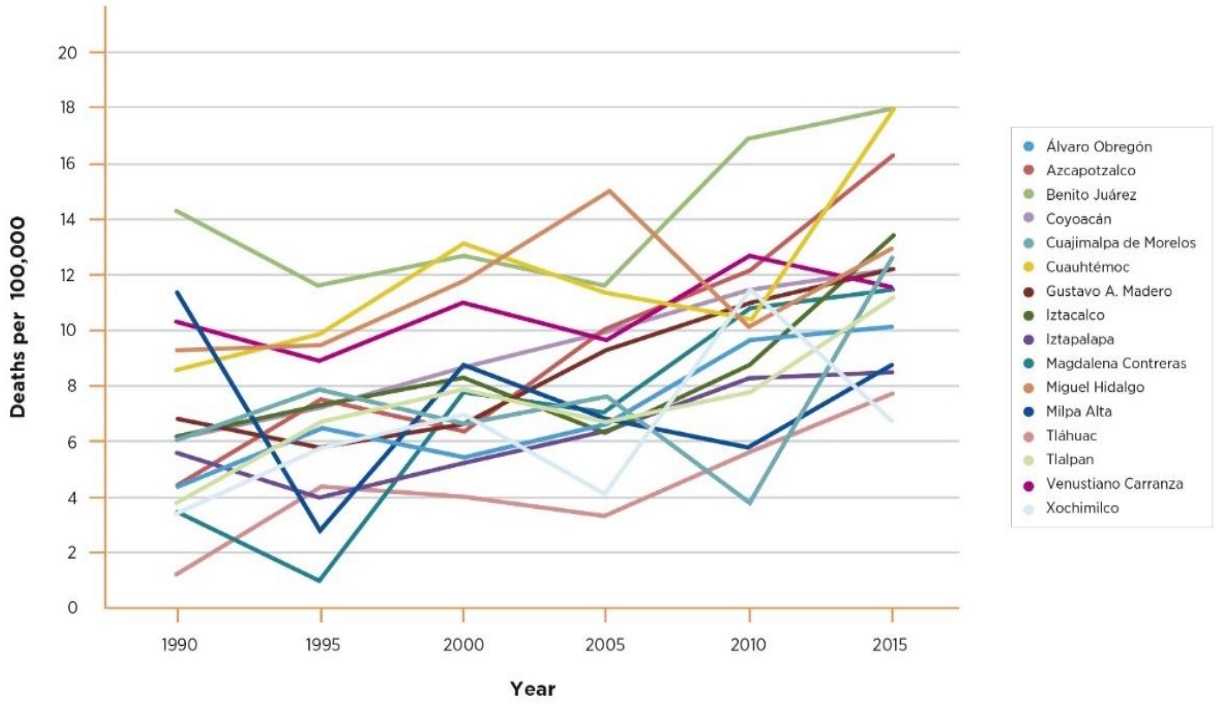
Figure 21. Time trends of alcaldía-*specific hypertension mortality rates (deaths per 100,000)*



Colon Cancer

The colon cancer mortality rate in Mexico City has augmented importantly, showing a more than 80% increase. Rates went from 6.6 to 12 deaths per 100,000 from 1990 to 2015. The *alcaldía*-specific rates of colon cancer mortality present a fairly unstable pattern but are consistent with this increase between 1990 and 2015 (Figure 22). Benito Juárez and Cuauhtémoc stand out with a more dramatic increase in the latter years. Maps of the changes in the spatial distribution of colon cancer mortality rates are found in Appendix VII.

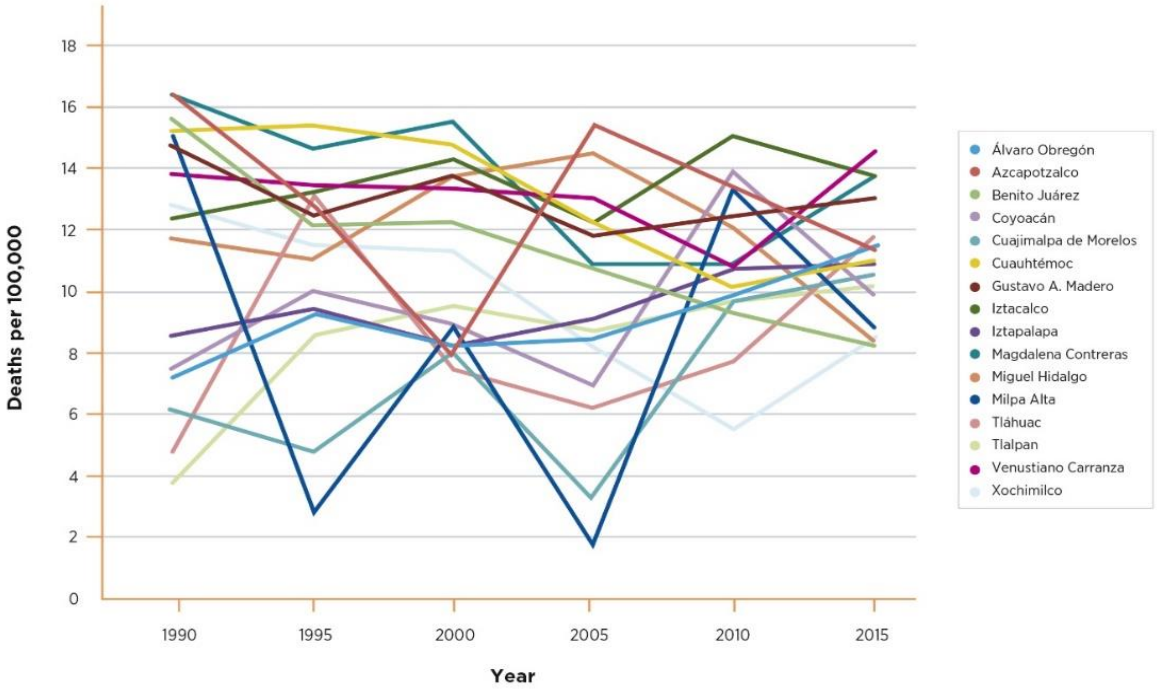
Figure 22. Time trends of alcaldía-*specific colon cancer mortality rates (deaths per 100,000)*



Stomach Cancer

The stomach cancer mortality rate in Mexico City has shown little change with 1990 and 2015 rates close to 11 deaths per 100,000. The initial scatter in stomach cancer rates among *alcaldías* was lost by 2015, having a tighter pattern by the end of the period -although with some *alcaldías* showing increasing trends, whereas others some improvements (Figure 23). *Alcaldías* with decreasing trends are Benito Juárez and Cuauhtémoc. Maps of the changes in the spatial distribution are found in Appendix VII.

Figure 23. Time trends of alcaldía*-specific stomach cancer mortality rates (deaths per 100,000)*

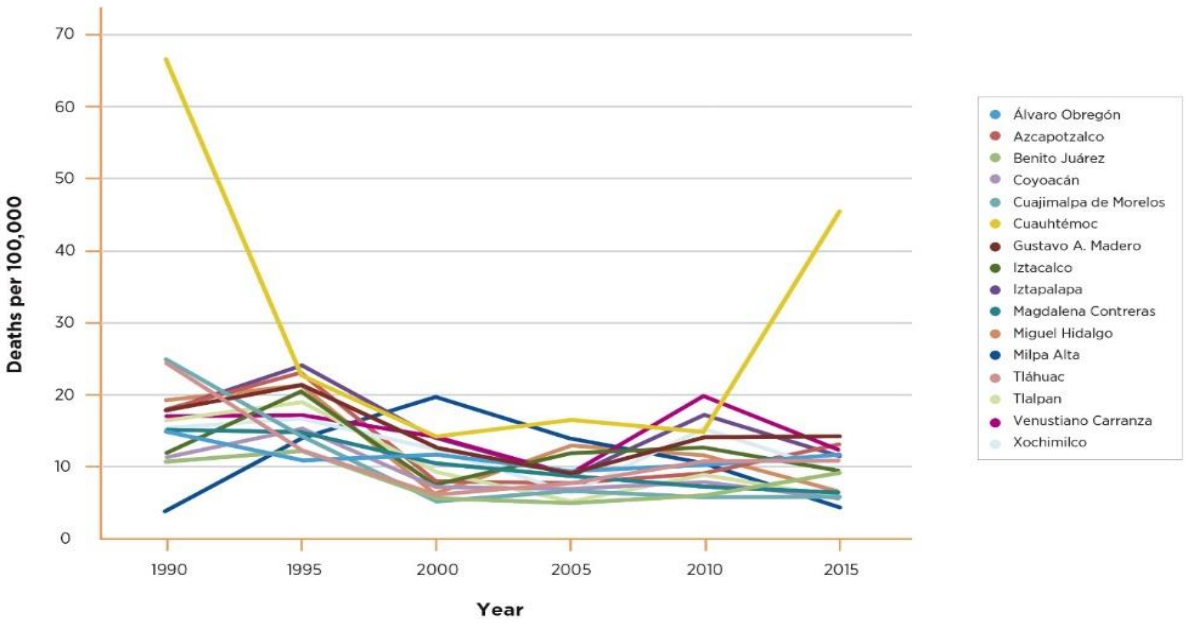


External Causes (Assault)

Deaths by assault include homicide and injuries inflicted by another person with intent to injure or kill, by any means. This category excludes injuries due to legal intervention, injuries due to operations of war, and injuries due to terrorism. We calculated the mortality rate for external causes (assault) for the population over 25 years of age.

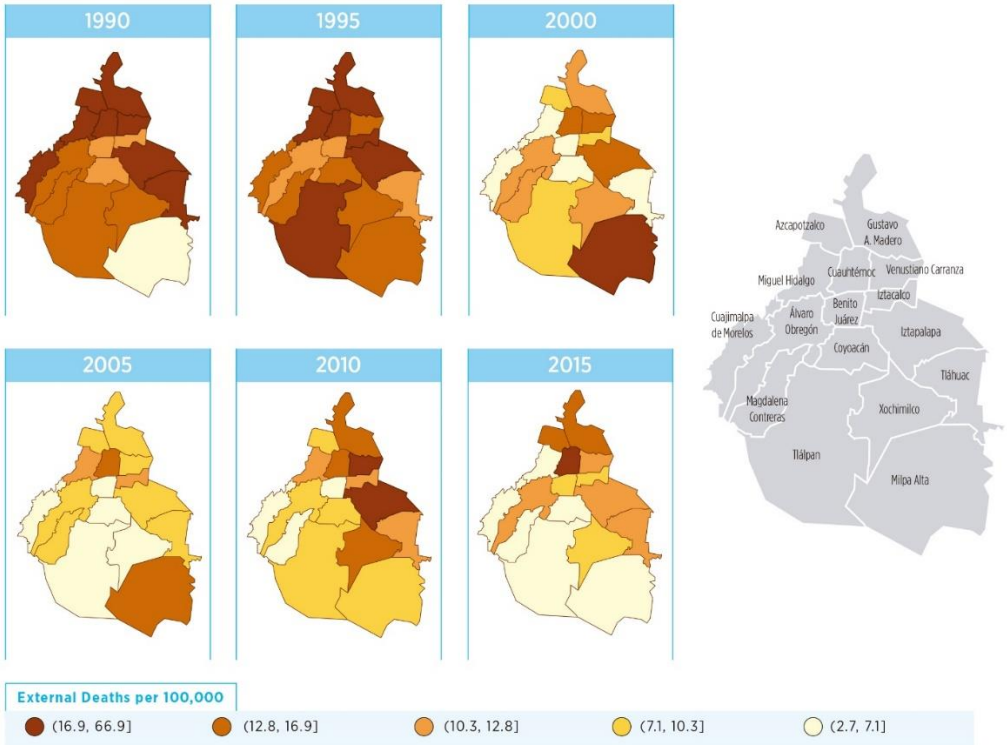
In Mexico City the mortality rate for external causes increased decreased from 19 to 11 per 100,000 deaths from 1990 to 2015. There is not much heterogeneity in the *alcaldía*-specific rates of mortality due to external causes. The most notable exception is Cuauhtémoc, showing initial and final rates that exceed significantly those from the rest of the *alcaldías* (Figure 24).

Figure 24. Time trends of alcaldía-*specific mortality rates of external causes (assault) (deaths per 100,000)*



Maps of *alcaldía*-specific mortality rates from external causes illustrate the changes in the spatial distribution over time (Figure 25).

Figure 25. Spatial distribution of alcaldía*-specific mortality rates of external causes (assault) (deaths per 100,000) by year*



RESULTS: HEALTH BENEFITS AND AIR POLLUTION

Correlations

In a preliminary analysis to examine potential associations, we calculated the correlation between the *alcaldía*- and year-specific measures of life expectancy versus corresponding measures of air pollution ($PM_{2.5}$ and O_3) and the socioeconomic position indicators. Table 3 shows these correlations for life expectancy at birth for the whole population, for men and women separately, and for temporary life expectancy for the two age groups, 0 to 4 years and 25 to 74 years, for the 1990-2015-time period.

Table 3 Correlations between alcaldía- *and year-specific life expectancy and temporary life expectancy with air pollution and socioeconomic position indicators*

Air Pollution and Socioeconomic Position Indicators		Life Expectancy			Temporary Life Expectancy	
		Total	Men	Women	0-4 Years	25-74 Years
Fine Particles	$PM_{2.5}$	-0.73	-0.68	-0.75	-0.65	-0.72
Ozone	O_3	-0.66	-0.59	-0.70	-0.67	-0.59
Low Income	PO2SM	-0.68	-0.56	-0.77	-0.62	-0.65
Overcrowding	VHAC	-0.64	-0.50	-0.75	-0.56	-0.57
Low Education	SPRIM	-0.57	-0.43	-0.66	-0.57	-0.52
Illiteracy	ANALF	-0.50	-0.36	-0.59	-0.53	-0.45
No Sewer nor Toilet	OVSDE	-0.44	-0.36	-0.47	-0.46	-0.40
No Electricity	OVSEE	-0.35	-0.30	-0.34	-0.48	-0.36
Soil Floor	OVPT	-0.26	-0.19	-0.30	-0.37	-0.25
No Running Water	OVSAE	-0.09	0.00	-0.15	-0.18	-0.06
Small Villages	PL<5000	-0.04	0.03	-0.09	-0.06	-0.03
Total Population	POB_TOT	0.07	0.08	0.06	0.11	0.03

There is a consistent negative correlation between air pollution ($PM_{2.5}$ and O_3) and each of the measures of life expectancy. That is, higher *alcaldía*- and year-specific air pollution is correlated with lower life expectancy. There also are negative correlations with many of the socioeconomic position indicators. These bivariate correlations are suggestive of associations, but also strongly indicate the importance of controlling for socioeconomic and other *alcaldía*- and year-specific factors as alternative explanations for the apparent associations between life expectancy or temporary life expectancy and air pollution.

Similarly, we also calculated the correlation between the *alcaldía*- and year-specific measures of years of life lost (for two age-groups and for specific mortality causes) versus corresponding measures of air pollution ($PM_{2.5}$ and O_3) and socioeconomic position indicators. Table 4 shows these correlations for years of life lost for the two age groups, 0 to 4 years and 25 to 74 years, and for the five-postulated air pollution-related mortality causes.

Table 4. Correlations between alcaldía- *and year-specific years of life lost with air pollution and socioeconomic position indicators*

		Life Years Lost						
Air Pollution and Socioeconomic Position Indicators		0-4 Years	25-74 Years	ALRI	IHD	Stroke	Lung Ca	COPD
Fine Particles	$PM_{2.5}$	0.65	0.72	0.46	0.45	0.37	0.43	0.60
Ozone	O_3	0.67	0.59	0.62	0.26	0.28	0.42	0.58
Low Income	PO2SM	0.62	0.65	0.73	0.05	0.28	0.15	0.45
Overcrowding	VHAC	0.56	0.57	0.73	-0.05	0.24	0.02	0.37
Low Education	SPRIM	0.57	0.52	0.79	-0.13	0.20	0.06	0.36
Illiteracy	ANALF	0.53	0.45	0.81	-0.25	0.10	-0.05	0.24
No Sewer nor Toilet	OVSDE	0.46	0.40	0.79	-0.27	-0.04	-0.04	0.14
No Electricity	OVSEE	0.48	0.36	0.79	-0.22	-0.14	-0.04	0.12
Soil Floor	OVPT	0.37	0.25	0.74	-0.40	-0.13	-0.20	-0.02
No Running Water	OVSAE	0.18	0.06	0.52	-0.37	-0.13	-0.20	-0.14
Small Villages	PL<5000	0.06	0.03	0.30	-0.39	-0.14	-0.32	-0.36
Total Population	POB_TOT	-0.11	-0.03	-0.17	0.06	0.08	0.03	0.05

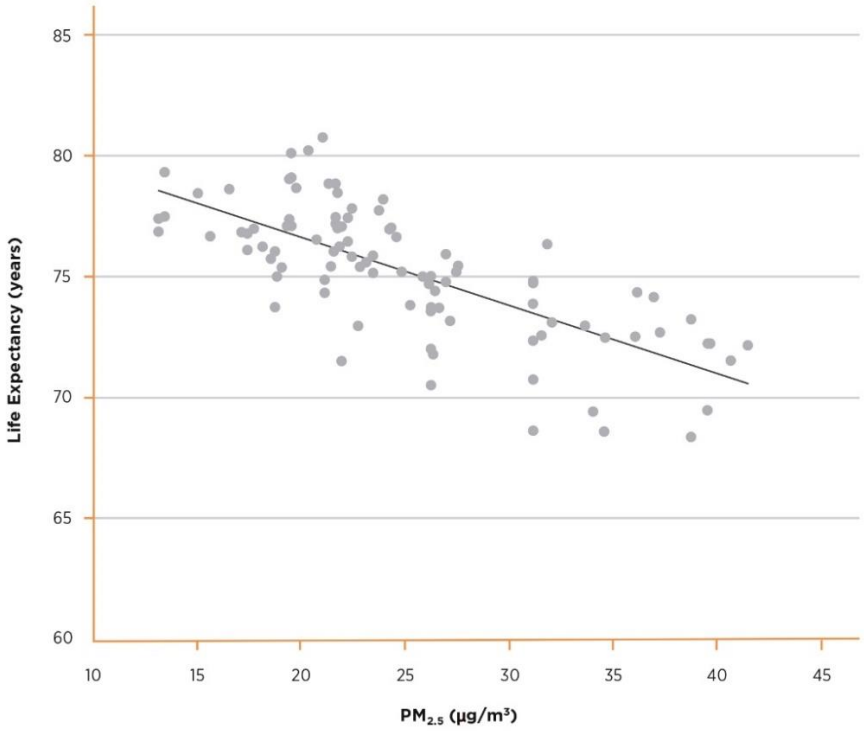
In this case, we see positive correlations between air pollution indicators and several of the socioeconomic position indicators with years of life lost. The observant reader will note that these correlations for years of life lost for ages 0-4 years and 25-54 years are the negative values of the previously presented correlations for temporary life expectancy for these corresponding age groups. This is because of the algebraic connection between these two measures of life expectancy. The interpretation here would be that higher *alcaldía*- and year-specific air pollution is correlated with higher number of years lost.

Bi-Variate Associations[16]

The objective of the regression analyses is to quantify the association between the various measures of *alcaldía*- and year-specific life expectancy and air pollution adjusting for alternative explanations, and confounding risk factors. To illustrate, let us consider first the bivariate association between $PM_{2.5}$ and total life expectancy. The Figure 26a is a scatter plot of these *alcaldía*- and year-specific data. There is a clear negative association between life expectancy and $PM_{2.5}$ concentrations. Note that we calculated this correlation as -0.73 as shown in Table 3 above.

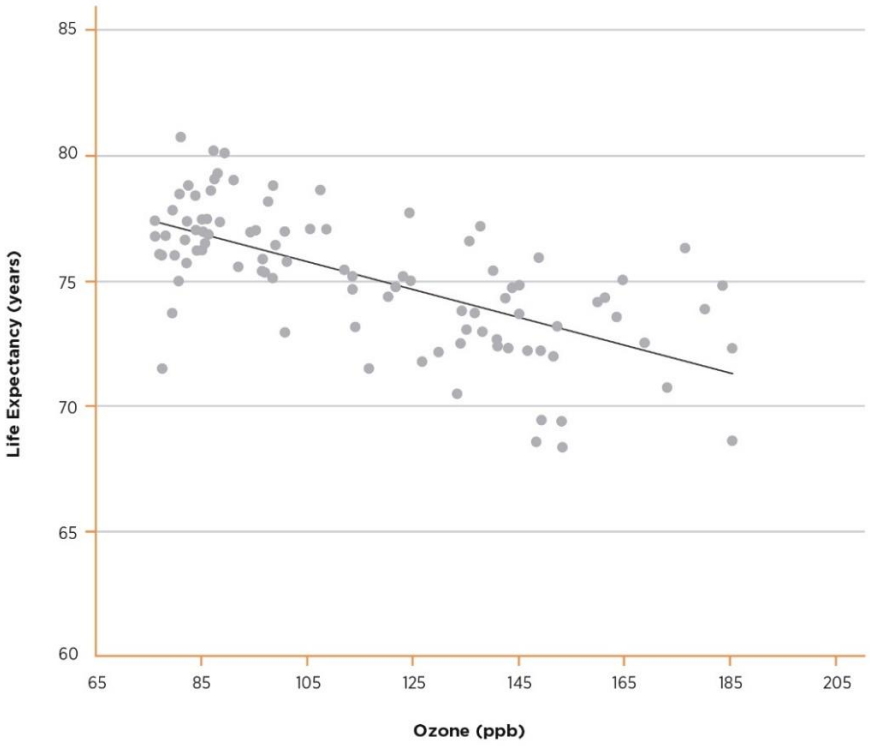
A similar representation for ozone is shown in Figure 26b, where we also find a negative association between life expectancy at birth and ozone concentrations. We computed this correlation as -0.66, as shown in Table 3.

Figure 26a. Scatterplot and fitted line of alcaldía- *and year-specific life expectancy versus annual average $PM_{2.5}$ concentrations*



[16] The scatter plots with the adjusted line included in this section show the response variable (life expectancy) and the data of the predictors ($PM_{2.5}$ or ozone). The plots include the regression line for all the data (Fig. 26 a and b), regression lines for each year (Fig. 27 a and b), and regression lines for each *alcaldía* (Fig. 28 a and b) to represent visually the cross-sectional and longitudinal associations between life expectancy and air pollutants.

Figure 26b. Scatterplot and fitted line of alcaldía- *and year-specific life expectancy versus seasonal (6 month) 1-hour maximum ozone concentrations (ppb)*



These data represent both the cross-sectional (spatial) associations across the *alcaldías* and the longitudinal (temporal) associations across the years. To illustrate, the following figures show the same data, but dividing the points by year, showing the cross-sectional associations (Figures 27a and b), and by *alcaldía,* showing the longitudinal associations (Figures 28a and b). Interestingly, there is a similar negative association between life expectancy and $PM_{2.5}$ or ozone, both cross-sectionally (within each year) and longitudinally (within each *alcaldía*). The cross-sectional associations (within each year) between life expectancy and ozone are not clear; however, there's a clear negative longitudinal association (within each *alcaldía*) between life expectancy and ozone.

Figure 27a. Scatterplot and fitted lines of alcaldía*-specific life expectancy versus annual average $PM_{2.5}$ concentrations by year*

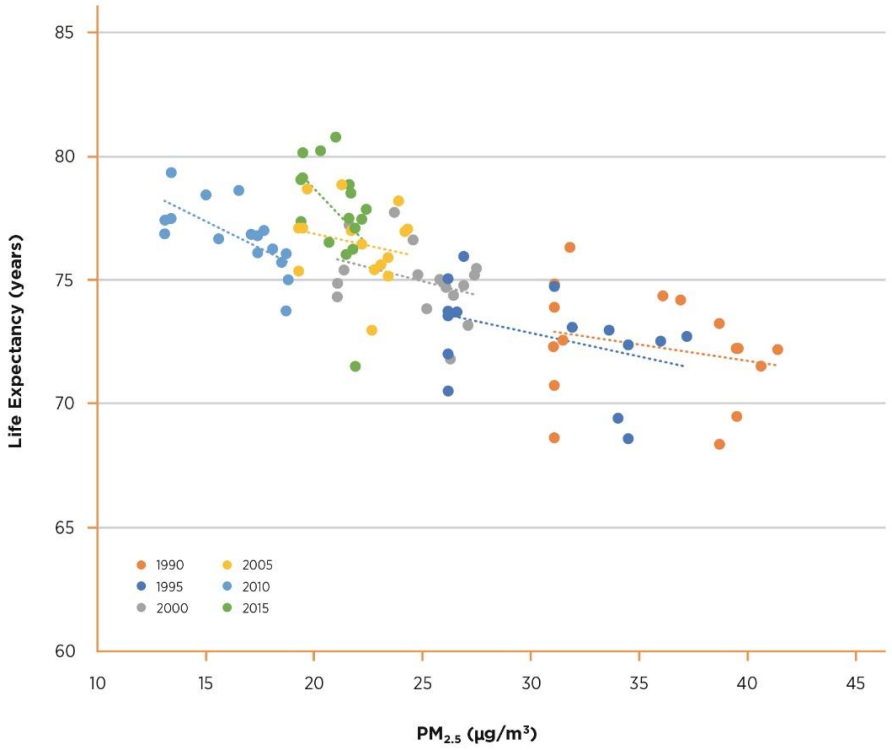


Figure 27b. Scatterplot and fitted lines of alcaldía*-specific life expectancy versus seasonal (6 month) 1-hour maximum ozone concentrations (ppb) by year*

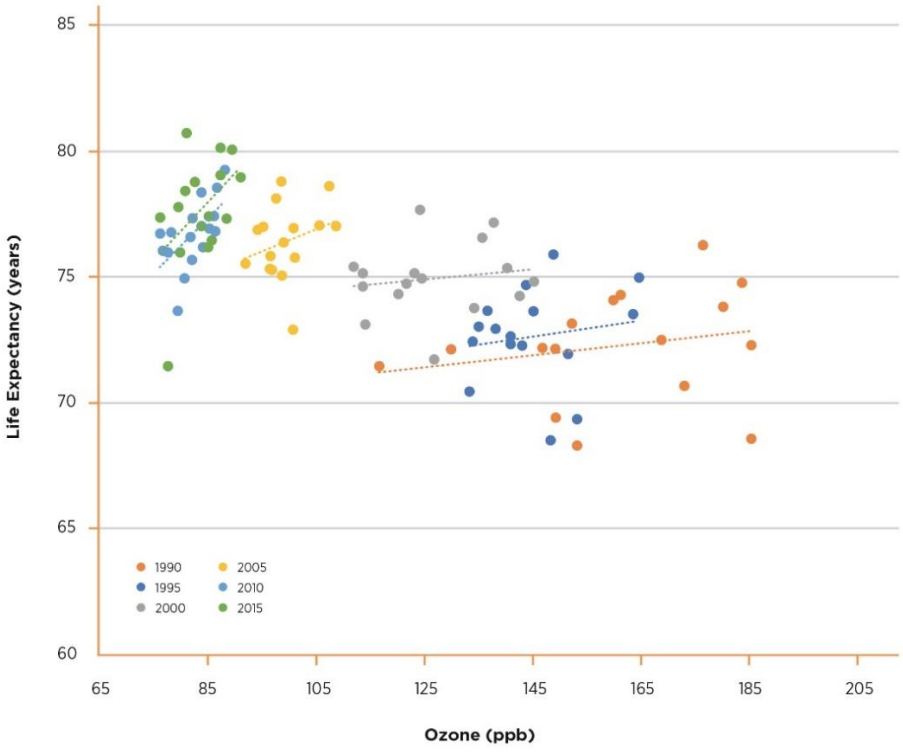


Figure 28a. Scatterplot and fitted lines of year-specific life expectancy versus annual average $PM_{2.5}$ concentrations by alcaldía

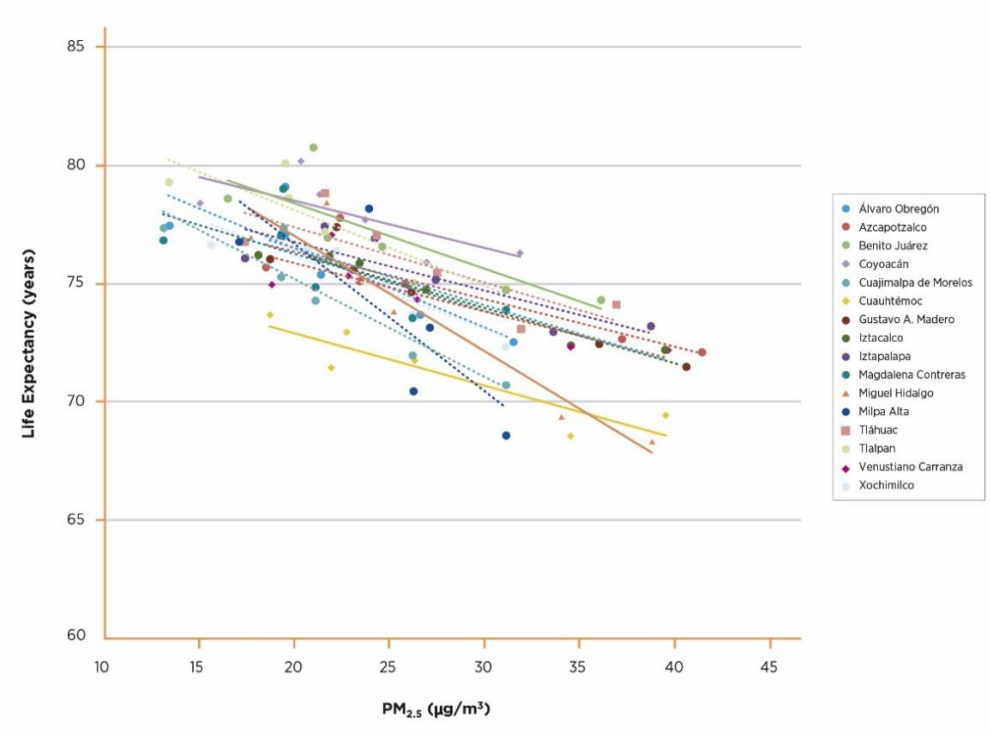
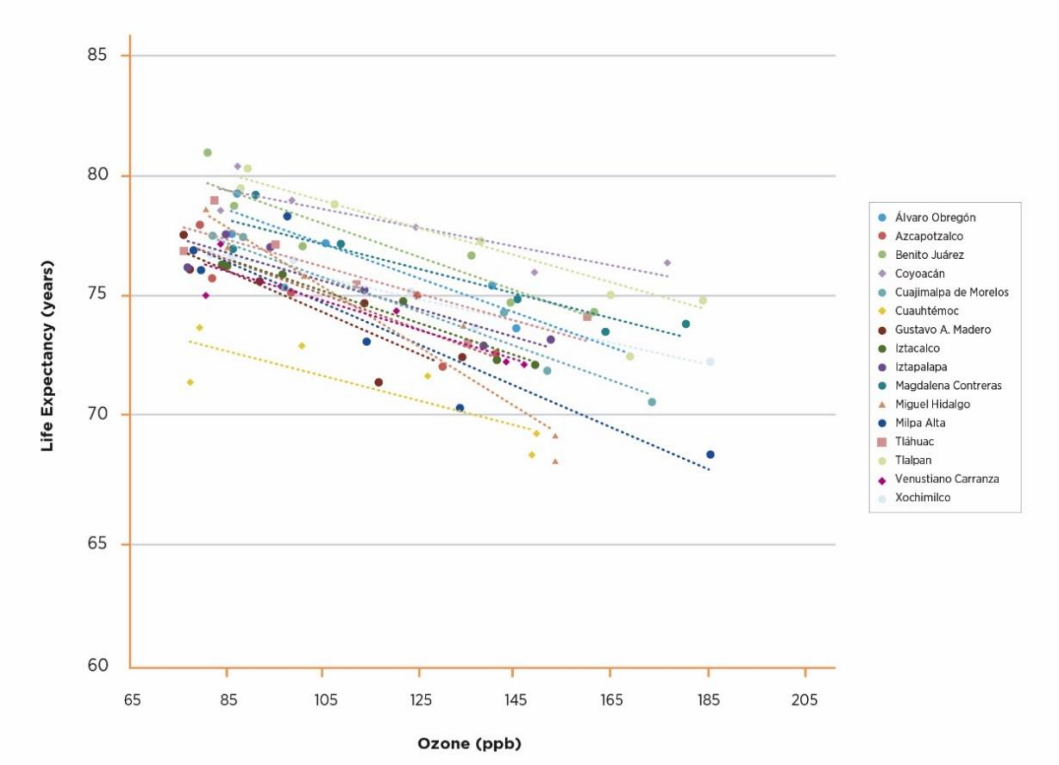


Figure 28b. Scatterplot and fitted lines of year-specific life expectancy versus seasonal (6 month) 1-hour maximum ozone concentrations (ppb) by alcaldía



Regression Analyses

As a general measure of population health, life expectancy reflects the net effects of the full range of risk factors including genetics, behaviors, the environment, community context, and efficacy of medical treatment and care. Thus, *alcaldía* air pollution levels are only one of a myriad of individual, population, and community factors affecting life expectancy. How can we control for possible confounding by these known and unknown factors?

Our first approach (Simple Model) assumes that each *alcaldía* has a unique set of characteristics that define life expectancy compared to the other *alcaldías*. To adjust for these *alcaldía*-specific characteristics, we allow each *alcaldía* to have its' own level, that is a random effect for each of the 16 *alcaldías*. Similarly, we assume that life expectancy across all the *alcaldías* changes year-to-year and allow each year to have its' own level, that is a random effect for each year. In this Simple Model we estimate a regression coefficient common to all *alcaldías* for each pollutant by assuming a fixed effect for each, $PM_{2.5}$ and O_3, adjusting for random effects of *alcaldía* and year. Note that the data set is small, consisting of life expectancy and air pollution for 16 *alcaldías* in each of six years, for a total of 96 data points. In this Simple Model, we use one degree of freedom for $PM_{2.5}$, one for O_3, 16 for *alcaldía*, and 6 for year.

We recognize that the base population for data point varies substantially between *alcaldía* and census year, ranging between 100 thousand and 2 million inhabitants. Therefore, in our regression models, we weight each point by the square root of the population that is giving more weight to the point with larger populations.

This Simple Model does not consider the *alcaldía*-specific socioeconomic position (SEP) data available for the census and intercensal count years. Therefore, in a second approach (SEP Model), we include fixed effects for these SEP characteristics in addition to the fixed effects of $PM_{2.5}$ and O_3 and the random effects of *alcaldía* and year in the population weighted regression. Each of the eight SEP indicators uses an additional degree of freedom. Because of the inconsistent and incomplete smoking prevalence data, we are not able to adjust directly for *alcaldía*-specific smoking rates in the SEP Model.

In the next step (Full Model), we additionally adjust for reference causes of death, which are general measures of population health. We include *alcaldía*- and census year-specific rates for five common causes of death which we expect are unrelated or weakly related to air pollution –diabetes, hypertension, colon cancer, stomach cancer, and external causes (assault). These reference death causes provide additional adjustment for confounding by other unmeasured factors that may affect life expectancy. In addition, we adjust for deaths rates of chronic obstructive pulmonary disease (COPD) and lung cancer, two health indicators known to be causally associated with smoking. While COPD and lung cancer deaths may also be related to air pollution, inclusion of these variables provides a conservative estimate of the effect of air pollution adjusting with these proxy indicators for the prevalence of cigarette smoking. In this Full Model, each reference death rate is treated as a fixed effect with one additional degree of freedom.

As noted above, this is a fairly small dataset. The Full Model with intercept, 17 fixed effect variables, and 22 random effect variables is straining the statistical power of these 96 observations. Therefore, we created a Parsimonious Model by backwards elimination of fixed effect variables with p-values greater than 0.10. Fixed effects of $PM_{2.5}$ and O_3, fixed effects of COPD and lung cancer death rates, and random effects of *alcaldía* and census year were forced into the population-adjusted Parsimonious Model.

We applied the Parsimonious Model to the health indicators of life years lost for children and for adults, and for causes of death determined to be related with air pollution exposures in the Global Burden of Disease Analyses and included in our Phase II work. Main explanatory variables were $PM_{2.5}$ and O_3, and models were adjusted for socioeconomic position indicators, and the remaining specific causes of mortality –that is, except the cause modeled as the health outcome of interest.

Regression Results

Total Life Expectancy: The results of the sequential weighted regression analyses of life expectancy versus $PM_{2.5}$ and O_3 are presented in Table 5. Our Simple Model shows a statistically significant association ($p<0.01$) of life expectancy with $PM_{2.5}$ and O_3, as well as the random effects of *alcaldía* and census year. The negative coefficient (beta) for $PM_{2.5}$ means that for each 10 $\mu g/m^3$ decrease on *alcaldía*-specific mean annual $PM_{2.5}$, life expectancy increased by 1.36 years with a standard error of 0.40 years, giving a 95% Confidence Interval (95% CI) of 0.58 to 2.15 years. Likewise, a 10 ppb decrease in average peak seasonal O_3 was associated with a 0.27-year increase in life expectancy, with a 95% CI of 0.10 to 0.44 years.

Including the eight socioeconomic position variables (SEP Model) resulted in no significant associations with $PM_{2.5}$, O_3, or any of the SEP variables. In the Full Model, we did find negative associations for $PM_{2.5}$ and a statistically significant negative association for O_3. We did not find statistically significant associations ($p>0.10$) for any SEP or death rates other than diabetes and COPD. This suggests the Full Model, with 40 degrees of freedom, may be overfitting the data.

The stepwise backwards regression found three significant ($p<0.10$) risk factors – overcrowding, diabetes death rates, and death rates from external causes. In addition, death rates for colon cancer were just over the criteria ($p=0.107$) and included in the Parsimonious Model. As previously noted, COPD and lung cancer death rates were included as population proxy indicators of smoking. This Parsimonious Model found a statistically significant ($P=0.028$) association of decreased $PM_{2.5}$ (10 $\mu g/m^3$ annual mean) with 0.89 years improvement in life expectancy (95% CI 0.14 to 1.65 years), and a significant association ($P=0.004$) of decreased O_3 (10 ppb seasonal hourly daily peak) with 0.24 years of improved life expectancy (95% CI 0.08 to 0.40 years).

Table 6 compares the Parsimonious Model results for the total population with analyses of life expectancy for men and women separately. For $PM_{2.5}$ we found the same effect estimates for men (0.79 years, 95% CI -0.14 to 1.72 years) and women (0.79 years, 95% CI 0.13 to 1.45 years). For O_3 women had a much smaller effect estimates (0.13 years, 95% CI -.03 to 0.28) compared to men (0.36 years, 95% CI 0.15 to 0.57), although these sex-specific effect estimates have overlapping confidence intervals.

Table 5. Weighted regression models of life expectancy versus fixed effects of air pollution adjusting sequentially for random effects of alcaldías *and census years (Simple), socio-economic position indicators (SEP), reference death rates and proxy indicators for prevalence of smoking (Full), and restricted model (Parsimonious)*

Variable	Simple Model			SEP Model			Full Model			Parsimonious Model		
	Beta	SE	p-value	Beta	SE	p-value	Beta	SE	p-value	Beta	SE	p-value
Intercept	81.77	1.24	<0.0001	81.49	1.08	<0.0001	90.74	2.02	<0.0001	90.43	1.73	<0.0001
$PM_{2.5}$ (10µg/m^3)	-1.36	0.40	0.0021	-0.70	0.55	0.21	-0.37	0.45	0.48	-0.89	0.38	0.028
O_3 (10 ppb)	-0.27	0.09	0.0031	0.12	0.11	0.27	-0.29	0.09	0.012	-0.24	0.08	0.004
Low Income				-0.07	0.05	0.14	0.00	0.04	0.94			
Overcrowding				-0.10	0.06	0.11	-0.18	0.03	0.11	-0.11	0.02	<0.0001
Low Education				0.06	0.25	0.80	-0.09	0.19	0.66			
Illiteracy				0.98	0.95	0.31	0.94	0.72	0.32			
Soil Floor				-0.05	0.25	0.84	-0.22	0.23	0.35			
No Electricity				-0.38	0.55	0.50	0.59	0.54	0.28			
No Sewer nor Toilet				-0.11	0.09	0.20	0.01	0.09	0.88			
No Running Water				0.07	0.08	0.37	0.02	0.07	0.79			
Diabetes (Death Rate)							-0.02	0.01	0.039	-0.02	0.01	0.002
Hypertension (Death Rate)							-0.02	0.03	0.55			
Colon Cancer (Death Rate)							-0.09	0.05	0.103	-0.08	0.05	0.107
Stomach Cancer (Death Rate)							0.04	0.05	0.40			
External Causes (Assault) (Death Rate)							-0.02	0.02	0.16	-0.02	0.01	0.094
COPD (Death Rate)							-0.06	0.02	0.018	-0.05	0.02	0.012
Lung Cancer (Death Rate)							-0.03	0.04	0.37	-0.03	0.03	0.37
Alcaldía (Random)			<0.0001			<0.0001			<0.0001			<0.0001
Census Year (Random)			<0.0001			0.059			0.93			0.078

Table 6. Parsimonious weighted regression models of life expectancy for total population, for men and for women versus fixed effects of air pollution, socio-economic position indicators, reference death rates, proxy indicators for prevalence of smoking and random effects of alcaldías *and census years*

Variable	Total Life Expectancy			Life Expectancy Men			Life Expectancy Women		
	Beta	SE	p value	Beta	SE	p value	Beta	SE	p value
Intercept	90.43	1.73	<0.0001	90.29	2.23	<0.0001	89.62	1.47	<0.0001
$PM_{2.5}$ (10µg/m³)	-0.89	0.38	0.028	-0.79	0.47	0.12	-0.79	0.33	0.026
O_3 (10 ppb)	-0.24	0.08	0.004	-0.36	0.11	0.0011	-0.13	0.08	0.108
Overcrowding	-0.11	0.02	<0.0001	-0.11	0.03	0.0015	-0.11	0.02	<0.0001
Diabetes Death Rate	-0.02	0.01	0.002	-0.02	0.01	0.0059	-0.01	0.01	0.024
Colon Cancer Colon Cancer Death	-0.08	0.05	0.107	-0.15	0.06	0.022	0.01	0.05	0.83
External Causes (Assault) Death Rate	-0.02	0.01	0.094	-0.05	0.02	0.014	-0.01	0.01	0.56
COPD Death Rate	-0.05	0.02	0.012	-0.06	0.02	0.021	-0.05	0.02	0.009
Lung Cancer Death Rate	-0.03	0.03	0.37	-0.03	0.04	0.52	-0.05	0.03	0.095
Alcaldía (Random)			<0.0001			<0.0001			0.0026
Census Year (Random)			0.078			0.30			0.080

Age and Cause-Specific Years Life Lost: Table 7 shows the Parsimonious Model to the years of life lost for two age groups of specific interest – 0 to 4 years and 25 to 74 years. The first line of the Table repeats the previous Parsimonious Model findings for reduced life expectancy at birth (Table 5). There was a modest, statistically non-significant increase in years of life lost between ages 0 and 4 years associated with $PM_{2.5}$ (0.0070 years or 2.5 days) and a significant small increase with O_3 (0.0037 years or 1.3 days). In contrast, there was a highly significant association of years lost between ages 25 and 74 years associated with both pollutants, $PM_{2.5}$ (0.56 years, 95% CI 0.28 to 0.83) and O_3 (0.10 years, 95% CI 0.03 to 0.17).

We also applied the parsimonious model to the estimated years of life lost due to specific causes expected to be associated with air pollution for children between 0 and 4 years old and for adults from 25 to 74 years old (Table 7). Among children we found no positive association with years of life lost from acute lower respiratory infections (ALRI).

Among adults aged 25 to 74 years, we found significantly increased years of life lost attributable to ischemic heart disease (0.094 years, 95% CI 0.027 to 0.16, p=0.018) and COPD (0.037 years, 95% CI 0.007 to 0.067, p=0.022) associated with $PM_{2.5}$ exposures. There were also positive but not statistically significant associations of lung cancer (p=0.090) and cerebrovascular stroke (p=0.07) with $PM_{2.5}$. There

was a nonsignificant association of O_3 with years life lost attributable to COPD (p=0.14) and lung cancer (p=0.14). In sensitivity analyses of the lung cancer associations without adjusting for COPD death rates, there was a significant association with $PM_{2.5}$ (0.20 years, 95% CI 0.004 to 0.035, p=0.02) and with O_3 (0.0043 years, 95% CI 0.0003 to 0.0088, p=0.040). For COPD, the associations with $PM_{2.5}$ and O_3 did not change after deletion of the lung cancer mortality from the Parsimonious Model; the $PM_{2.5}$ effect estimate was robust and remained significant (0.037 years, 95% CI 0.010 to 0.064, p=0.010).

Table 7. Effects of $PM_{2.5}$ and O_3 in Parsimonious Models of years of life lost, ages 0 to 4 years, 25 to 74 years, and causes of death associated with air pollution (One model for each health outcome indicator)

Health Outcome (Dependent Variable)	$PM_{2.5}$ ($10\mu g/m^3$)			O_3 (10ppb)		
	Beta	CI 95%	p value	Beta	CI 95%	p value
Total Life Expectancy	0.89	0.38	0.028	0.24	0.08	0.004
0-4 years	**0.0070**	**0.0068**	**0.33**	**0.0037**	**0.0018**	**0.049**
ALRI	-0.0033	0.0014	0.023	0.0004	0.0003	0.17
25-74 años	**0.555**	**0.138**	**0.00012**	**0.103**	**0.037**	**0.0065**
Ischemic Heart Disease	0.094	0.034	0.018	0.003	0.008	0.73
Cerebrovascular Stroke	0.023	0.012	0.070	0.001	0.003	0.74
Lung Cancer	0.013	0.007	0.090	0.003	0.002	0.14
COPD	0.037	0.015	0.022	0.005	0.003	0.14

DISCUSSION

This project confirmed recent findings from studies conducted in other countries: Mexico City inhabitants have adverse effects from air pollution exposures, that are manifested in changes in life expectancy equivalent to those seen in the United States and other developed countries. Longitudinal prospective studies have consistently found that people living in communities with higher average $PM_{2.5}$ concentrations die earlier, that is have additional years of life lost. We found that over the past 25 years in Mexico City with each 10 $\mu g/m^3$ improvement in annual average $PM_{2.5}$ there's an association with 0.89 years (95% CI 0.14 to 1.65 years) longer life expectancy at birth, and simultaneously, and independently, that with each 10 ppb improvement in seasonal hourly peak O_3 there's an association with 0.24 years (95% CI 0.08 to 0.40 years) longer life expectancy.

Our analyses show stronger associations with years of life lost with $PM_{2.5}$ and O_3 among adults 25 to 74 years of age. Each 10 $\mu g/m^3$ improvement in $PM_{2.5}$ was associated with 0.56-year (95% CI 0.28 to 0.83) reduction in life years lost, and each 10 ppb improvement in O_3 with 0.10 (95%CI 0.03 to 0.17) reduction in life years lost. This is of interest because it shows consistency with the cohort studies, such as the Harvard Six Cites study (Dockery et al., 1993), which examined mortality in a longitudinal follow-up of adults 25 to 74 years of age and found that for $PM_{2.5}$ exposures survival was lower and mortality rates higher in the dirtiest city than in the cleanest one. For every 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations, mortality rates increased by approximately 1.5%.

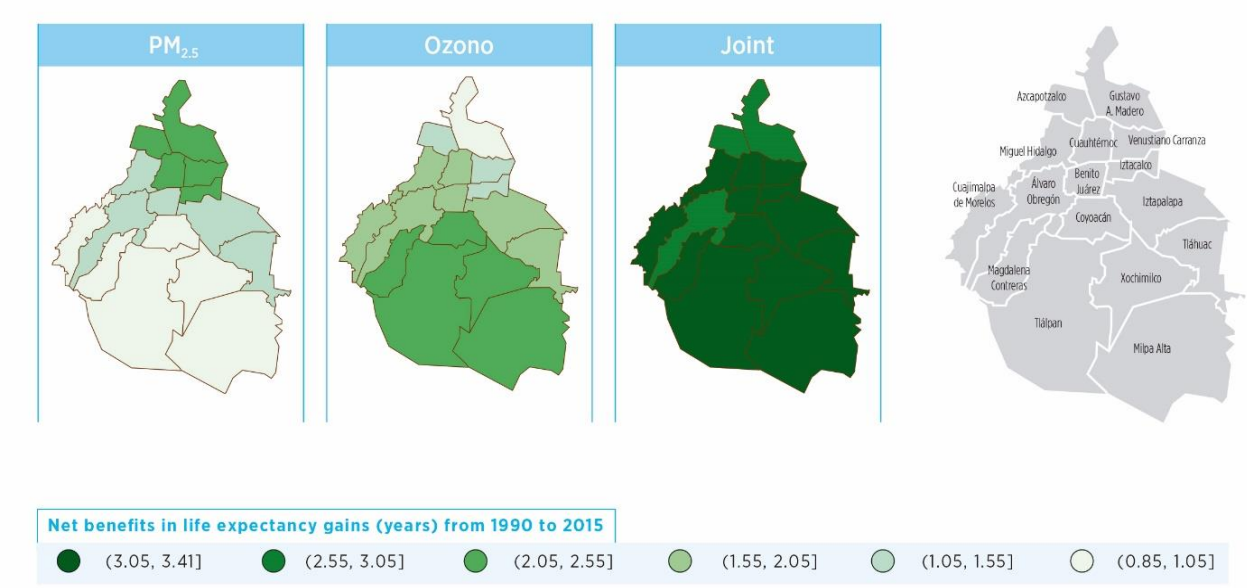
Our by-sex results, with one model life expectancy model for men and one for women, generate hypothesis for future research. There was a much smaller O_3 effect for women than for men and similar $PM_{2.5}$ effects for both. Different time-activity patterns with differing times spent in outdoor and indoor microenvironments between men and women may partly explain our findings. If men spend more time outdoors --for example, due to their commuting time to and from work—are more highly exposed to ozone, since most of O_3 exposures occur outdoors (Brauer and Brook, 1997; Weschler, 2006); it follows that if women spend more time indoors, they would be less exposed to ozone. Conversely, $PM_{2.5}$ exposures for men and women are likely to be similar, even under different time-spent indoors and outdoors scenario, because fine particles are less reactive than ozone and penetrate easily to indoor environments which results in similar exposures in outdoor and indoor environments (Sarnat et al., 2013).

Indeed, the evidence for shorter life expectancy in Mexico City associated with $PM_{2.5}$ is very consistent with similar studies of changes in county-specific life expectancy in the United States. Pope et al. (2009) reported that life expectancy in 211 counties in 51 metropolitan areas in the United States increased by 0.61 years (95% CI 0.22 to 1.00) associated with each 10 $\mu g/m^3$ improvement in $PM_{2.5}$ between 1980 and 2000. Correia et al. (2017) extended to 545 counties between 2000 and 2007, finding 0.35 years (95% CI 0.04 to 0.66) improved life expectancy associated with each 10 $\mu g/m^3$ improvement in $PM_{2.5}$ annual average concentrations.

There is limited evidence that living in communities with higher O_3 is associated with increased mortality and shorter life expectancy. It is likely that the differential spatial variability pattern of O_3 and $PM_{2.5}$

concentrations in Mexico City –with high O_3 levels in the southwest vs. high $PM_{2.5}$ levels in the north and northeast-- allowed the identification of an independent effect for O_3. The finding in Mexico City that improvements in life expectancy are associated significantly with reductions in O_3 may have been also possible due to the wide range of concentrations seen across the study period, spanning approximately between 80 and 160 ppb, which allows to have the statistical power to detect an association. This is an important contribution to the scientific evidence of population health benefits that result from air quality improvements.

Figure 29 Independent and joint net benefits measured as life expectancy gains (years) from improved annual average $PM_{2.5}$ concentrations and seasonal maximum 1-hour daily ozone concentrations in Mexico City, 1990 – 2015



Our Parsimonious Model shows that net benefits associated with improved $PM_{2.5}$, with decreased annual concentrations of almost 15 $\mu g/m^3$ from 1990 to 2015, are in the order of 1.3 years increased life expectancy. Improvements for ozone, with decreased seasonal 1-hour maximum daily concentrations of almost 80 ppb, represent an increase in life expectancy of close to 1.9 years. Thus, the joint net benefit associated with improvements in both pollutants represents an increase in life expectancy of 3.2 years. As seen in Figure 29, net benefits present a different spatial pattern for $PM_{2.5}$ and O_3. Greater improved $PM_{2.5}$ air quality in the north has led to larger gains in life expectancy (up to 1.7 years) in those *alcaldías* attributable to $PM_{2.5}$. Greater improvements in O_3 air quality in the south have led to larger gains in life expectancy (up to 2.6 years) attributable to O_3. Together, the joint effects of the improvements in $PM_{2.5}$ and O_3 has led to substantial improvements in life expectancy (2.6 to 3.4 years) in all *alcaldías* (Figure 29).

Apte et al. (2018) have recently estimated that current (2016) $PM_{2.5}$ exposures reduce life expectancy globally by 1.03 years, and O_3 exposures by 0.05 years in a risk assessment based on the Global Burden of Disease exposure-response functions. They suggest that if all countries met the World Health

Organization Air Quality Guideline for $PM_{2.5}$ of 10 $\mu g/m^3$, median life expectancy could increase by 0.6 year (interquartile range of 0.2−1.0 year), a benefit of a magnitude that is similar to that of eradicating lung and breast cancer together. In their supplemental data, they report an average $PM_{2.5}$ for Mexico (i.e. the whole country) of 18.3 $\mu g/m^3$, which implies an average of 0.48 years life lost due to $PM_{2.5}$.

Our results, based on analyses using direct *alcaldía*-specific information on air quality and health-related outcomes, adjusting for socioeconomic position, general health indicators and proxy indicators of accumulated exposure to smoking, are consistent with world-wide most recent findings that indicate that air quality improvements have beneficial public health effects by reducing premature deaths –which is equivalent to increased life expectancy or reduced life years lost. Public policies aimed at improving air quality will benefit the population. Our study shows that significant benefits are expected most importantly for adults aged 25 to 74 years old and from increases in years of life attributable to ischemic heart disease and chronic obstructive pulmonary disease.

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Contents

APPENDIX I. METHODS FOR LIFE EXPECTANCY

Methods for Life Expectancy and Years Life Lost

1. *Data and Data Source*

This project uses different data and data sources.

The population data came from census (1990, 2000, 2010) and intercensal surveys (1995, 2005,2015). We download the information from the National Institute of Geography and Informatics (INEGI), from the website: http://www.beta.inegi.org.mx/proyectos/ccpv/2010/

The total number of deaths (1990 – 2015) comes from INEGI:
http://www.beta.inegi.org.mx/proyectos/registros/vitales/mortalidad/

The specific causes of death were provided directly by SEDEMA

2. *Population Corrections (Distribution of "Unspecified")*

Most of the data bases contain not specified values. We solve this problem though proration, this mean distribute the unspecified values into the specified cases accordingly to the distribution of latter.

The next section explains the methodology for population data, but it is the same for mortality data.

$$TP_i = Total\ population\ i$$

$$TUP_i = Total\ unspecified\ population\ i$$

$$P_{x,x+n,i} = Population\ between\ the\ age\ x\ and\ x+n\ for\ the\ general\ gruop\ i$$

$$P^*_{x,x+n,i} = P_{x,x+n,i} + \frac{P_{x,x+n,i}}{TP_i - TUP_i} \times TUP_i$$
$$= prorrated\ population\ between\ the\ age\ x\ and\ x+n\ for\ the\ genral\ group\ i$$

Where *i* indicates men or women

3. *Life Tables – Single decrements process*

To simplify the notation, this and the next sections will assume that the data are prorated. This procedure was done for men and women separately, then we omit the super index * and the sub index *i*.

To calculate life expectancy, we calculated the entire life table, the methodology used is based on the one described in Preston et all 2001 Chapter 3, therefore in this section is presented a summary of that methodology.

$$P_{x,x+n} = Observed\ Population\ between\ ages\ x\ and\ x+n$$

$$D_{x,x+n} = Observed\ number\ of\ deaths\ between\ ages\ x\ and\ x+n$$

$$m_{x,x+n} = \frac{D_{x,x+n}}{P_{x,x+n}} = mortality\ rate\ between\ ages\ x\ and\ x+n$$

$$q_{x,x+n} = \frac{2n\ \times m_{x,x+n}}{2 + n \times m_{x,x+n}} = probability\ of\ dying\ between\ ages\ x\ and\ x+n$$

$$p_{x,x+n} = 1 - q_{x,x+n} = probability\ of\ surviving\ between\ ages\ x\ and\ x+n$$

$$l_0 = 100{,}000 = radix, initial\ theoretical\ population$$

$$l_{x,x+n} = l_{x-n,x} \times p_{x,x+n} = number\ of\ survivors\ bwteen\ ages\ x\ and\ x+n$$

$$d_{x,x+n} = l_{x,x+n} \times q_{x,x+n} = number\ of\ deaths\ between\ ages\ x\ and\ x+n$$

$$a_{x,x+n} = average\ person - years\ lived\ in\ the\ interval\ by\ those\ dying\ in\ the\ interval$$

For convention $a_{x,x+n} = 2.5$ for all ages from x=5, for x=0 and x=5 $a_{x,x+n}$was calculated with the correction showed in Preston *et al*., 2001 Chapter 3, section 3.3.

$$L_{x,x+n} = n \times l_{x,x+n} + a_{x,x+n} \times d_{x,x+n} = person - years\ liven\ between\ ages\ x\ and\ x+n$$

For the open-ended interval $L_{x,w} = \frac{l_x}{m_{x,w}}$ with *w* = the las age group, i.e. 75+ or 85+

$$T_{x,x+n} = \sum_{k=x}^{w} L_{x,x+n} = Person - years\ lived\ above\ age\ x$$

$$e_x^o = \frac{T_{x,x+n}}{l_{x,x+n}} = expectation\ of\ life\ at\ age\ x$$

4. Life Tables – Multiple decrement process

We based the multiple decrement process analysis upon the methodology explained by Preston *et al*., 2001, Chapter 4. We present a summary of the most representative formulas used in our study.

In this section the super index *i* indicates the *i-th* specific cause of death

$$D_{x,x+n}^i = numbre\ of decrements\ (observed\ deaths)\ from\ cause\ i\ in\ the\ age\ interval\ x\ to\ x+n$$

$$m_{x,x+n}^i = \frac{D_{x,x+n}^i}{P_{x,x+n}} = mortality\ rate\ from\ cause\ i\ between\ ages\ x\ and\ x+n$$

$$q_{x,x+n}^i = \frac{m_{x,x+n}^i}{m_{x,x+n}} = \frac{D_{x,x+n}^i}{D_{x,x+n}} = probability\ of\ dying\ from\ cause\ i\ between\ ages\ x\ and\ x+n$$

$$d_{x,x+n}^i = q_{x,x+n}^i \times l_x = number\ of\ deaths\ from\ cause\ i\ between\ ages\ x\ and\ x+n$$

$$R_{x,x+n}^i = proportion\ of\ deaths\ from\ cause\ i\ between\ ages\ x\ and\ x+n$$

$$p^i_{x,x+n} = \left[p_{x,x+n}\right]^{R^i_{x,x+n}} probability\ of\ surviving\ between\ ages\ x\ and\ x+n\ from\ casue\ i$$

$$l^i_{x,x+n} = l_{x,x+n} \times p^i_{x,x+n} = number\ of\ survivors\ between\ ages\ x\ and\ x+n, from\ cause\ i$$

$$a^i_{x,x+n} = average\ person - years\ lived\ in\ the\ interval\ by\ those\ dying\ in\ the\ interval, from\ cause\ i$$

For convention $a^i_{x,x+n} = 2.5$ for all ages from x=5, for x=0 and x=5 $a^i_{x,x+n}$was calculated with the correction showed in Preston *et al*., 2001 Chapter 4, section 4.3

$$L^i_{x,x+n} = n \times l^i_{x,x+n} + a^i_{x,x+n} \times d^i_{x,x+n} = person - years\ liven\ between\ ages\ x\ and\ x+n, from\ cause\ i$$

For the open-ended interval $L^i_{x,x+n} = \frac{l^i_{x,w}}{m^i_{x,w}}$ with *w* = the las age group, i.e. 75+ or 85+

$$T^i_{x,x+n} = \sum_{k=x}^{w} L^i_{x,x+n} = Person - years\ lived\ above\ age\ x, from\ casue\ i$$

$$e^{o,i}_x = \frac{T^i_{x,x+n}}{l^i_{x,x+n}} = expectation\ of\ life\ at\ age\ x, from\ cause\ i$$

5. *Temporary Life Expectancy*

We used the method or formula described by Arriaga 1894. For simplicity we present just the general formula, but it can be applied to a specific cause of death.

$$e_{x,j} = \frac{T_x - T_{x+j}}{l_x} = temporary\ life\ expectancy\ between\ ages\ x\ and\ x+j$$

6. *Potential Years of Life Lost*

We based this section upon the work of Andersen et all 2013, but we approximate their methodology using discrete approximations. We use notation previously defined in sections 2 through 4 of this appendix.

$$\text{ך}_{x,x+n} = \frac{d^i_{0,x*}}{d_{0,x*}} \times \frac{n}{2} \times \left(l_x - {}^{l_{x+n}}/_{l_0}\right) + n \times \left(1 - {}^{l_x}/_{l_0}\right) \times \frac{d^i_{x,x+n}}{d_{x,x+n}} = potential\ yeras\ of\ life\ lost\ between\ ages\ x\ and\ a+n, from\ specific\ cause\ i$$

Where the interval *0, x** means from zero until *x* but excluding this point, or in other words, all events before age *x*.

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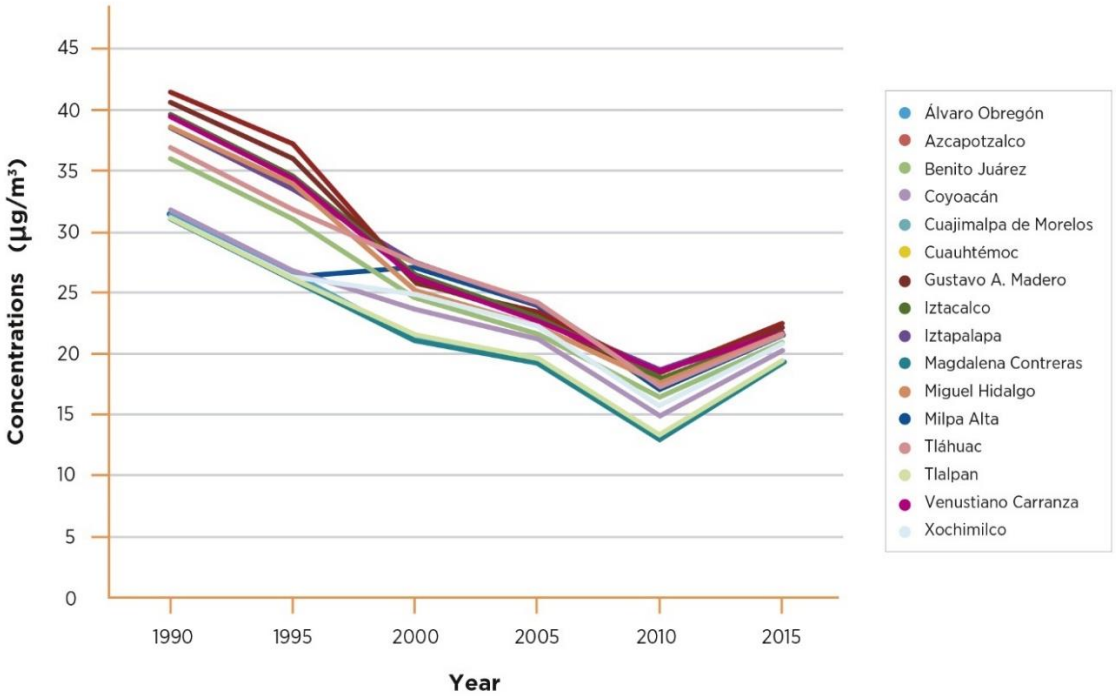
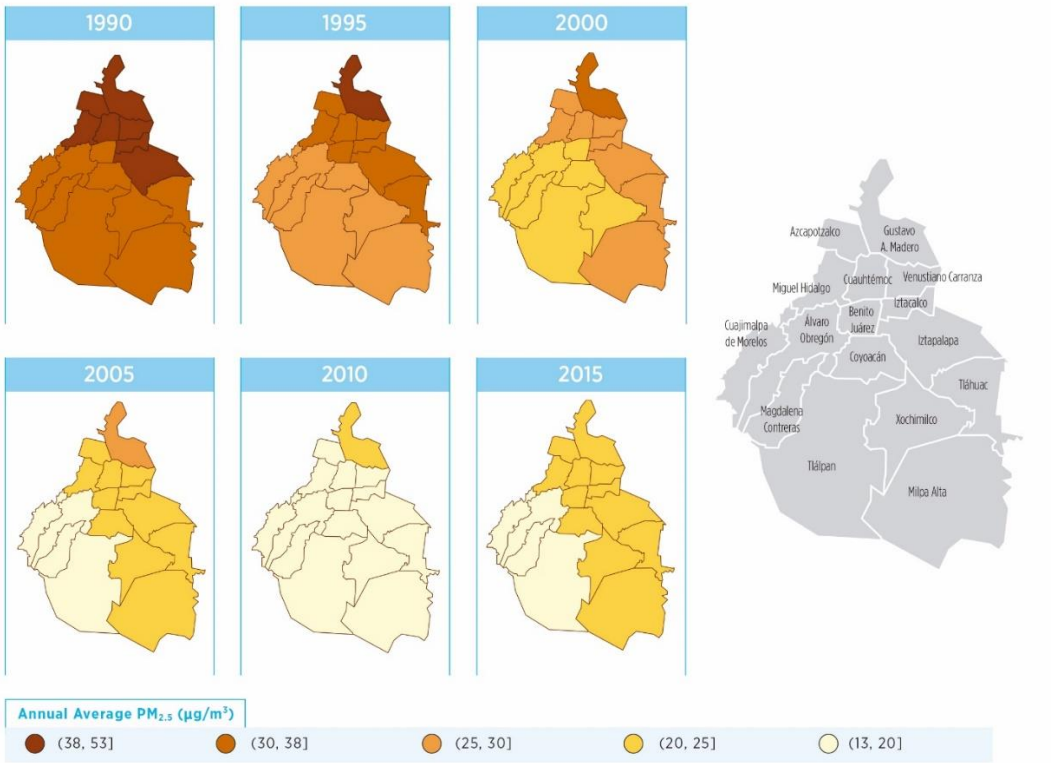
APPENDIX II. AIR POLLUTION

Fine Particles ($PM_{2.5}$) Annual Mean

II.1. Time trends of alcaldía-*specific annual average $PM_{2.5}$ concentrations ($\mu g/m^3$)*

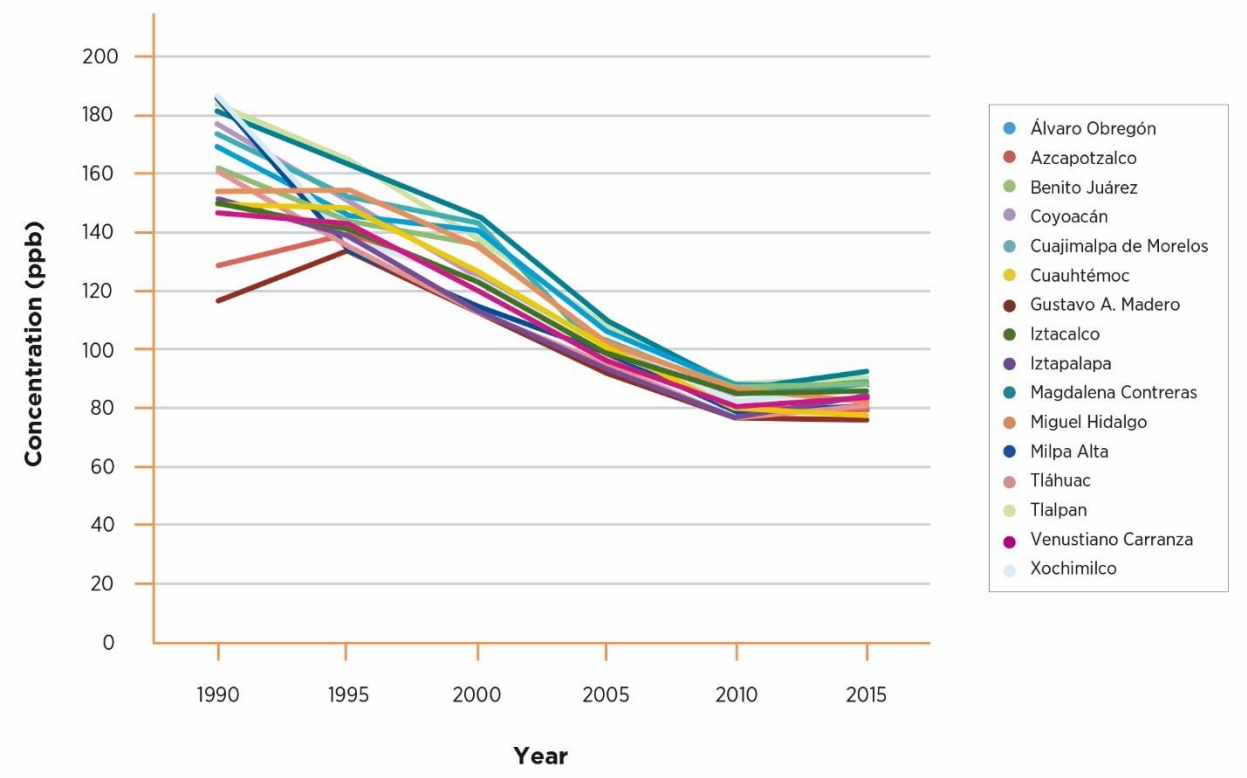


II.2. Spatial distribution of alcaldía-*specific annual average $PM_{2.5}$ concentrations ($\mu g/m^3$) by year*

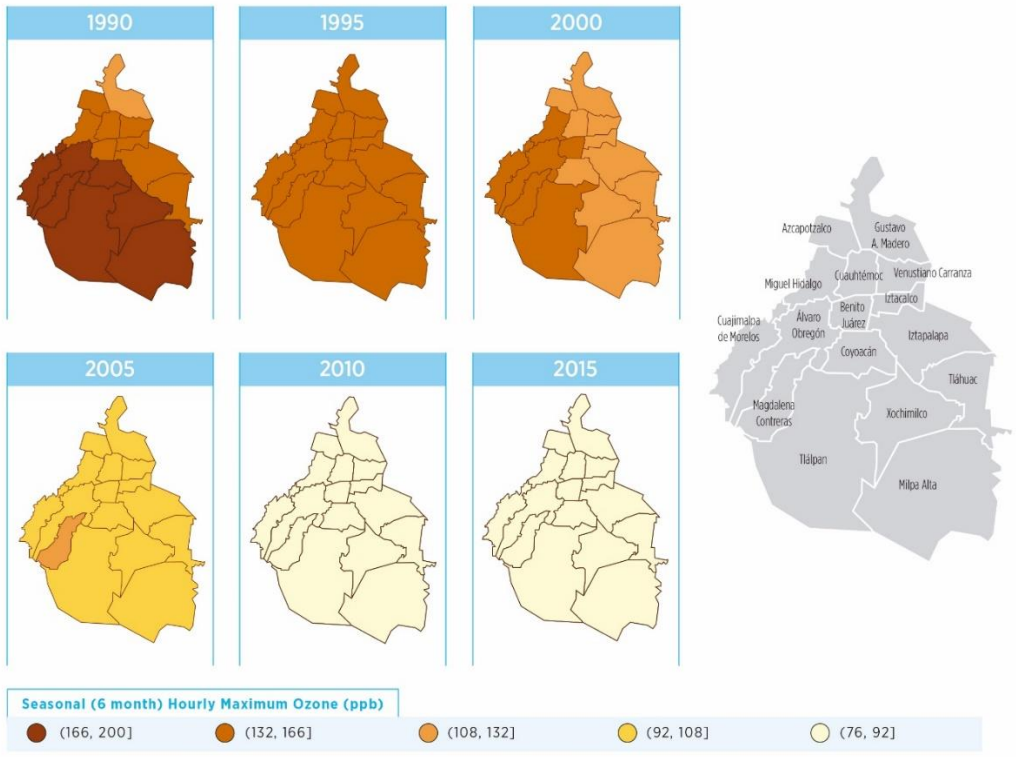


Ozone (O_3) Seasonal 1-Hour Maximum

II.3. Time trends of alcaldía-*specific seasonal (6 month) 1-hour maximum ozone concentrations (ppb)*



II.4. Spatial distribution of alcaldía-*specific seasonal (6 month) 1-hour maximum ozone concentrations (ppb) by year*



APPENDIX III. LIFE EXPECTANCY

Life Expectancy at Birth

Fig. III.1. Time trends of alcaldía*-specific population life expectancy at birth (years)*

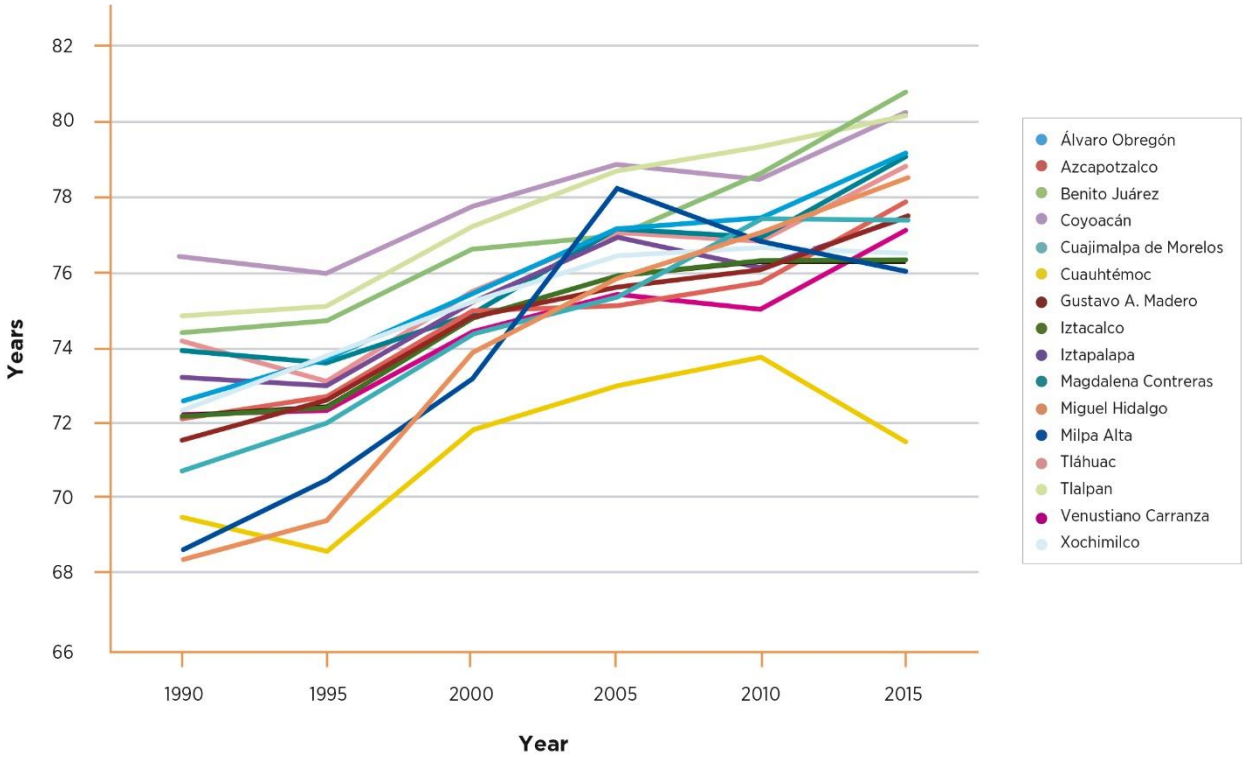
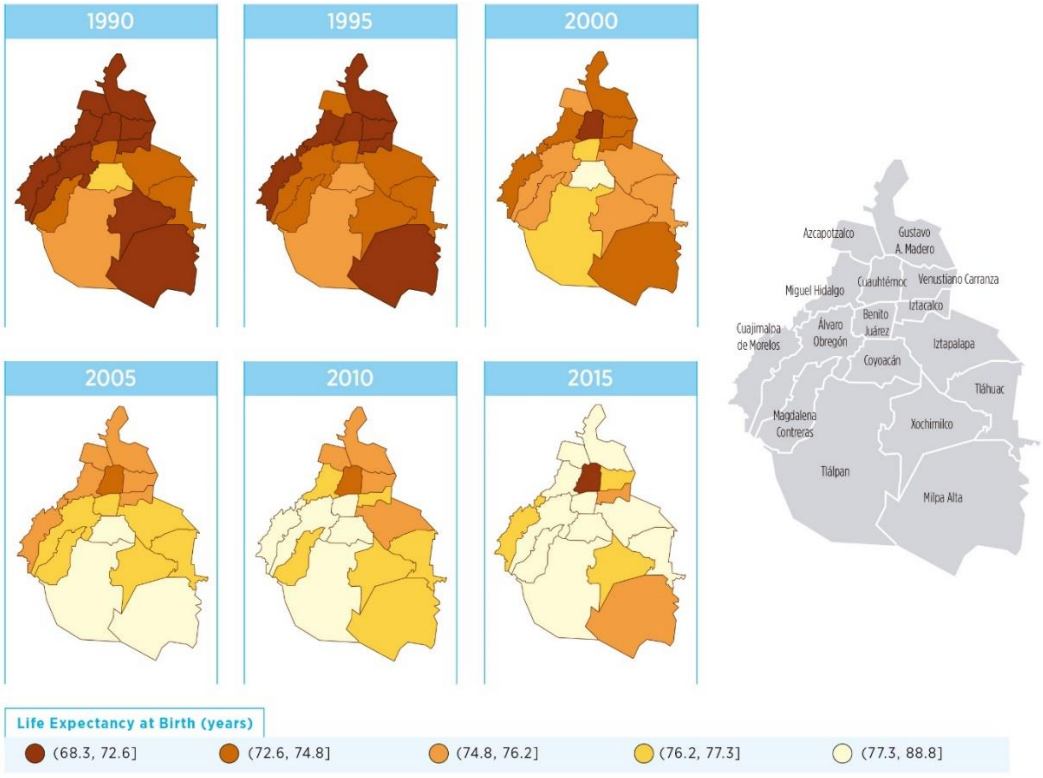


Fig. III.2. Spatial distribution of alcaldía*-specific life expectancy at birth (years) by year*



Life Expectancy for Men

Fig. III.3. Time trends of alcaldía*-specific population life expectancy at birth (years) for men*

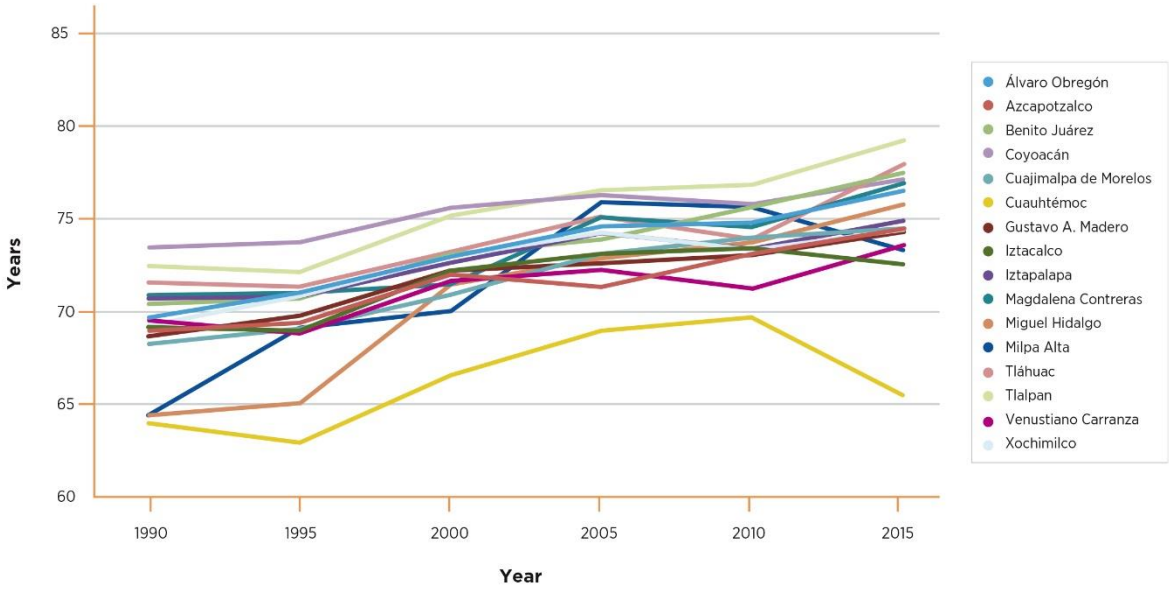
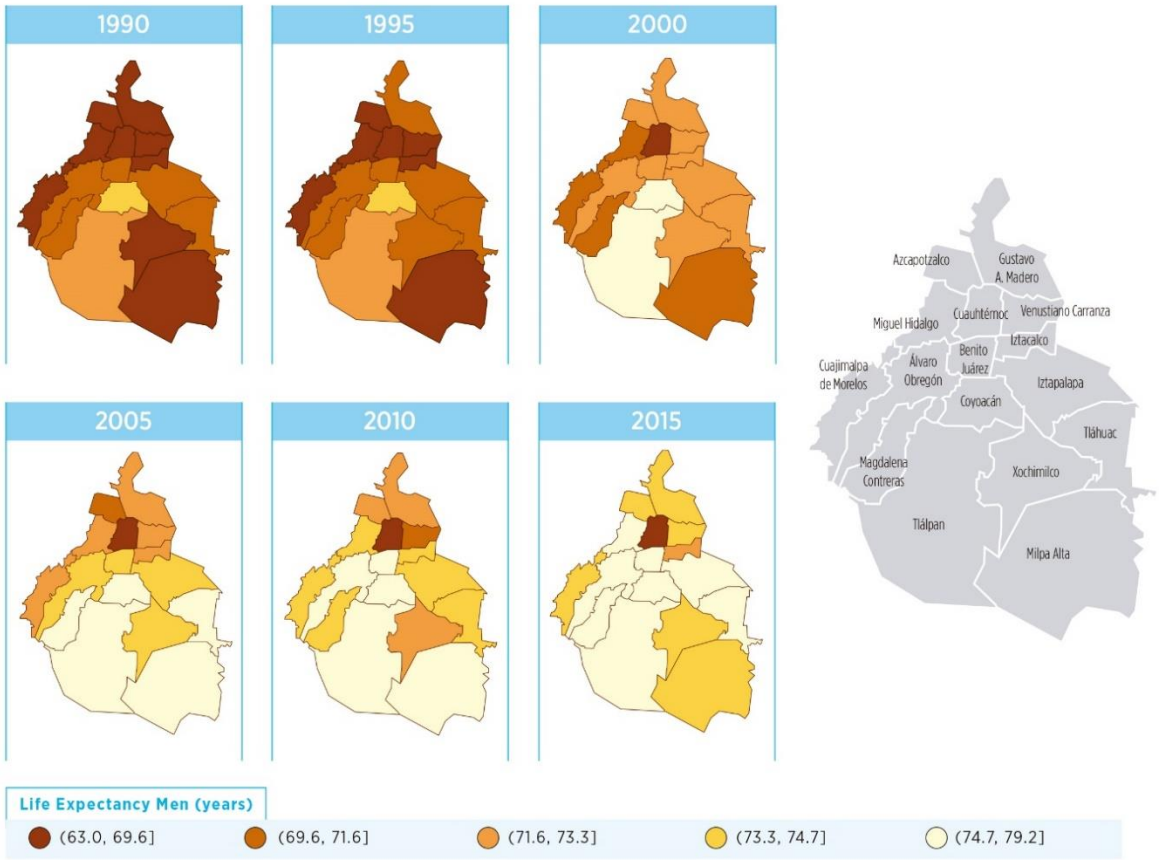


Fig. III.4. Spatial distribution of alcaldía*-specific life expectancy at birth (years) by year for men*



Life Expectancy for Women

Fig. III.5. Time trends of alcaldía-*specific population life expectancy at birth (years) for women*

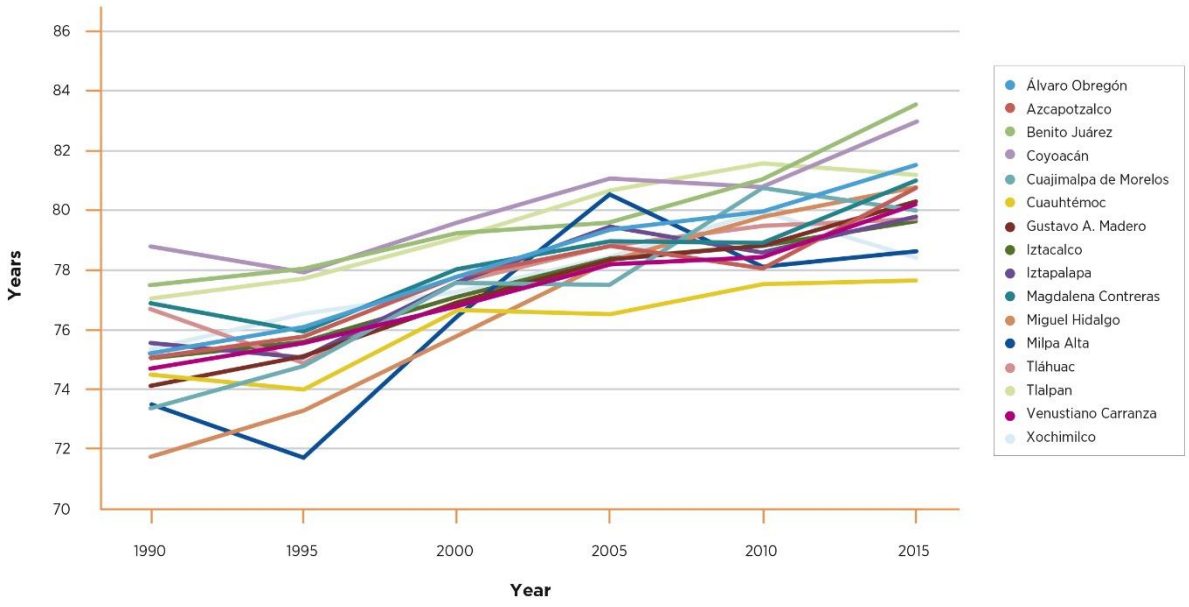
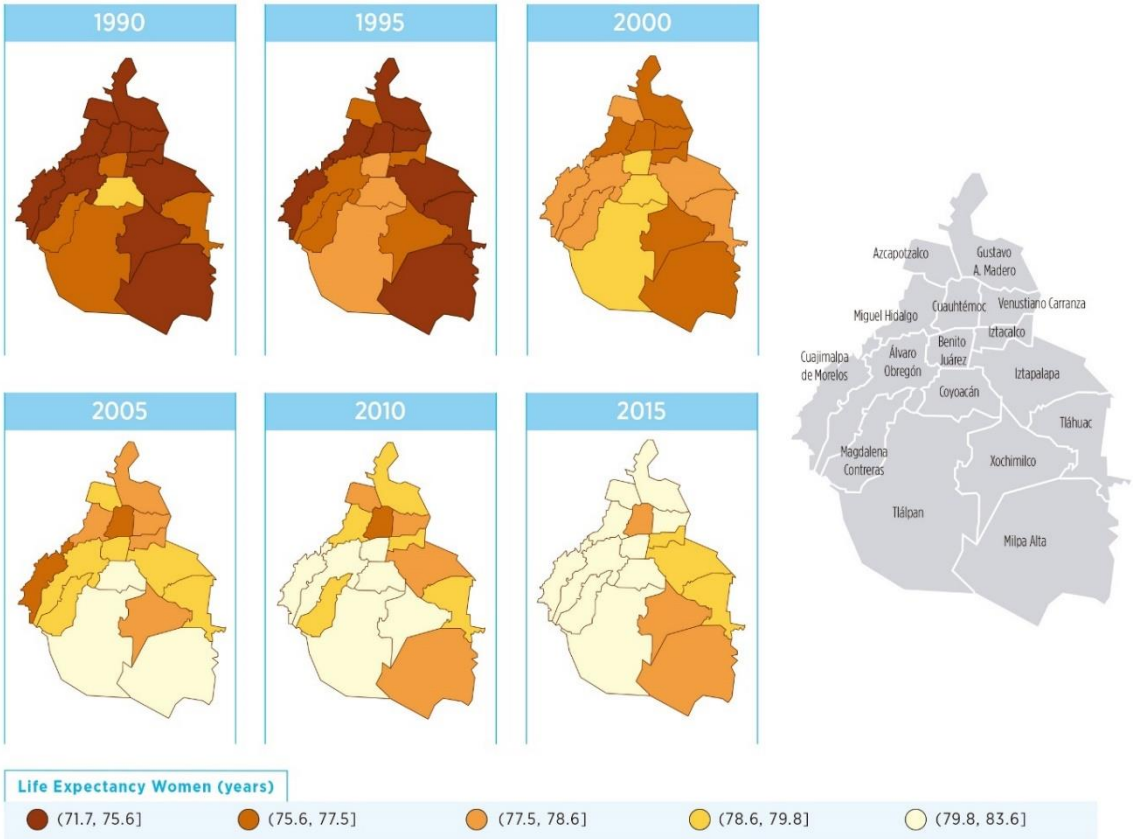


Fig. III.6. Spatial distribution of alcaldía-*specific life expectancy at birth (years) by year for women*



Temporary Life Expectancy (0-4 years)

Fig. III.7. Time trends of alcaldía*-specific population temporary life expectancy (years) for population between 0 to 4 years old*

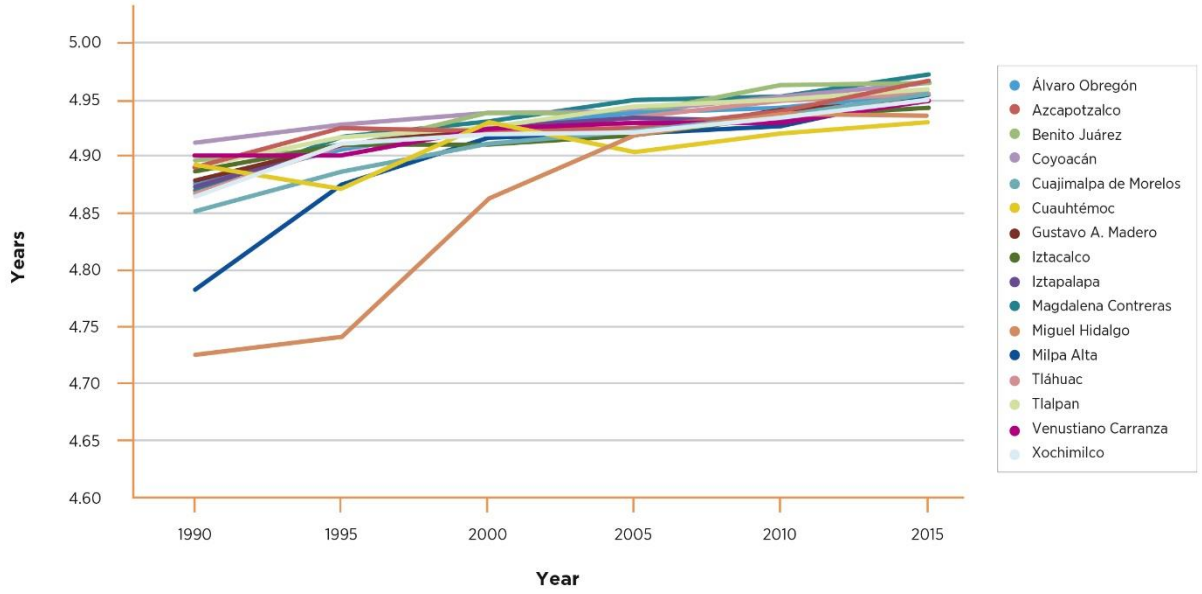
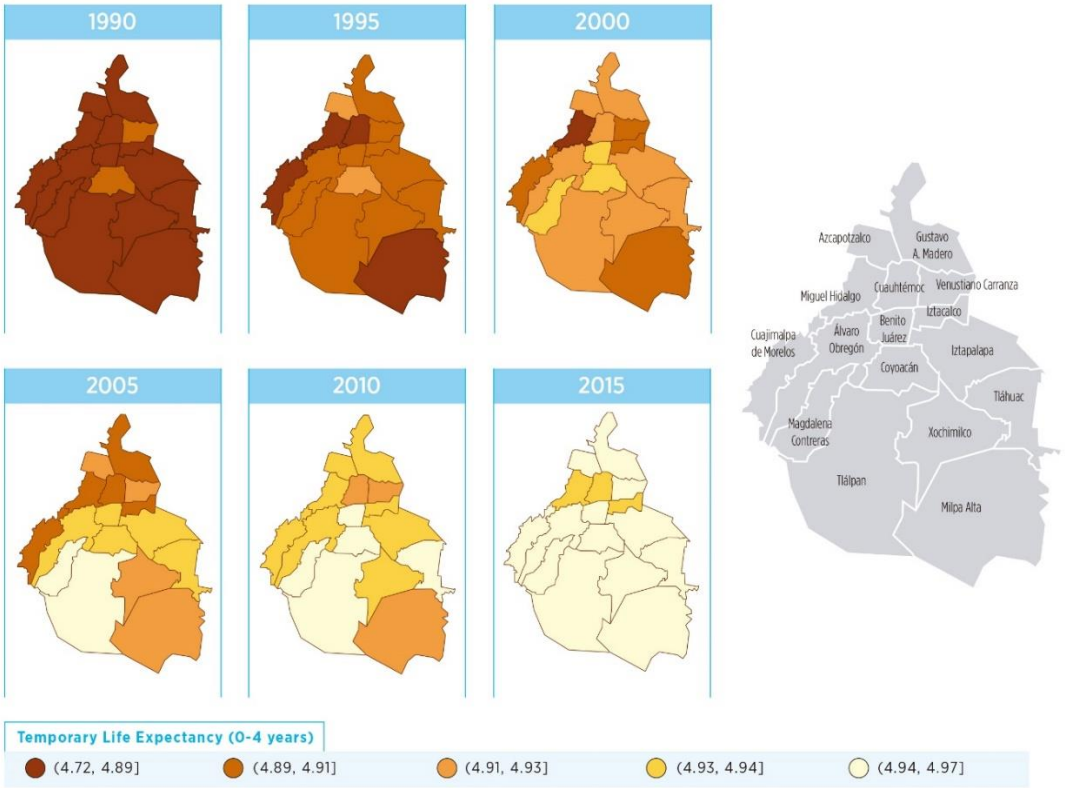


Fig. III.8. Spatial distribution of alcaldía*-specific temporary life expectancy (years) for population between 0 to 4 years old by year*



Temporary Life Expectancy (25-74 years)

Fig. III.9. Time trends of alcaldía*-specific temporary life expectancy (years) for population between 25 to 74 years old*

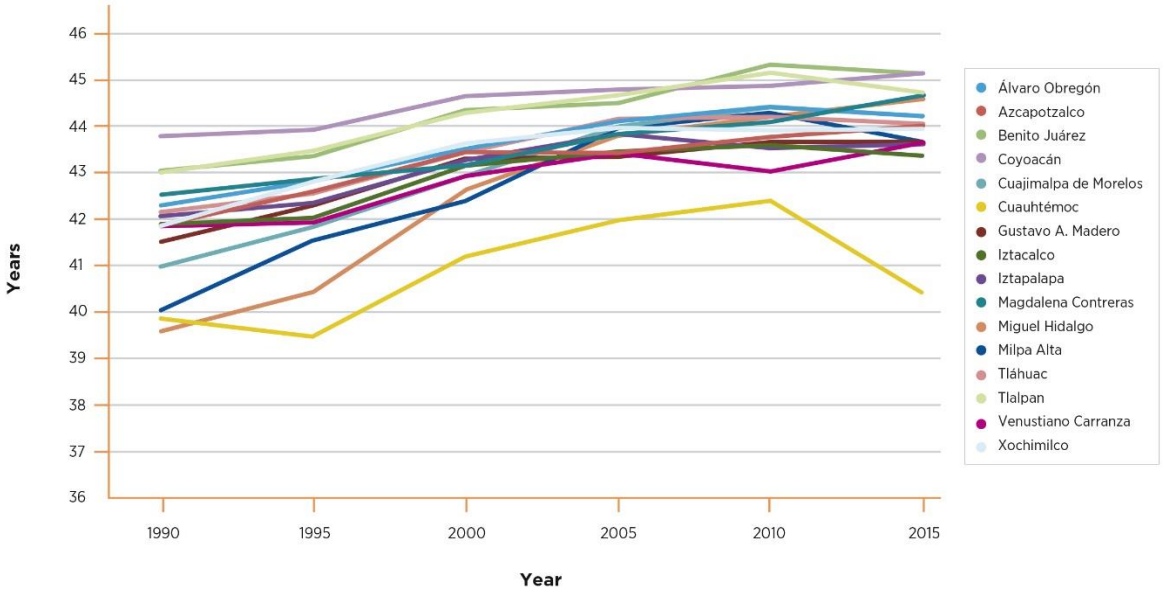
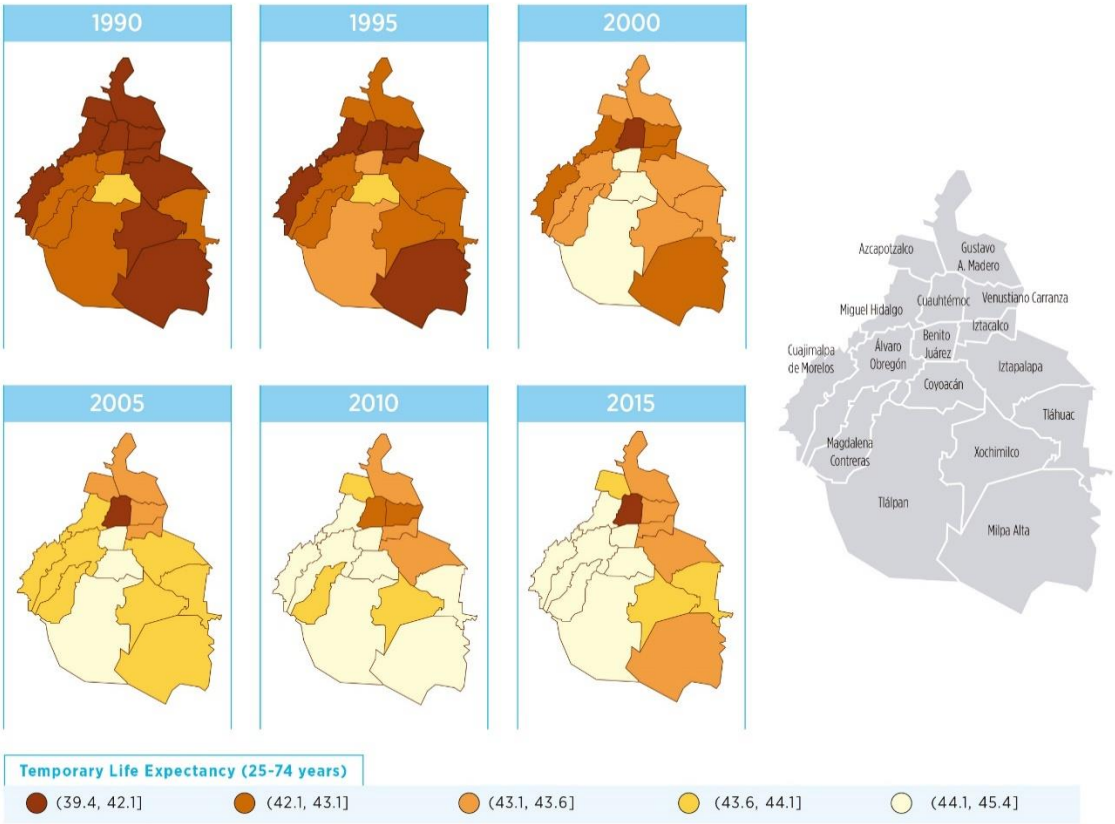


Fig. III.10. Spatial distribution of alcaldía*-specific temporary life expectancy (years) for population between 25-74 years old by year*



APPENDIX IV. SMOKING RELATED DISEASES

Chronic Obstructive Pulmonary Disease (≥25 years)

Fig. IV.1. Time trends of alcaldía*-specific chronic obstructive pulmonary disease mortality rates (deaths per 100,000)*

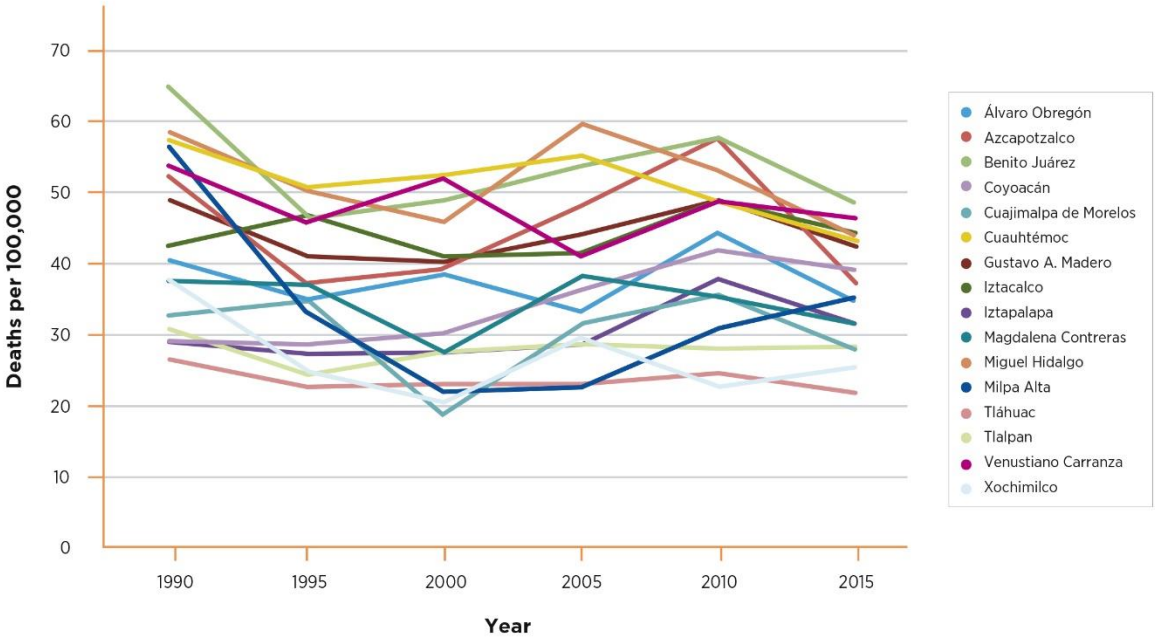
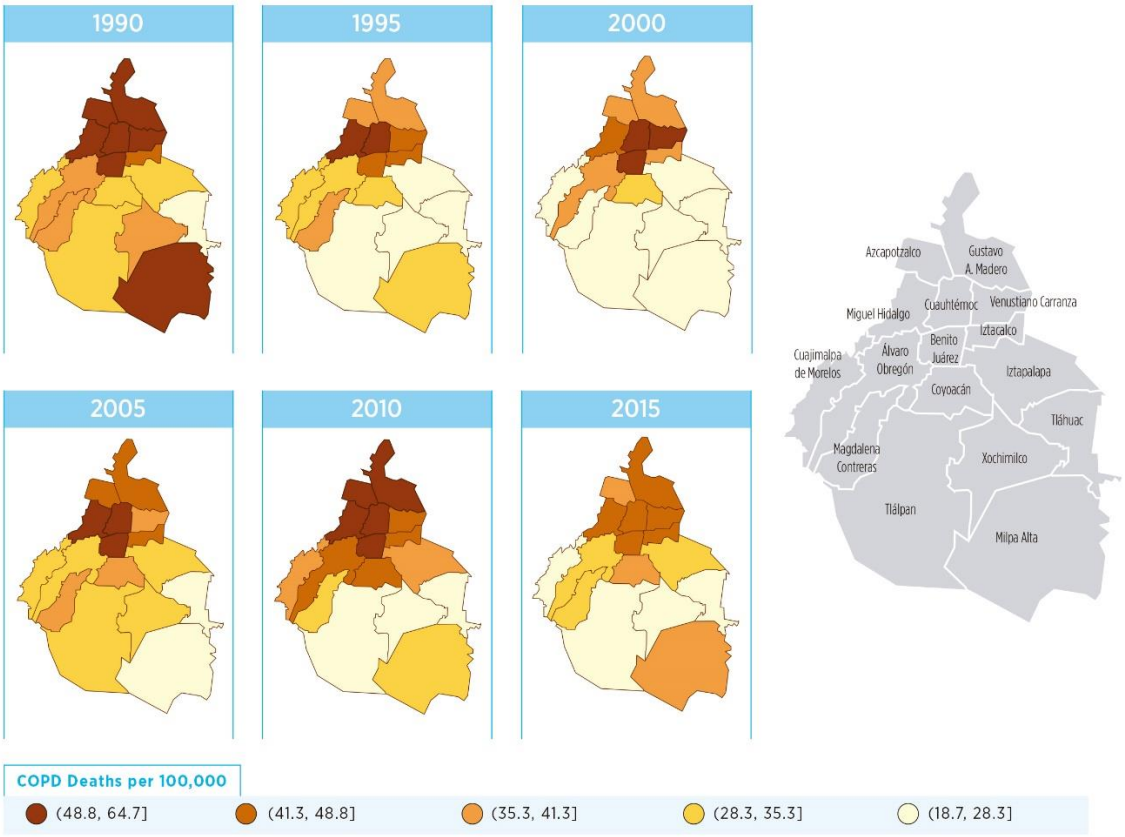


Fig. IV.2. Spatial distribution of alcaldía*-specific COPD mortality rates (deaths per 100,000) by year*



Lung Cancer (≥25 years)

Fig. IV.3. Time trends of alcaldía*-specific lung cancer mortality rates (deaths per 100,000)*

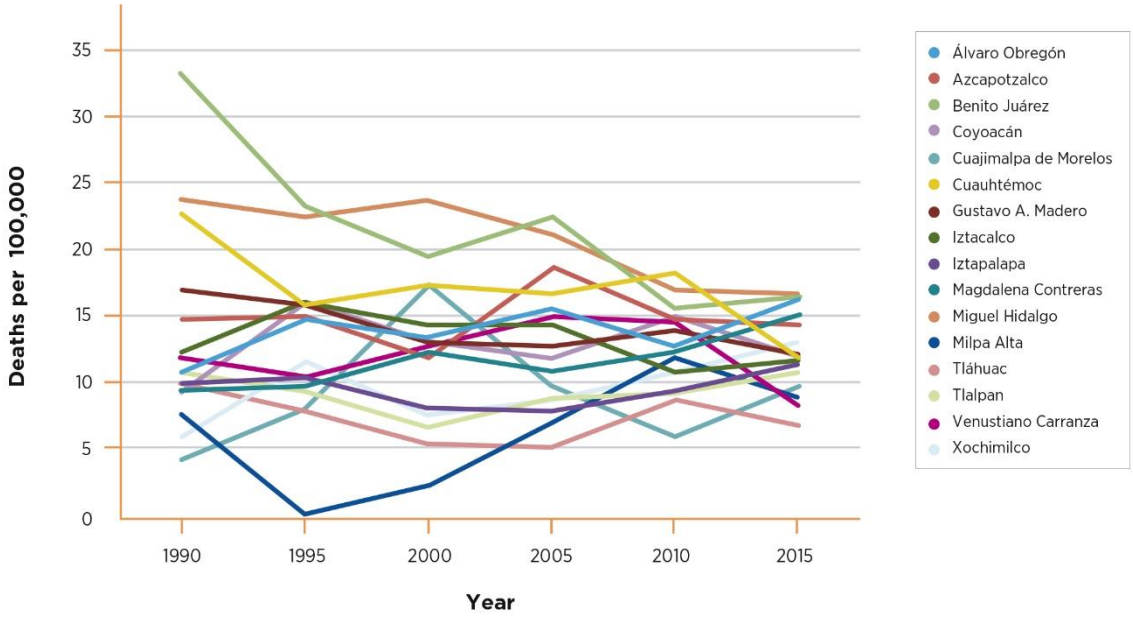
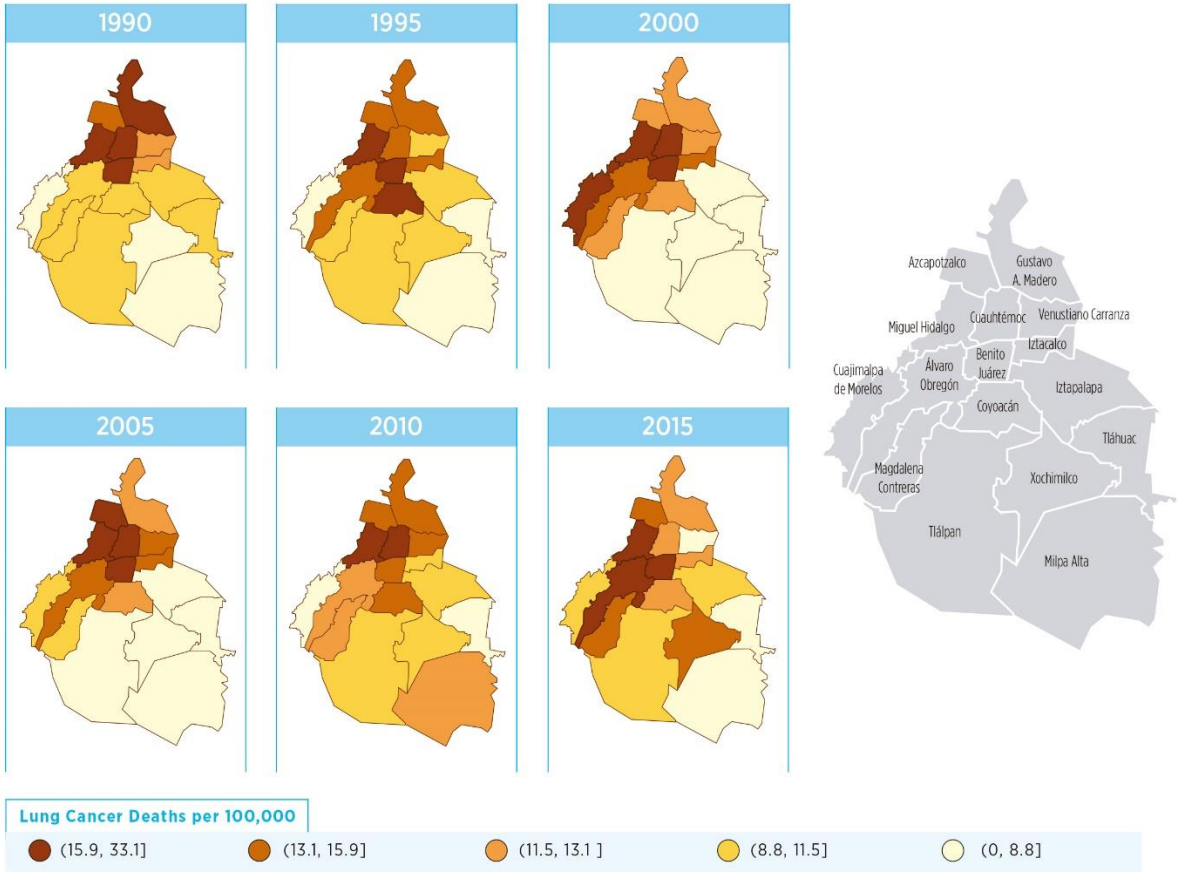


Fig. IV.4. Spatial distribution of alcaldía*-specific lung cancer mortality rates (deaths per 100,000) by year*



Smoking Prevalence & National Surveys

ENSA, 2000 was a household probabilistic survey, with stratified multistage sample conglomerate selection. This survey had national and state-level representativeness (Sepúlveda et al., 2007). Its successor, ENSANUT, 2006 improved the sampling scheme, allowing to estimate indicators with urban and rural areas' representativeness in each state (Abúndez et al., 2006). ENSANUT 2012 added the metropolitan stratum; for Mexico City all households were classified as metropolitan (Romero-Martínez et al., 2012). ENSA and ENSANUT included adults 20 years and older.

ENA, 2011 and ENA, 2016 were applied to household residents, aged between 12 and 65 years old throughout the country. Sociodemographic data and information on tobacco, alcohol and another drugs' use were collected. ENA, 2011 and 2016 have a probabilistic design, stratified with multistage sample conglomerate selection: the basic geostatistical area (known as an AGEB) within an *alcaldía*, blocks or segments, households and individuals. These two surveys were designed to have national and regional representativeness. Mexico City was one of the regions in which the country was divided (Villatoro et al., 2012, Secretaría de Salud, 2016).

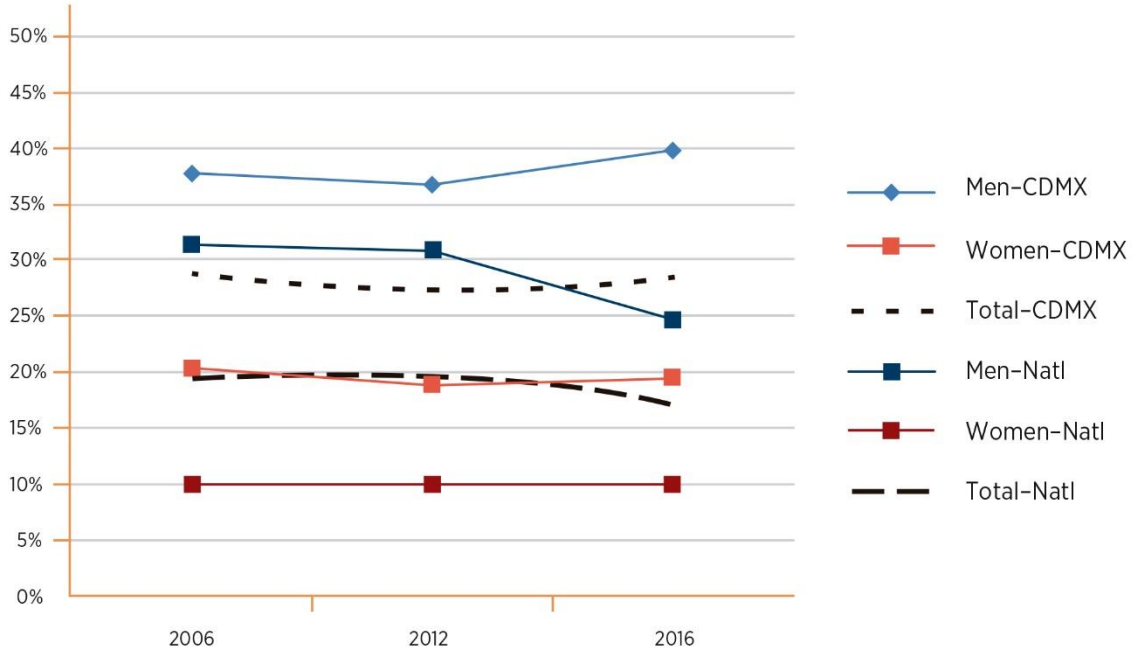
Table IV.1. Smoking Prevalence in Mexico City by Sex and Age-Group (Adults 25-64 years) 2000, 2006, 2011, 2012 and 2016

Age-Group	Women		Men		Total Population	
	%	n	%	n	%	n
ENSA 2000						
25 - 29	19.7	115	58.9	44	26.4	159
30 - 34	29.3	110	46.1	39	31.9	149
35 - 39	18.3	118	39.0	51	23.0	169
40 - 44	27.6	91	61.6	38	38.7	129
45 - 49	16.9	76	30.9	32	20.1	108
50 - 54	23.1	68	44.8	26	27.6	94
55 - 59	28.3	42	35.0	25	30.4	67
60 - 64	8.7	46	13.7	25	10.2	71
25 and older	21.9	666	44.2	280	27.0	946
ENSANUT 2006						
25 - 29	16.9	84	35.6	58	26.1	142
30 - 34	22.0	93	40.9	79	30.2	172
35 - 39	21.5	78	41.7	68	31.7	146
40 - 44	26.3	69	38.2	55	31.4	124
45 - 49	24.2	54	41.0	64	33.4	118
50 - 54	18.1	55	25.5	50	21.6	105
55 - 59	14.0	41	37.9	44	27.4	85
60 - 64	13.4	31	41.7	24	27.3	55
25 and older	20.3	442	37.7	505	28.8	947
ENA 2011						
25 - 29	28.2	84	35.3	73	31.5	157
30 - 34	18.8	73	32.9	73	26.5	146
35 - 39	12.0	86	44.8	72	28.6	158
40 - 44	12.3	72	29.8	62	20.1	134
45 - 49	24.6	58	37.1	42	29.0	100
50 - 54	20.4	72	30.3	52	24.7	124
55 - 59	11.2	56	39.0	44	24.8	100
60 - 64	10.3	53	25.5	50	18.4	103
25 and older	18.3	554	34.8	468	26.1	1022
ENSANUT 2012						
25 - 29	15.4	59	40.4	54	28.1	113
30 - 34	34.8	88	36.1	58	35.4	146
35 - 39	8.5	99	41.2	65	22.1	164
40 - 44	18.1	93	40.0	59	27.1	152
45 - 49	21.9	76	46.2	67	34.6	143
50 - 54	7.1	60	33.5	46	18.9	106
55 - 59	23.8	56	27.3	47	25.5	103
60 - 64	15.6	48	22.5	47	19.1	95
25 and older	18.8	579	36.9	443	27.2	1022
ENA 2016						
25 - 29	26.2	94	42.0	78	33.3	172
30 - 34	23.0	88	53.1	70	37.0	158
35 - 39	20.1	87	39.8	73	30.1	160
40 - 44	25.7	97	36.5	66	30.2	163
45 - 49	18.9	86	35.2	75	27.1	161
50 - 54	16.7	77	37.3	65	27.1	142
55 - 59	5.8	89	35.0	59	19.6	148
60 - 64	14.0	127	32.9	53	20.1	180
25 and older	19.4	745	39.8	539	28.7	1284

Sources: ENSA, 2000; ENSANUT, 2006 and 2012; ENA, 2011 and 2016.

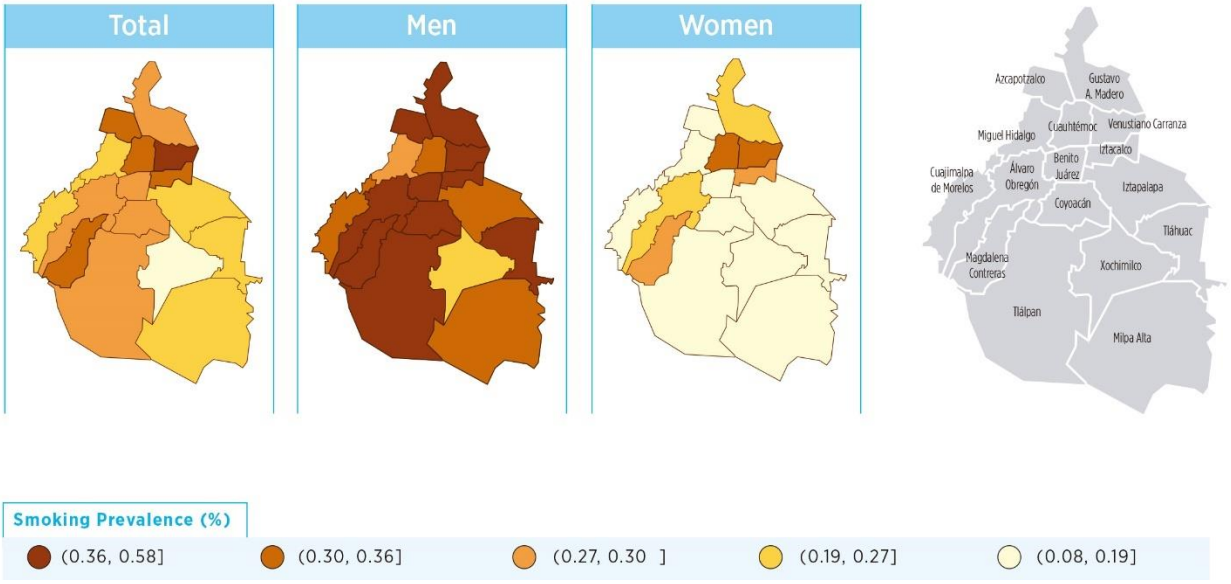
Smoking (Adults 25-64 years)

Fig. IV. 5. Smoking prevalence (%) in adults aged 25 to 64 years in Mexico and in Mexico City for 2006, 2012 and 2016



Sources: ENSANUT, 2006 and 2012; ENA, 2016.

Fig. IV.6. Spatial distribution of alcaldía*-specific smoking prevalence (%) in adults aged 25 to 64 years stratified by sex (Aggregated data from 2000, 2006 and 2012)*



Sources: ENSA, 2000; ENSANUT, 2006 and 2012.

APPENDIX V. SOCIOECONOMIC POSITION INDICATORS

Illiteracy (≥15 years) (ANALF)

Fig. V.1. Time trends of alcaldía*-specific illiterate population aged 15 years and above (%)*

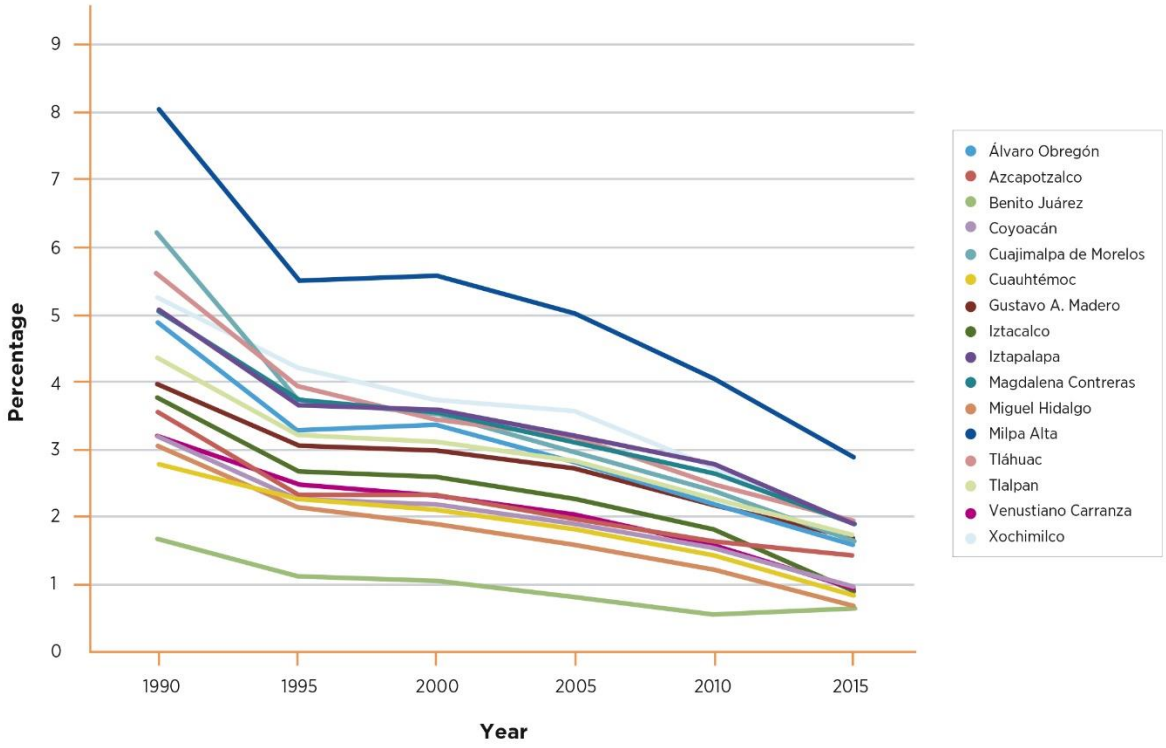
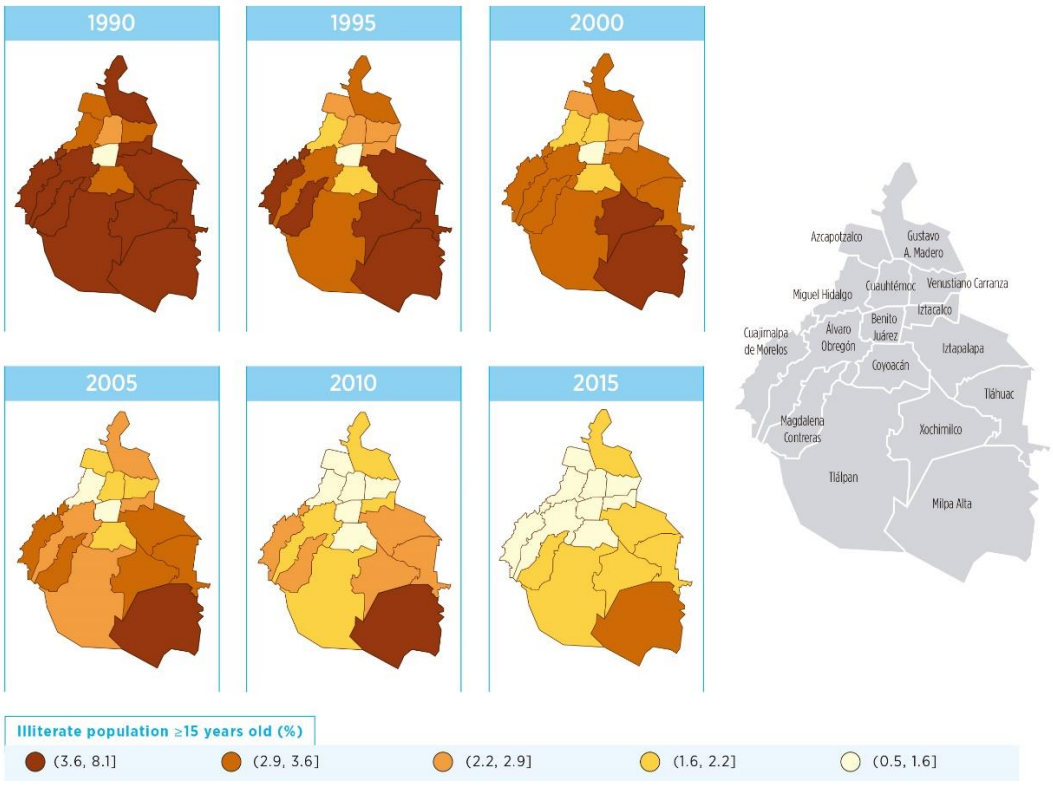


Fig. V.2. Spatial distribution of *alcaldía*-specific illiterate population aged 15 years and above (%) by year



Low Education (≥15 years) (SPRIM)

Fig. V.3. Time trends of alcaldía-*specific population aged 15 years and above without complete primary school education (%)*

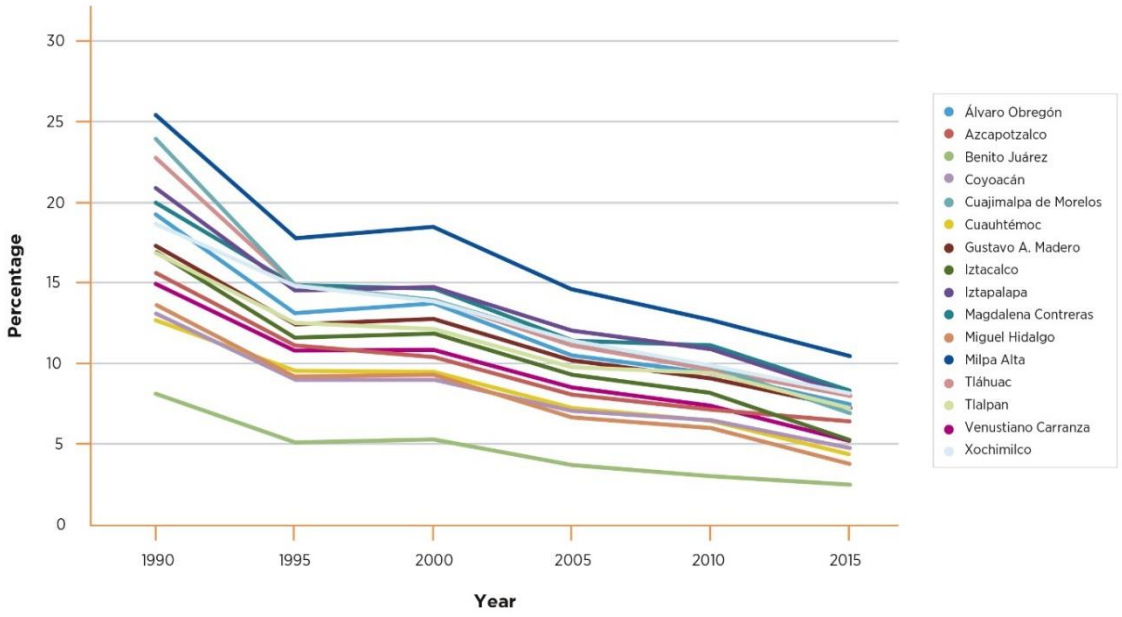
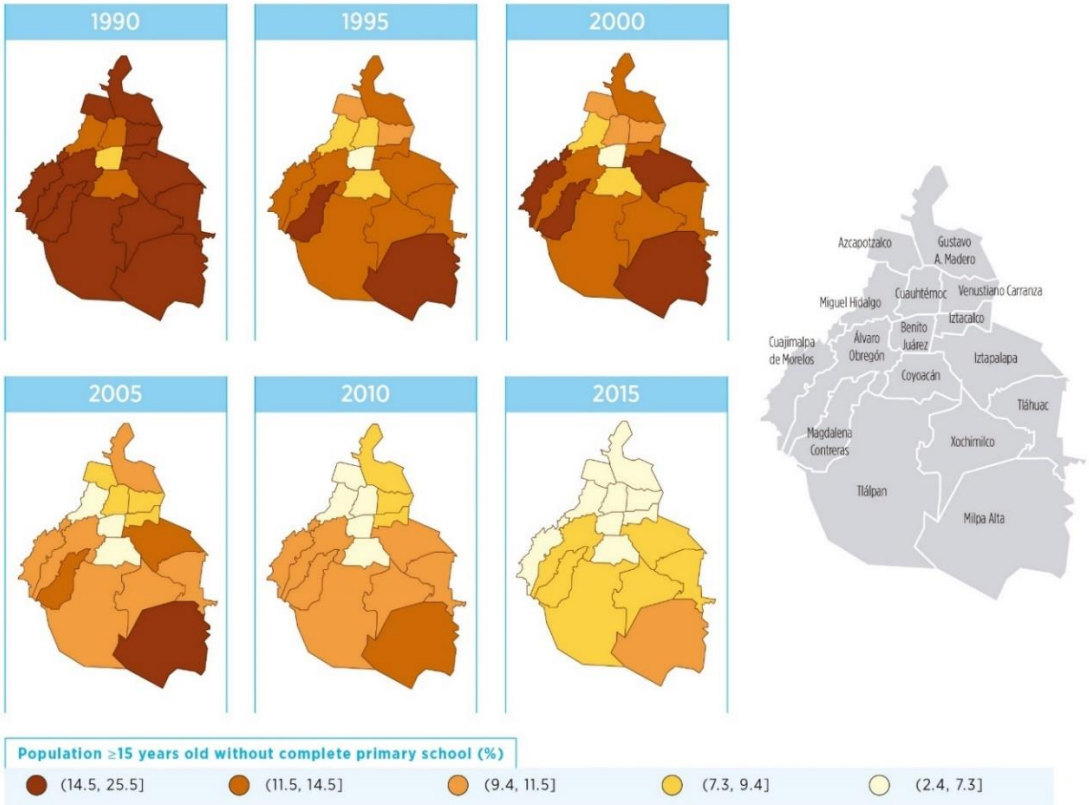


Fig. V.4. Spatial distribution of alcaldía-*specific population aged 15 years and above without complete primary school education (%) by year*



No Sewer nor Toilet (OVSDE)

Fig. V.5. Time trends of alcaldía-*specific occupants in households without sewer nor toilet (%)*

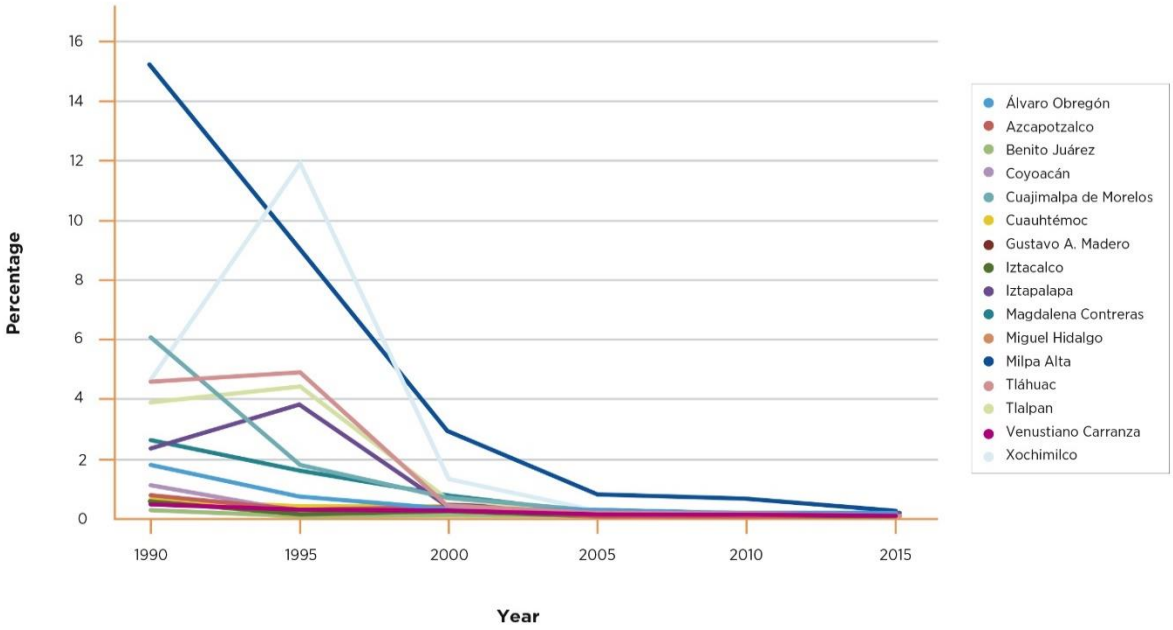
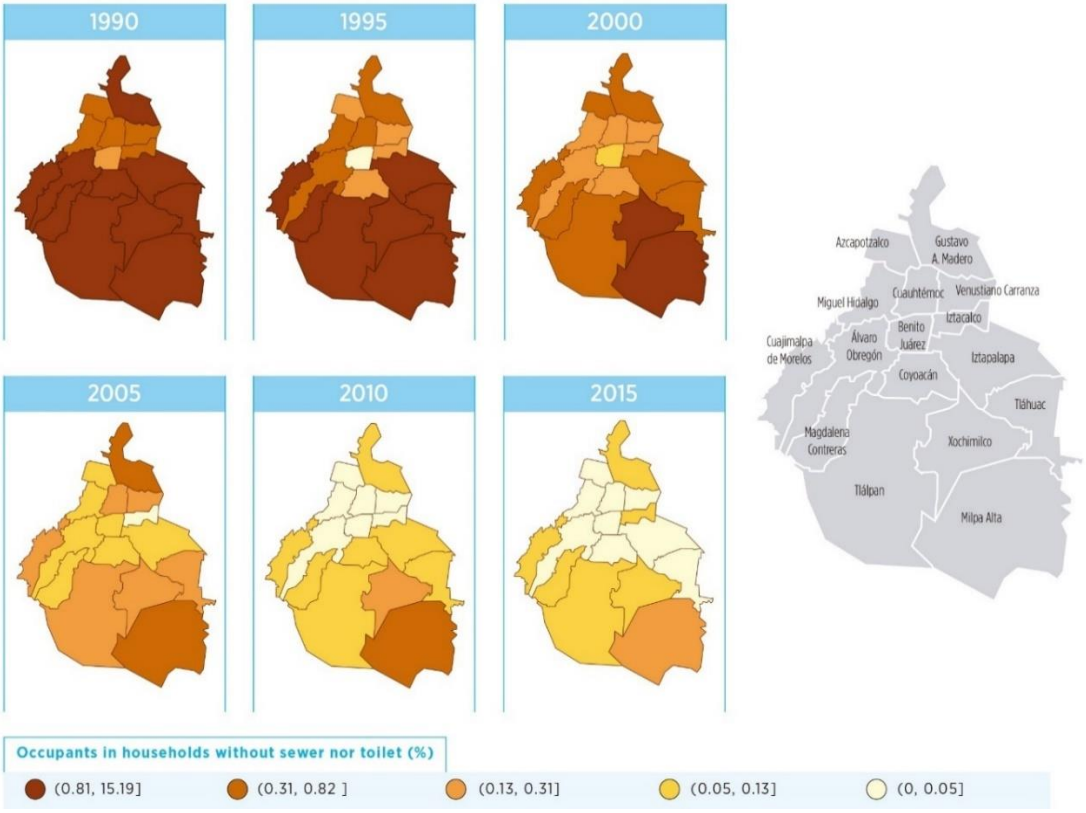


Fig. V.6. Spatial distribution of alcaldía-*specific occupants in households without sewer nor toilet (%) by year*



No Electricity (OVSEE)

Fig. V.7. Time trends of alcaldía*-specific occupants in households without electricity (%)*

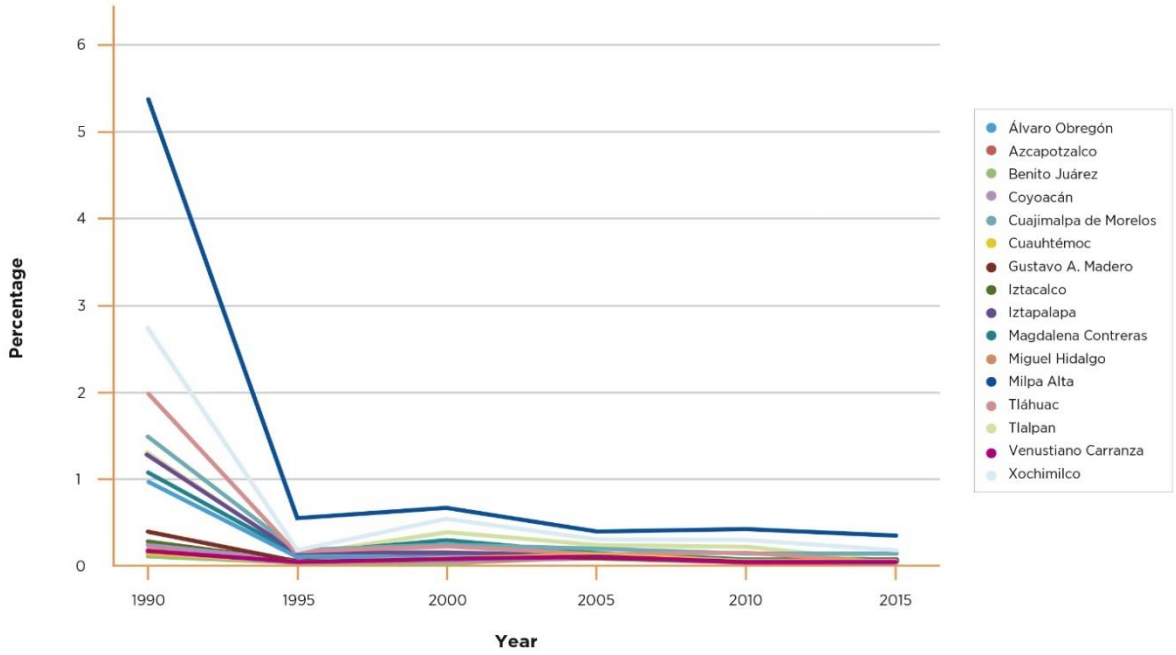
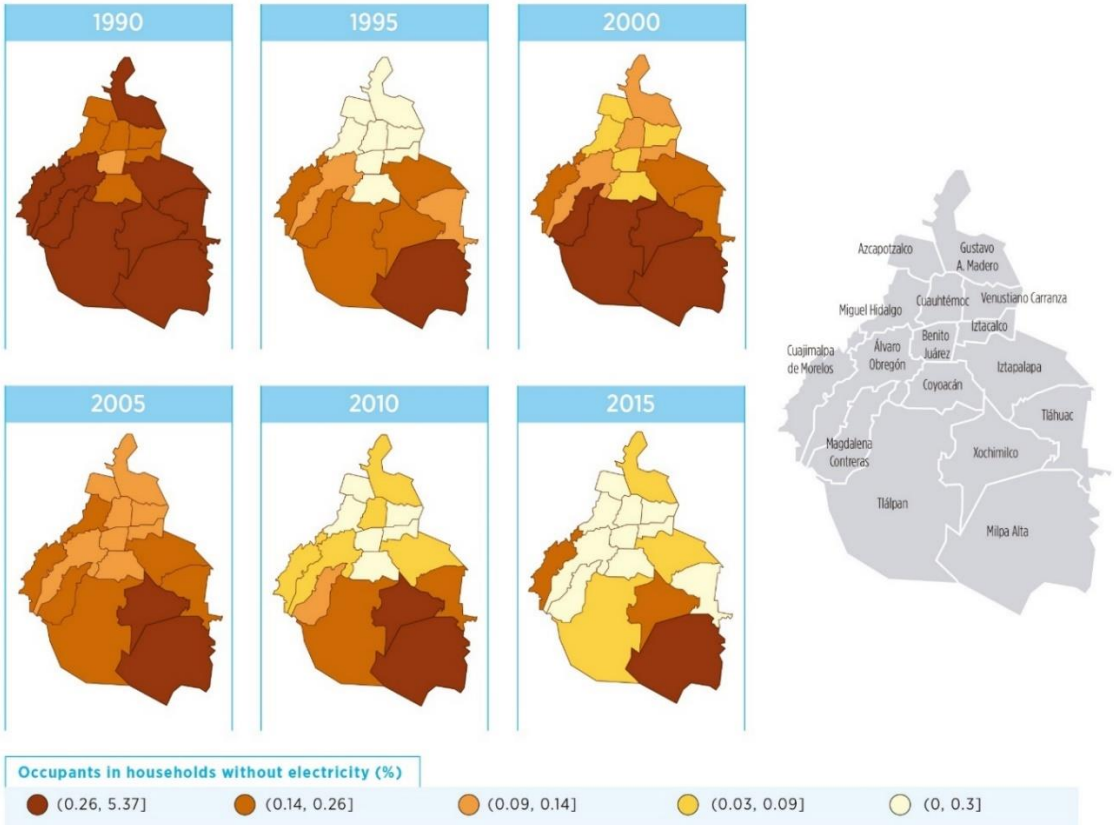


Fig. V.8. Spatial distribution of alcaldía*-specific occupants in households without electricity (%) by year*



No Running Water (OVSAE)

Fig. V.9. Time trends of alcaldía*-specific occupants in households without running water (%)*

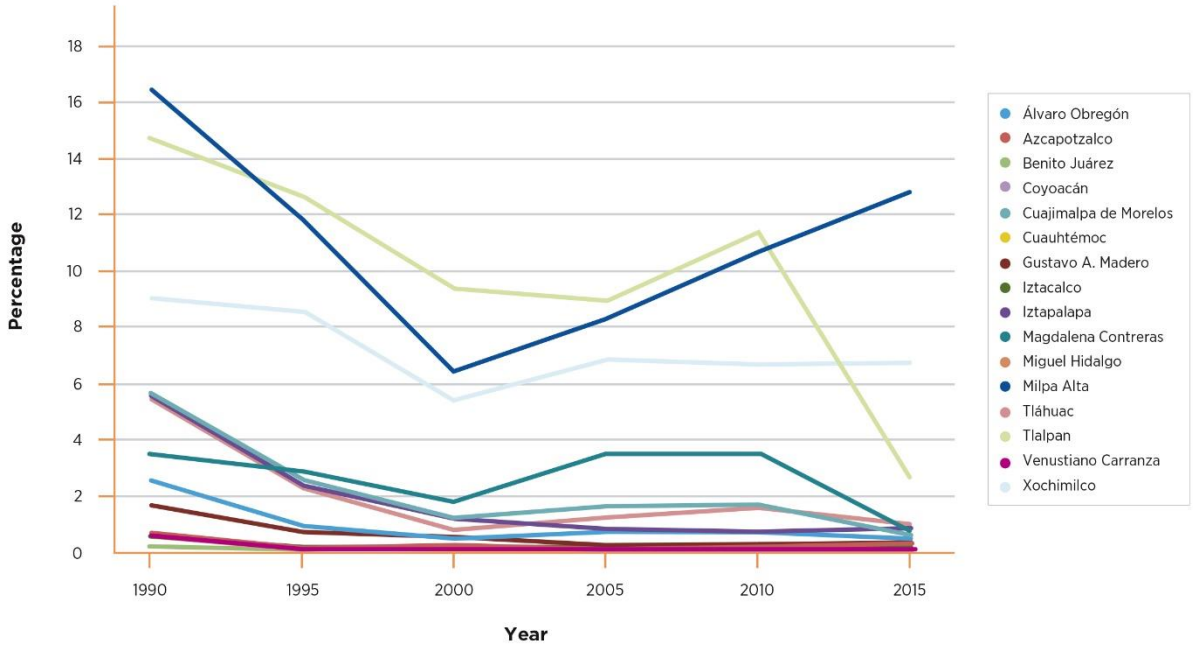
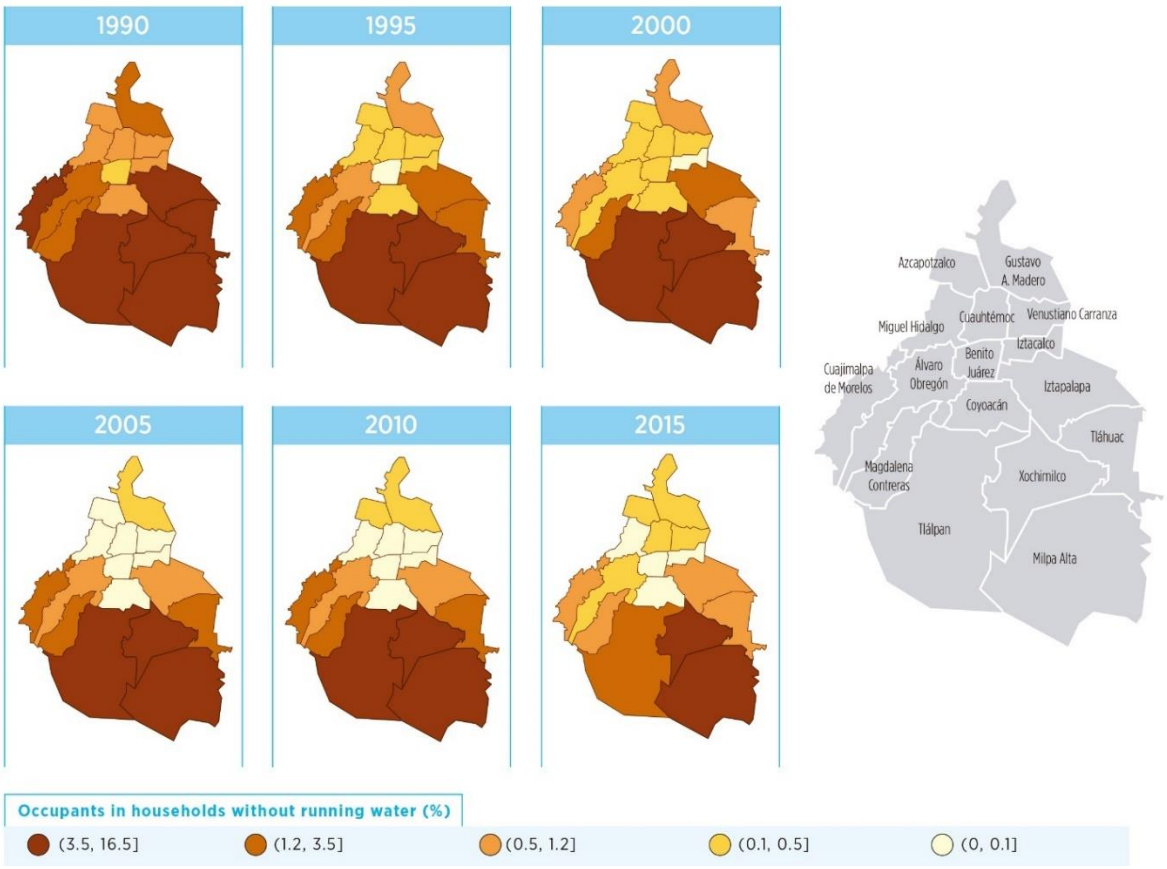


Fig. V.10. Spatial distribution of alcaldía*-specific occupants in households without running water (%) by year*



Overcrowding (VHAC)

Fig. V.11. Time trends of alcaldía*-specific households with some degree of overcrowding (%)*

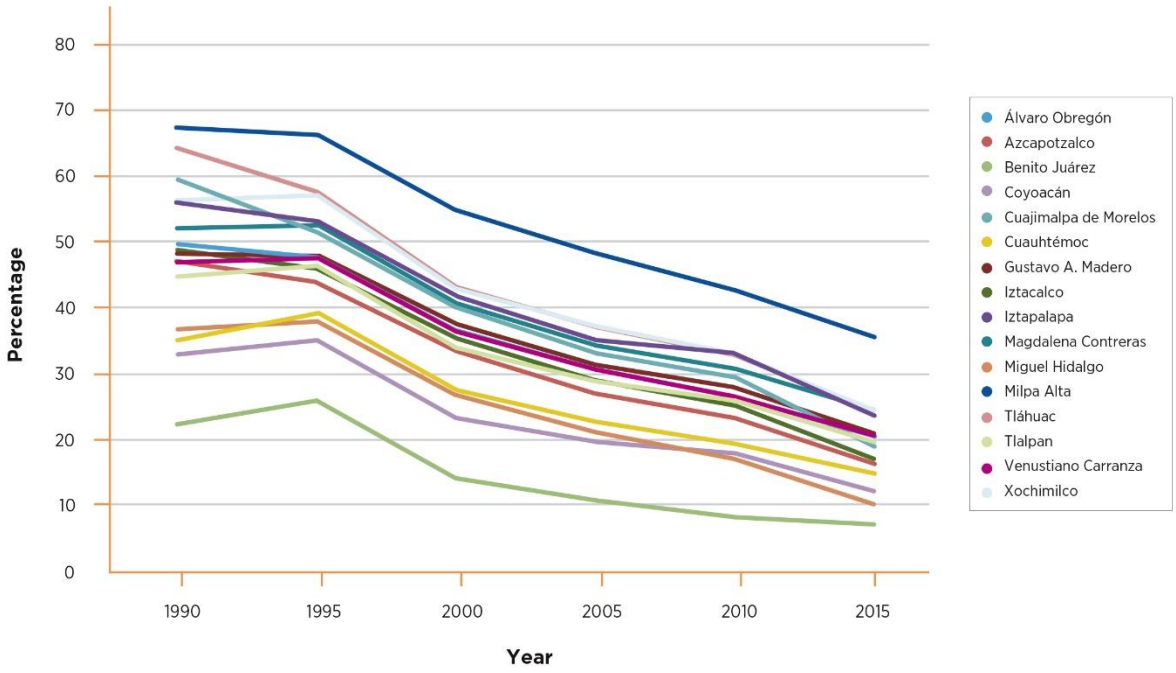
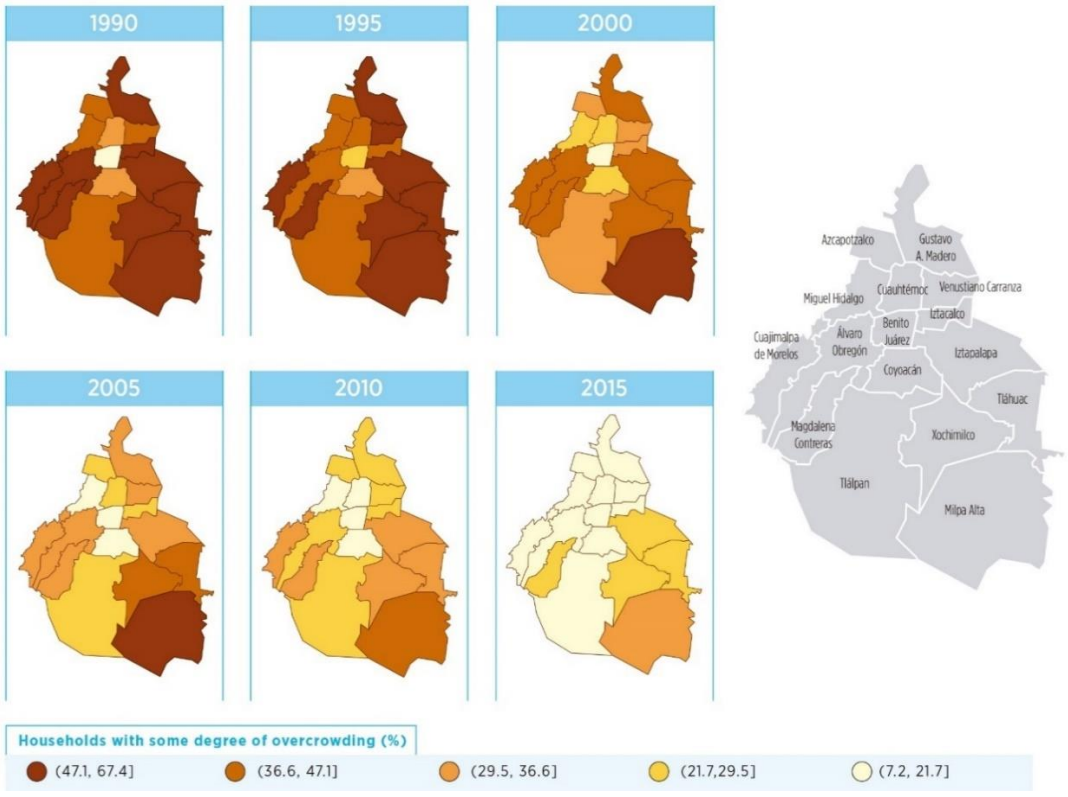


Fig. V.12. Spatial distribution of alcaldía*-specific households with some degree of overcrowding (%) by year*



Soil Floor (OVPT)

Fig. V.13. Time trends of alcaldía-*specific occupants in households with soil floor (%)*

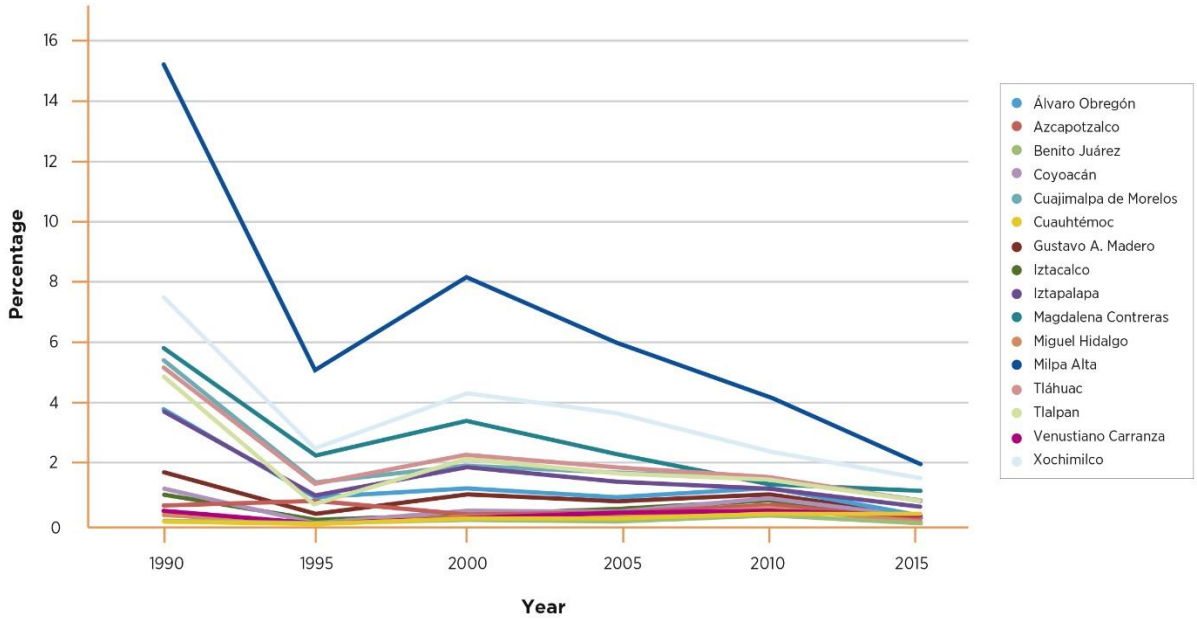
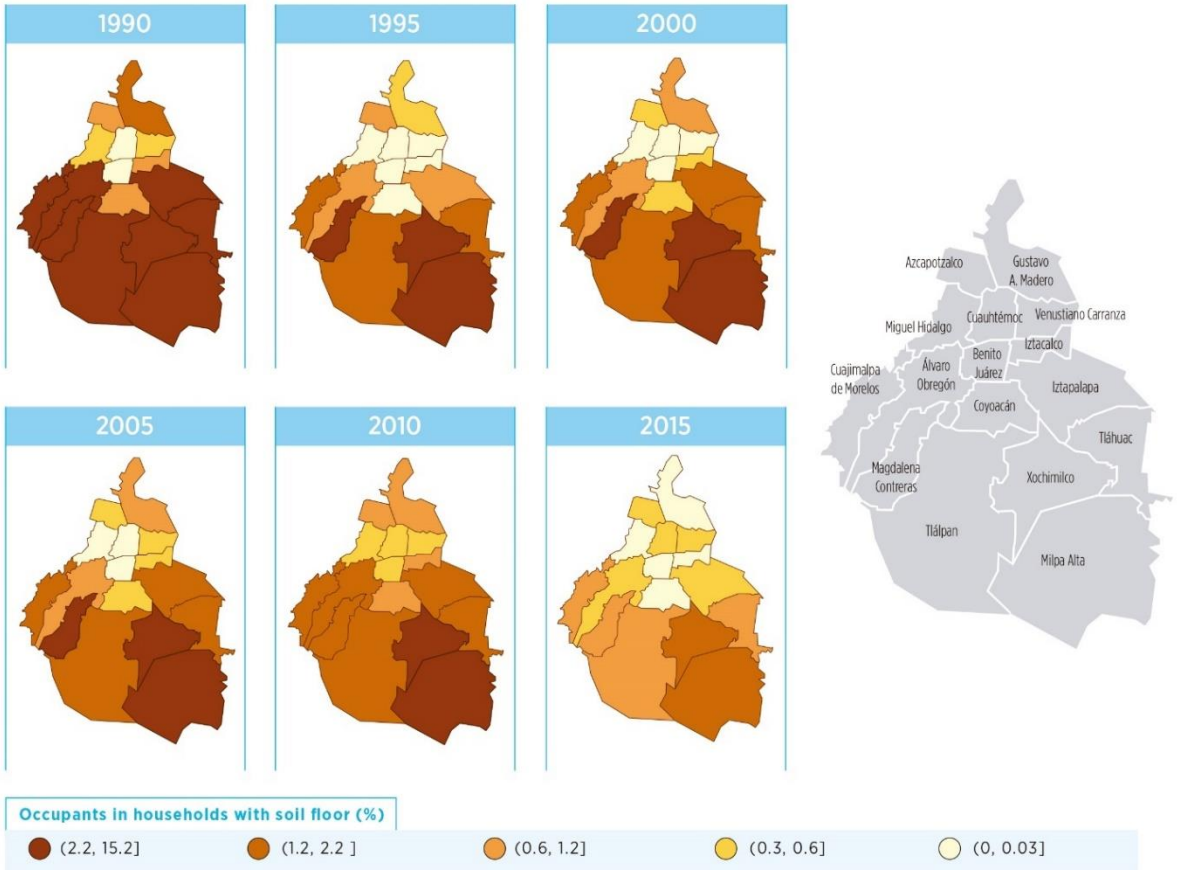


Fig. V.14. Spatial distribution of alcaldía-*specific occupants in households with soil floor (%) by year*



Low Income (PO2SM)

Fig. V.15. Time trends of alcaldía-*specific population with a job of up to two minimum wages (%)*

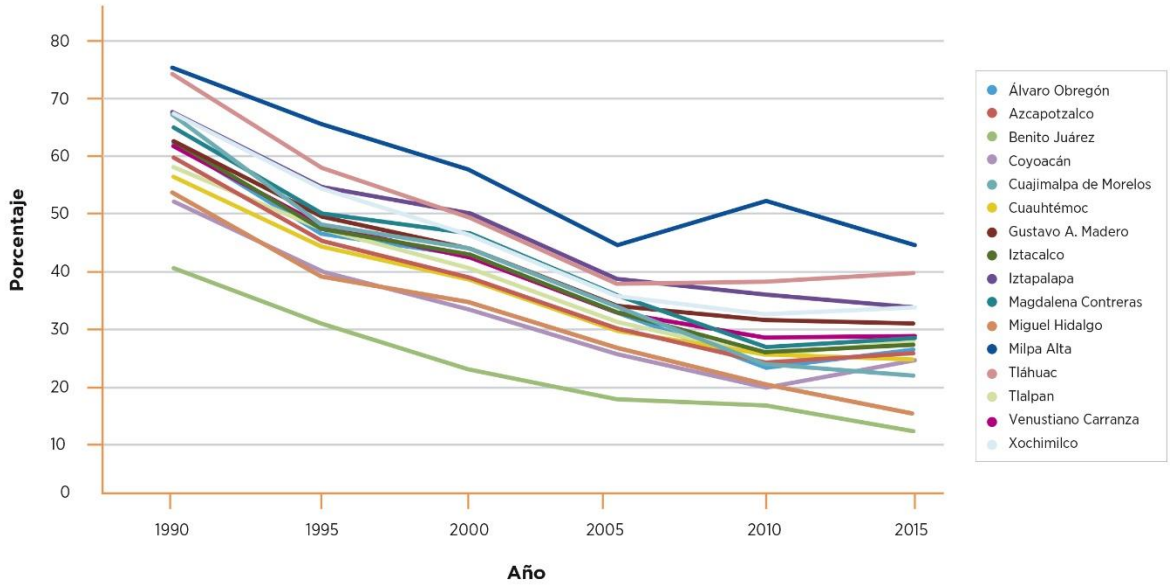
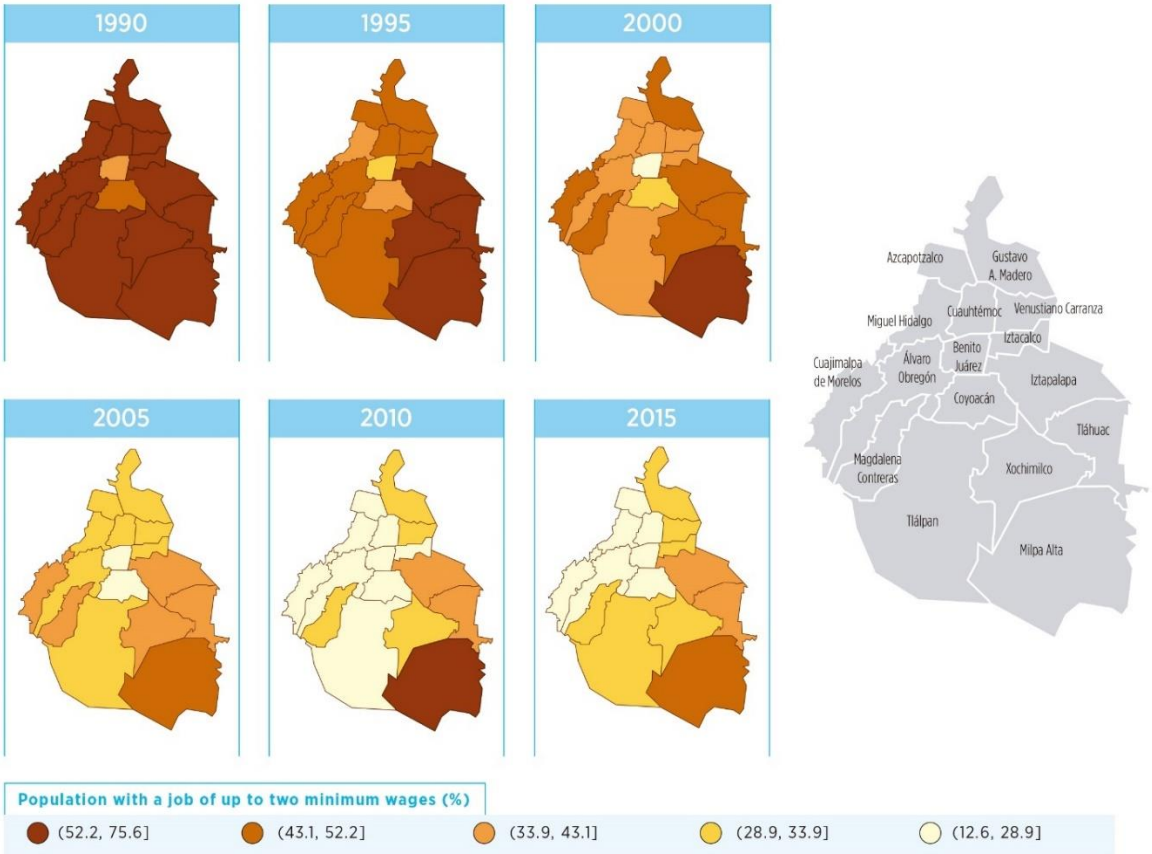


Fig. V.16. Spatial distribution of alcaldía-*specific population with a job of up to two minimum wages (%) by year*



APPENDIX VI. AIR POLLUTION RELATED DISEASES

Ischemic Heart Disease (≥25 years)

Fig. VI.1. Time trends of alcaldía*-specific ischemic heart disease mortality rates (deaths per 100,000)*

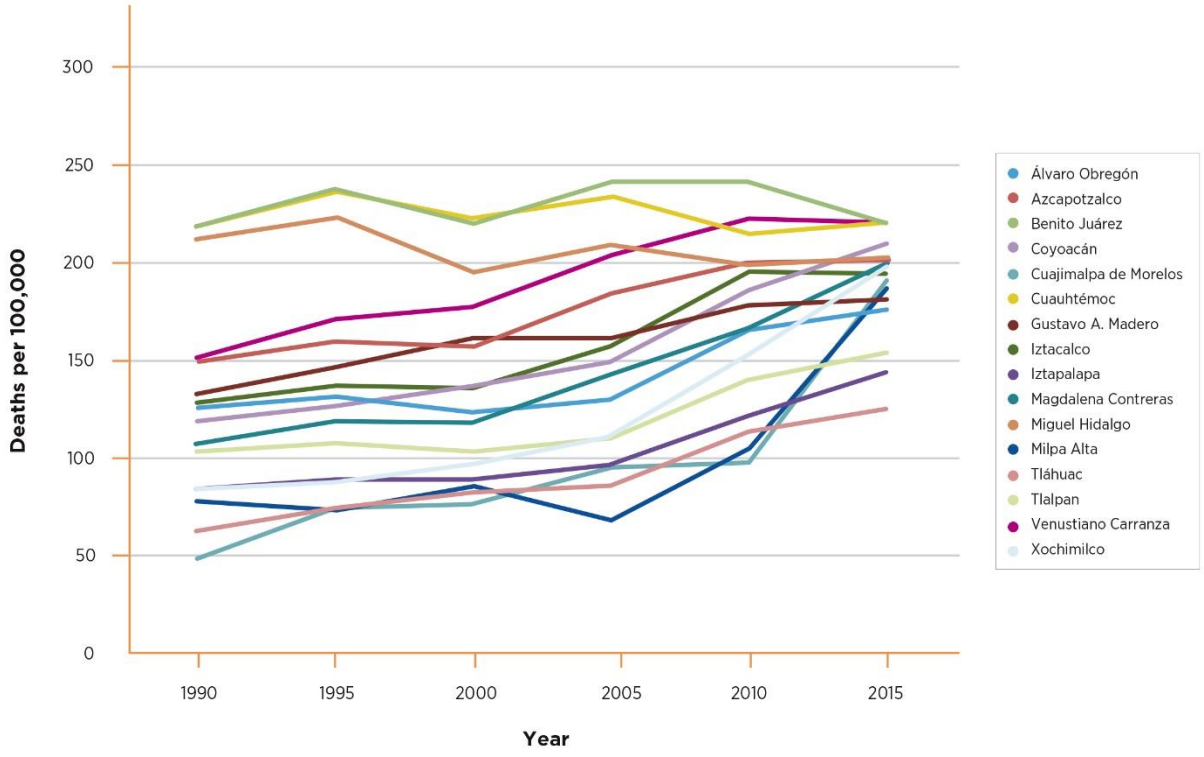
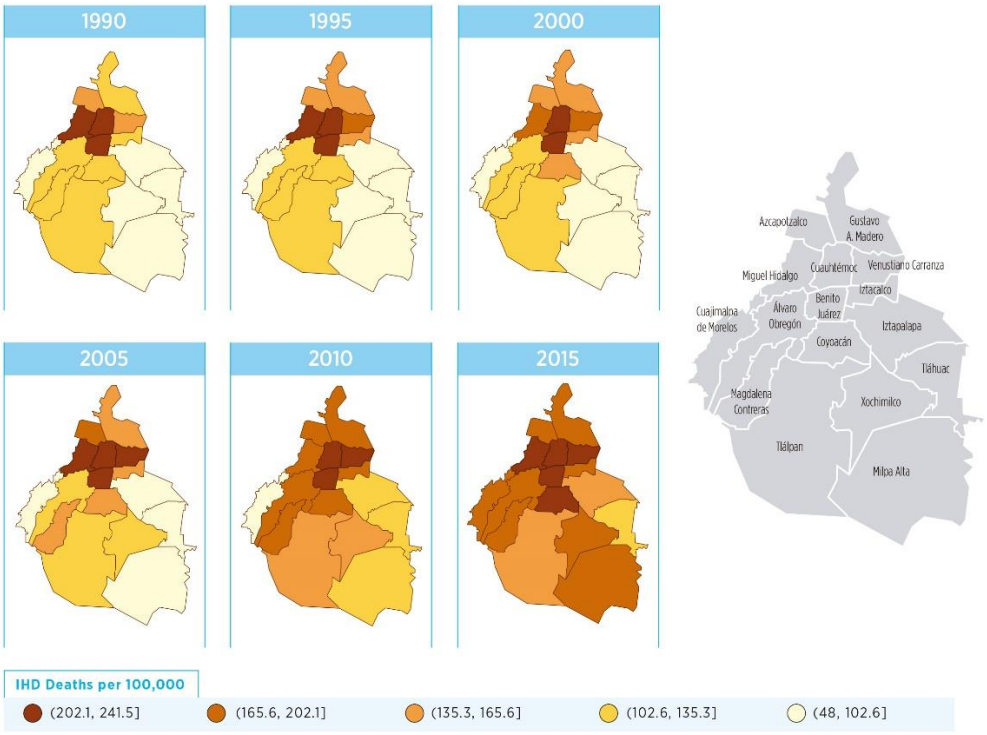


Fig. VI.2. Spatial distribution of alcaldía*-specific ischemic heart disease mortality rates (deaths per 100,000) by year*



Cerebrovascular Stroke (≥25 years)

Fig. VI.3. Time trends of alcaldía*-specific cerebrovascular stroke mortality rates (deaths per 100,000)*

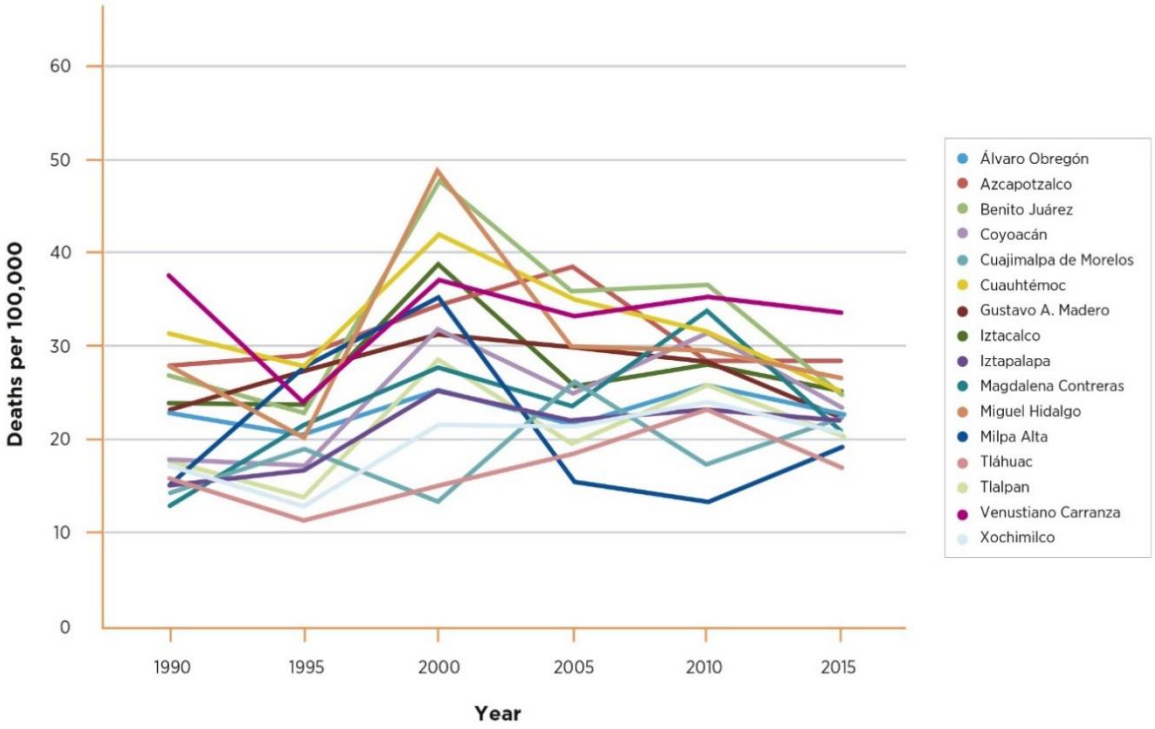
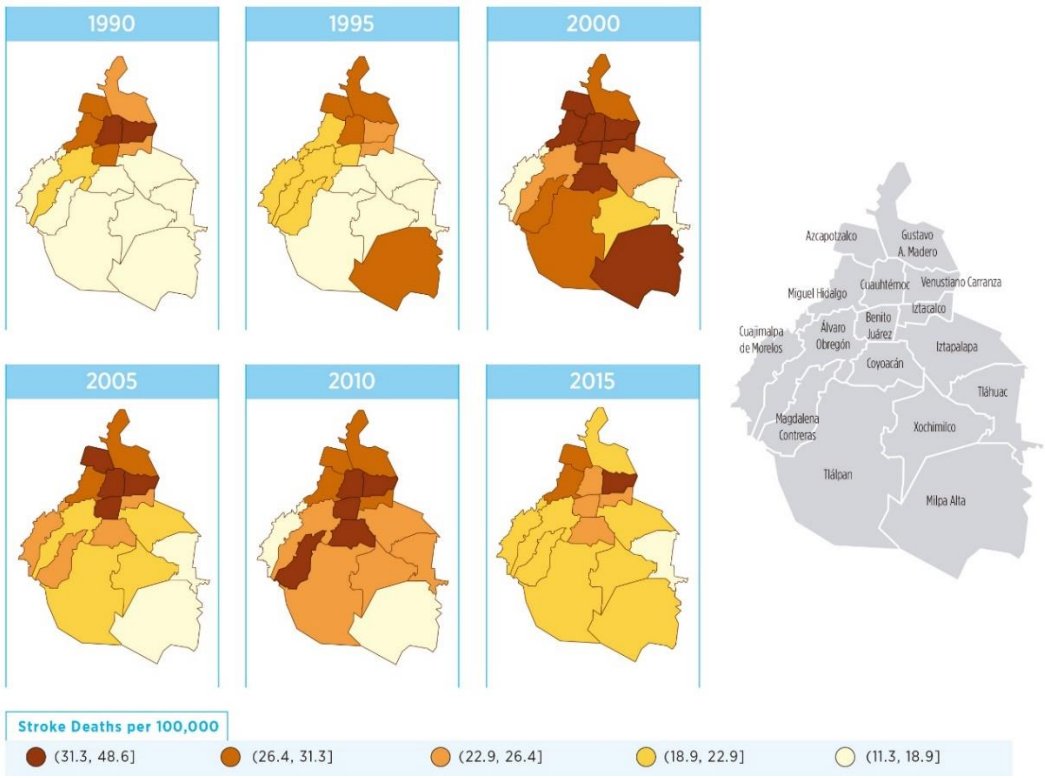


Fig. VI.4. Spatial distribution of alcaldía*-specific cerebrovascularstroke mortality rates (deaths per 100,000) by year*



Acute Lower Respiratory Illness (0-4 years)

Fig. VI.5. Time trends of alcaldía*-specific acute lower respiratory illness mortality rates (deaths per 100,000)*

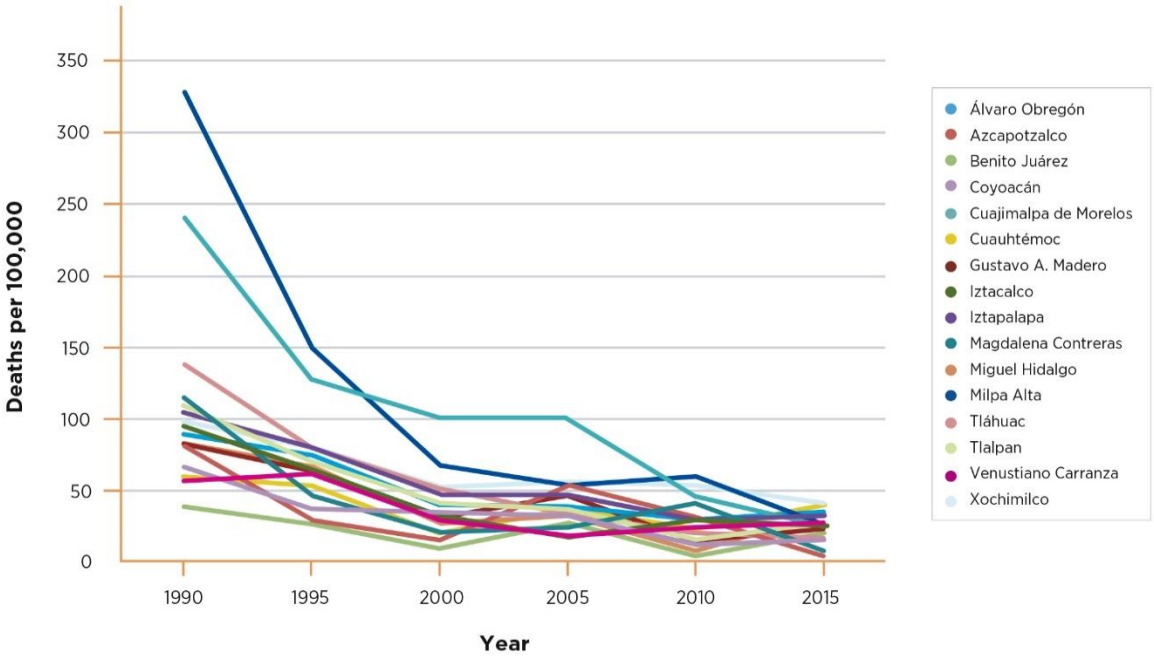
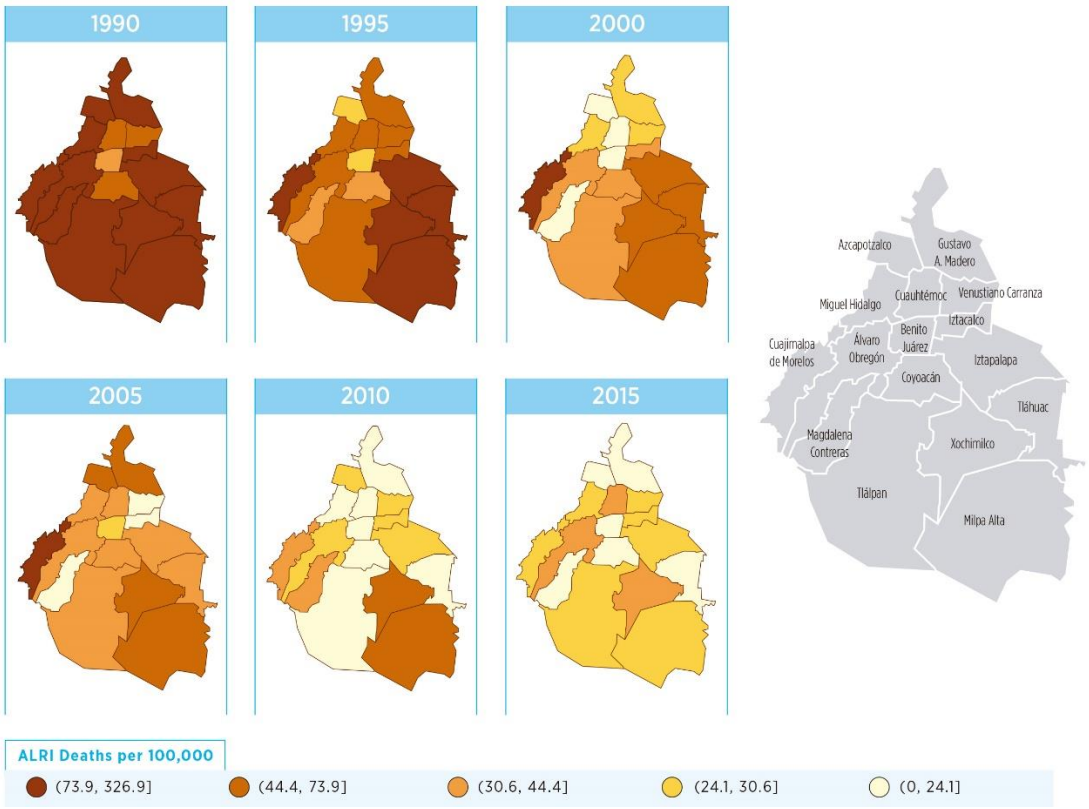


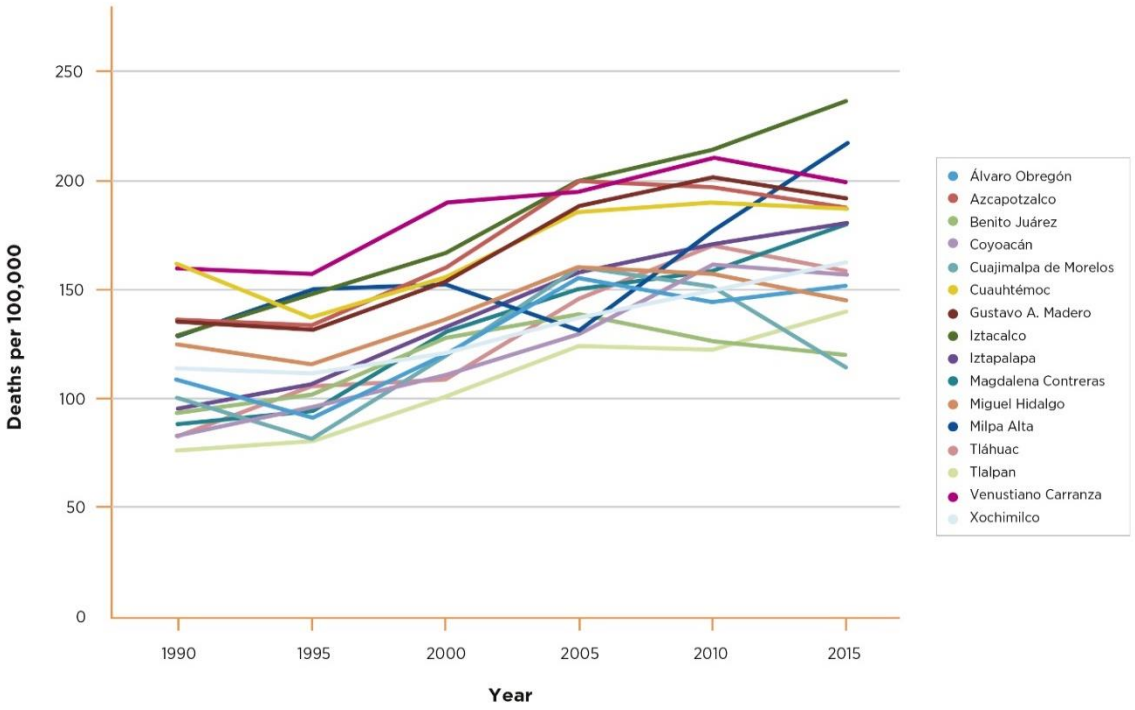
Fig. VI.6. Spatial distribution of alcaldía*-specific acute lower respiratory illness mortality rates (deaths per 100,000) by year*



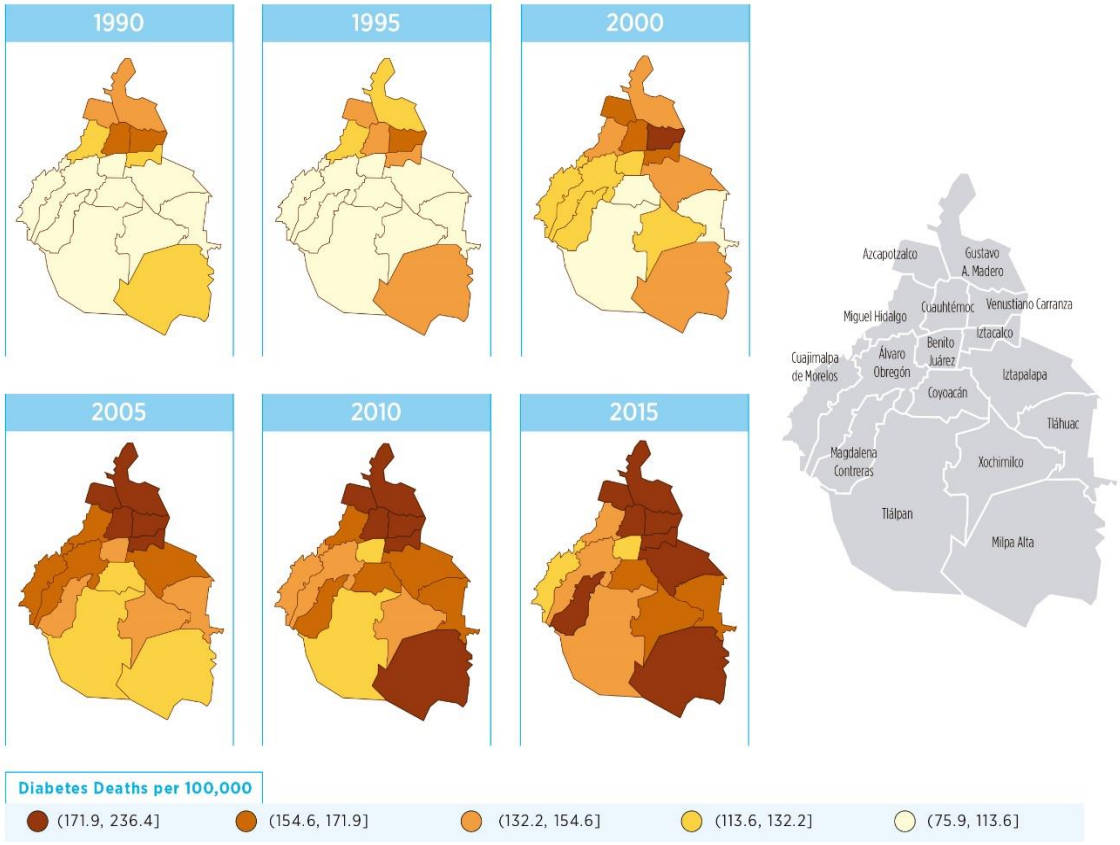
APPENDIX VII. REFERENCE DISEASES

Diabetes (≥25 years)

Fig. VII.1. Time trends of alcaldía*-specific diabetes mortality rates (deaths per 100,000)*

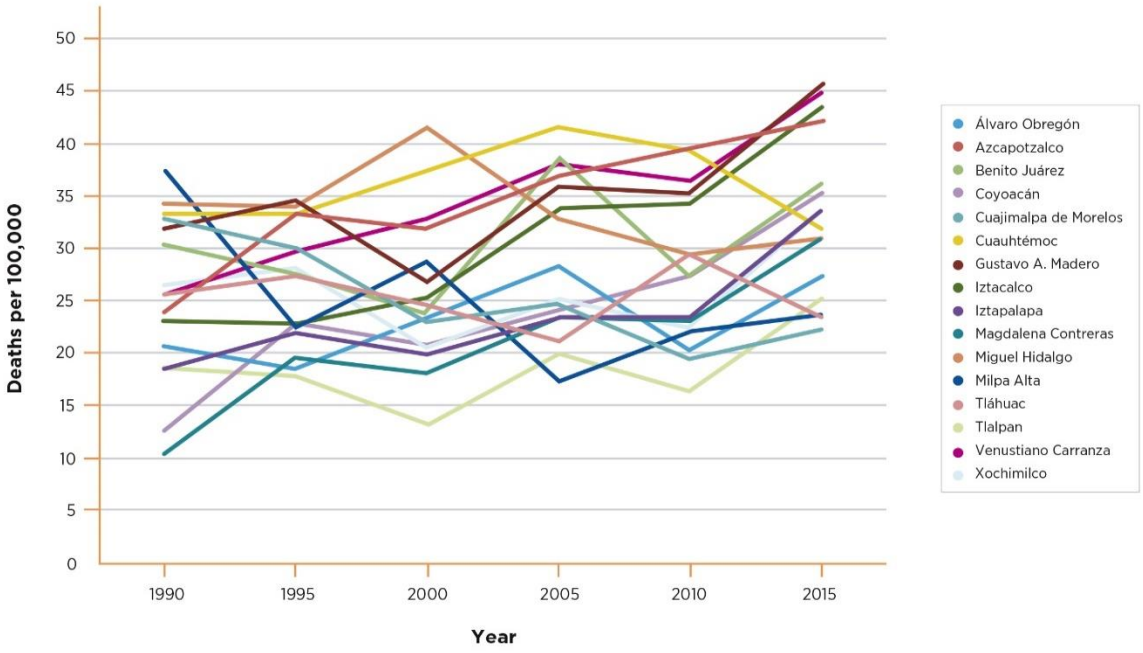


VII.2. Spatial distribution of alcaldía*-specific diabetes mortality rates (deaths per 100,000) by year*

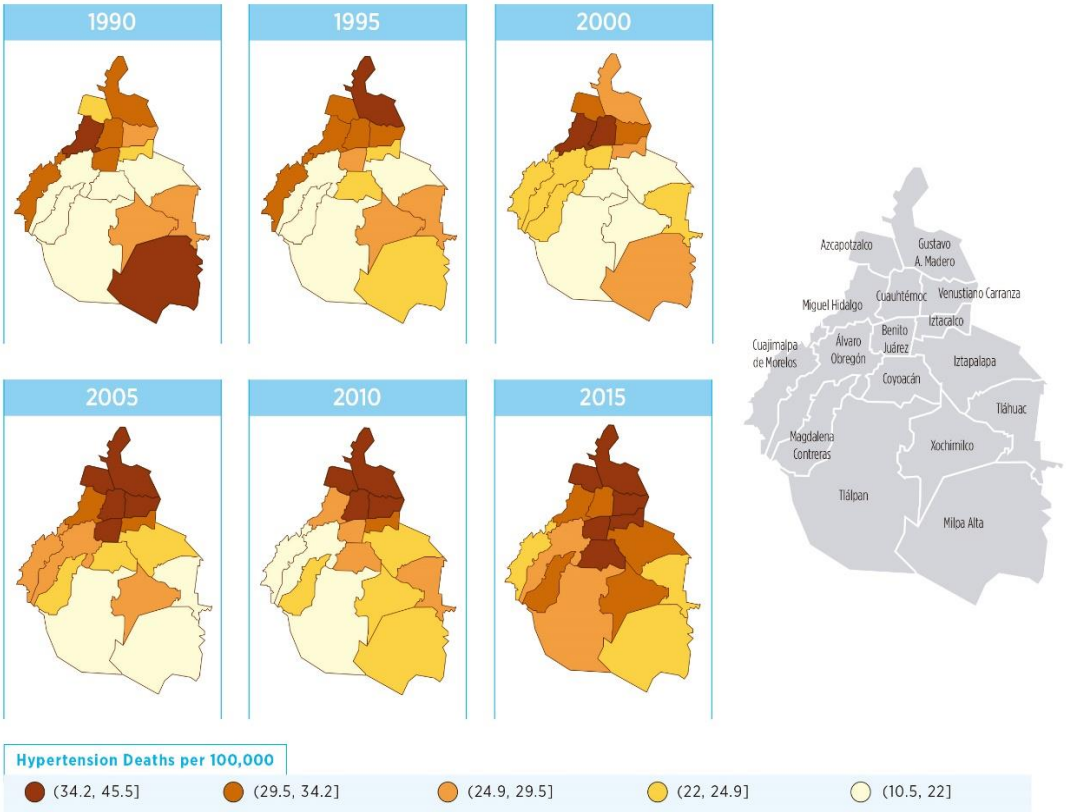


Hypertension (≥25 years)

VII.3. Time trends of alcaldía-*specific hypertension mortality rates (deaths per 100,000)*

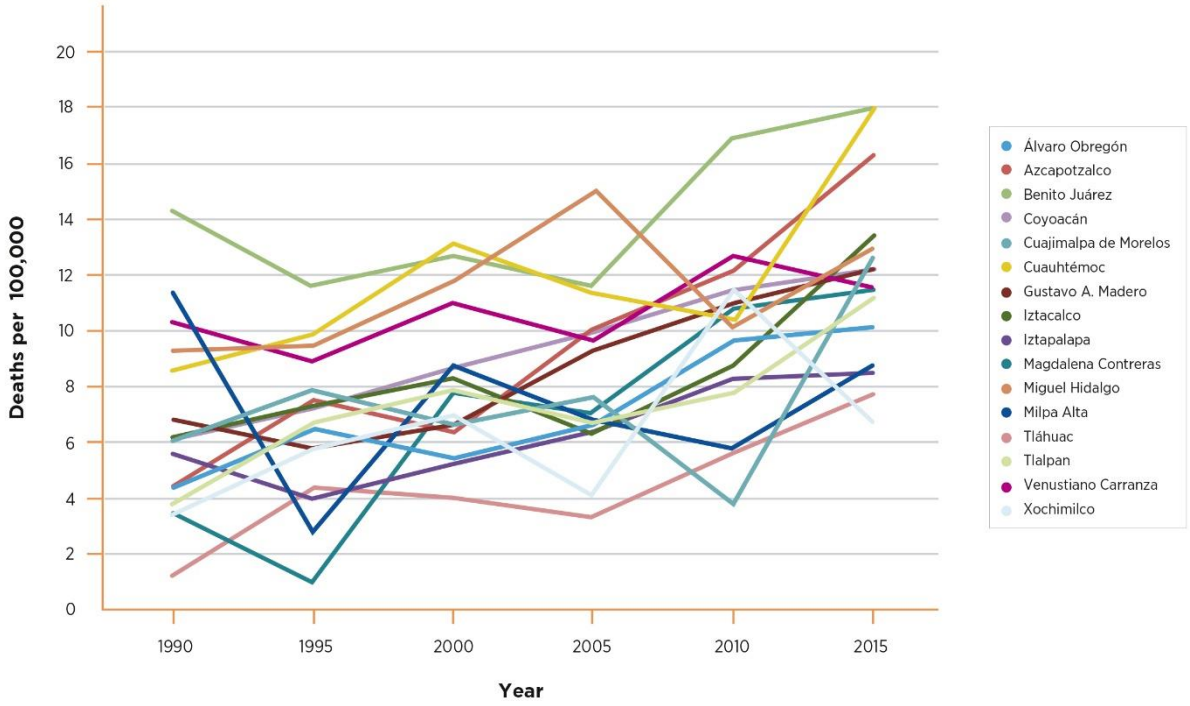


VII.4. Spatial distribution of alcaldía-*specific hypertension mortality rates (deaths per 100,000) by year*

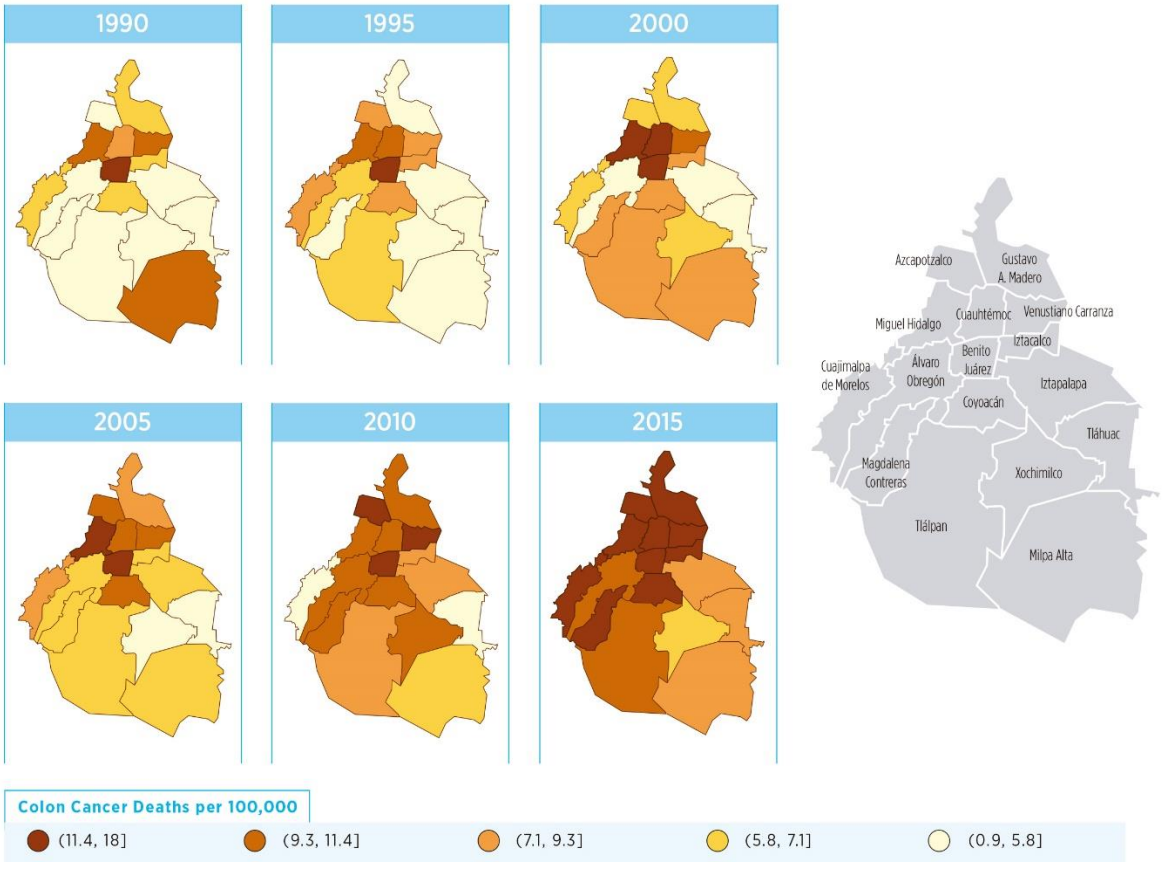


Colon Cancer (≥25 years)

VII.5. Time trends of alcaldía-*specific colon cancer mortality rates (deaths per 100,000)*

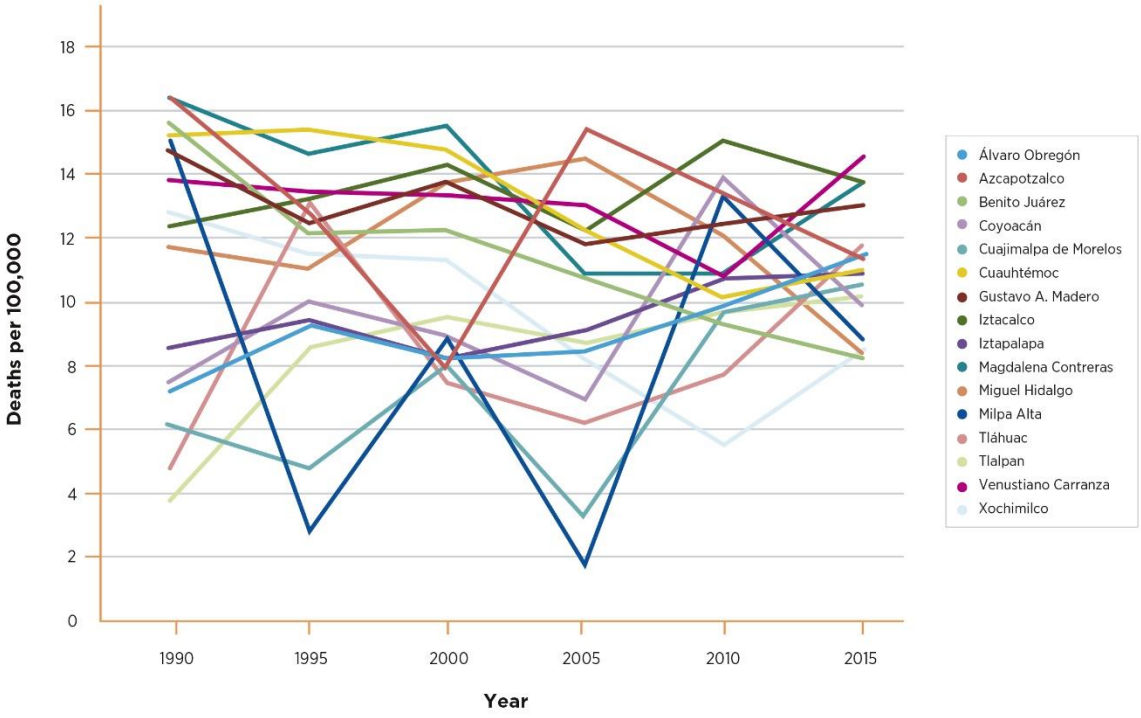


VII.6. Spatial distribution of alcaldía-*specific colon cancer mortality rates (deaths per 100,000) by year*

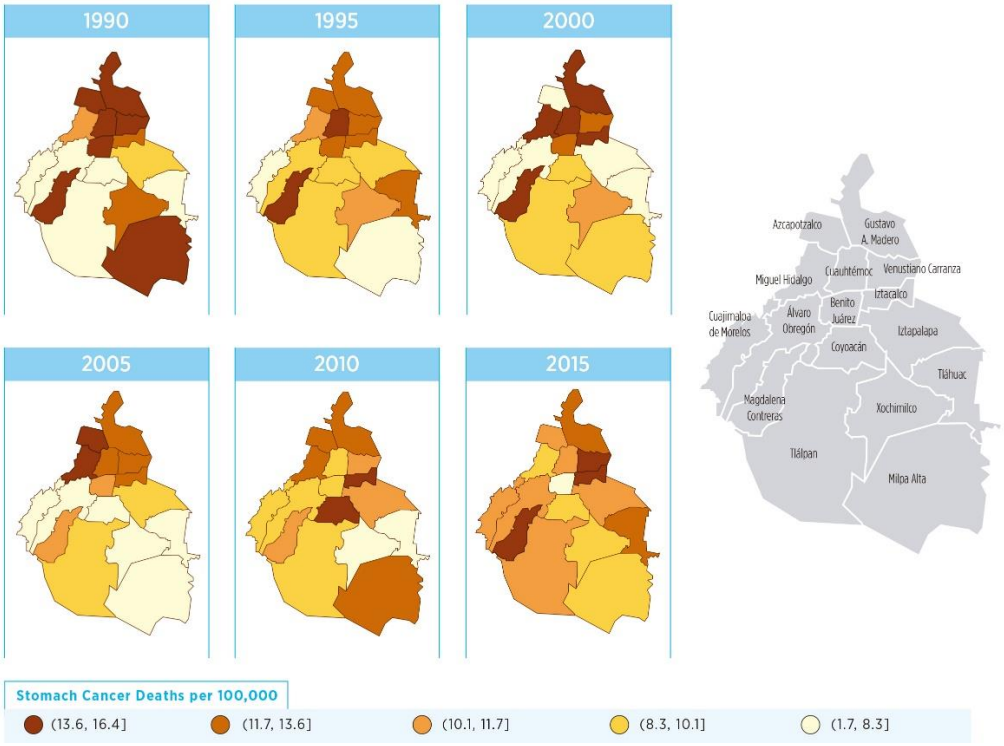


Stomach Cancer (≥25 years)

VII.7. Time trends of alcaldía*-specific stomach cancer mortality rates (deaths per 100,000)*



VII.8. Spatial distribution of alcaldía*-specific stomach cancer mortality rates (deaths per 100,000) by year*



External Causes (Assault) (≥25 years)

Fig. VII.9. Time trends of alcaldía-*specific external causes (assault) mortality rates (deaths per 100,000)*

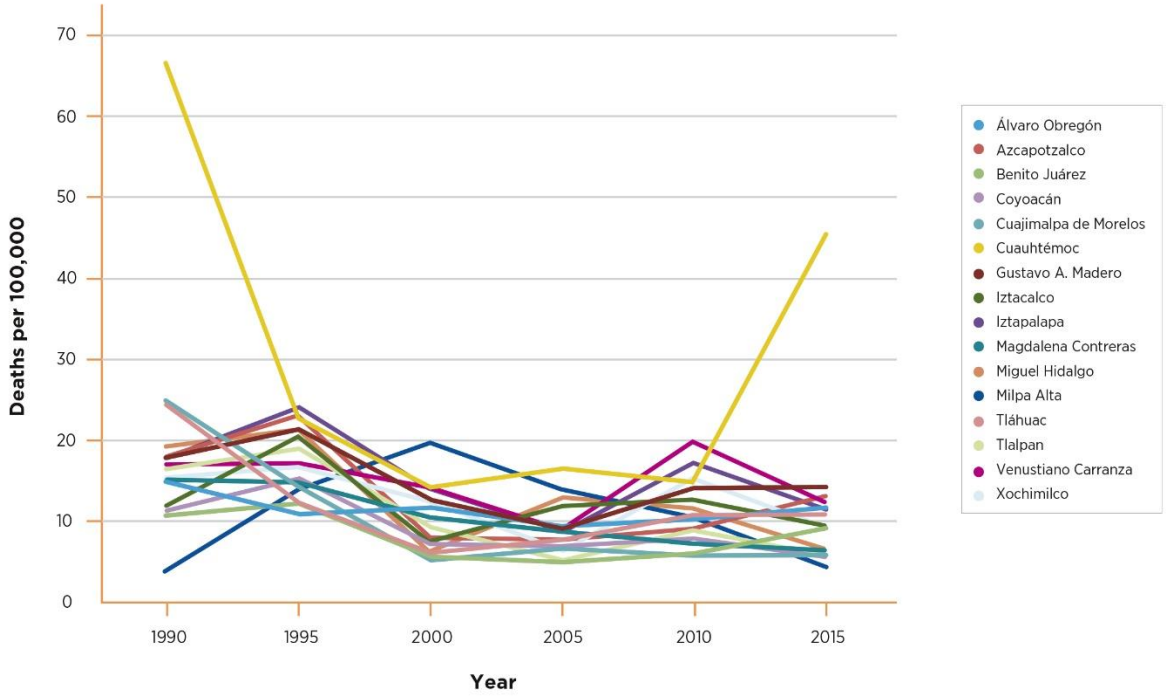
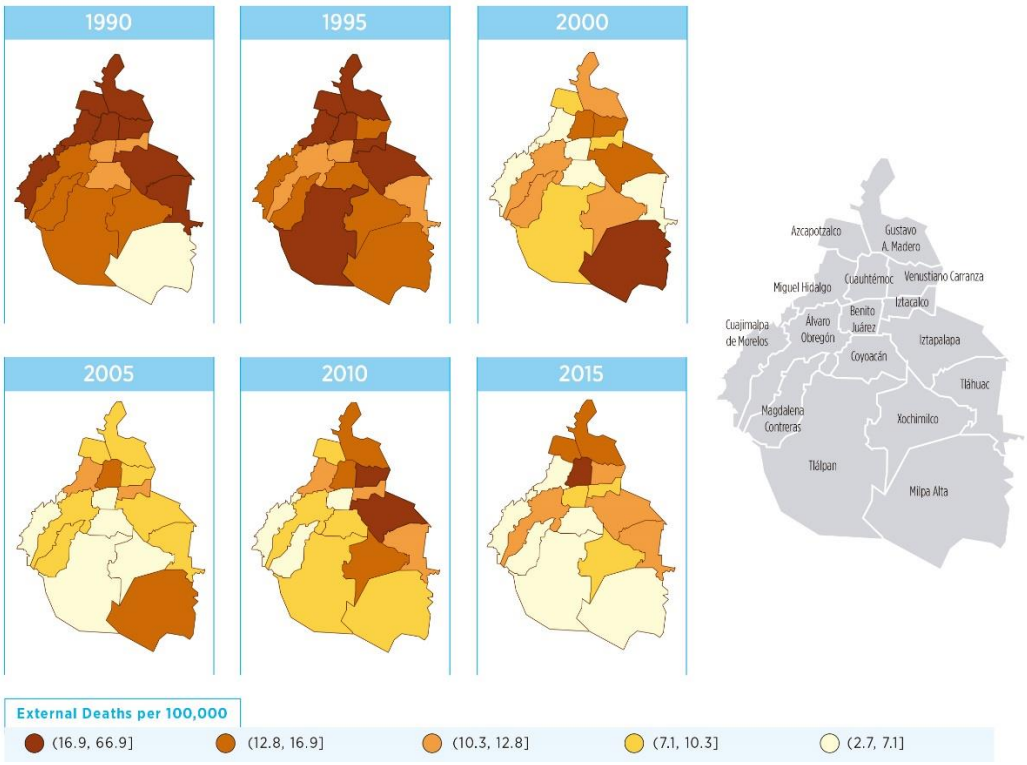


Fig. VII.10. Spatial distribution of alcaldía-*specific external causes (assault) mortality rates (deaths per 100,000) by year*



APPENDIX VIII. MEXICO CITY SUMMARY CHARACTERISTICS

Table VIII.1. Summary Characteristics of Alcaldías *(n=16) in Mexico City, 1990 and 2015*

Broad Characteristics	Variable / Indicator		Mean	SD	Median
Air Pollution	Ozone (ppb)	1990	160.70	±19.47	160.59
		2015	83.81	± 4.24	84.45
		Change	-76.89	±16.87	-78.77
	$PM_{2.5}$ (µg/m³)	1990	35.61	± 4.02	36.50
		2015	21.03	± 1.04	21.55
		Change	-14.59	± 3.16	-15.20
Life Expectancy	Life Expectancy (years)	1990	72.31	±2.15	72.29
		2015	77.75	±2.13	77.65
		Change	5.45	±1.77	5.24
	Life Expectancy in Men (years)	1990	69.12	±2.66	69.35
		2015	74.91	±2.99	74.7
		Change	5.78	±2.22	5.89
	Life Expectancy in Women (years)	1990	75.32	±1.71	75.15
		2015	80.38	±1.47	80.24
		Change	5.05	±1.53	4.86
	Temporary Life Expectancy 0-4 (years)	1990	4.87	±0.05	4.88
		2015	4.95	±0.01	4.95
		Change	0.09	±0.04	0.08
	Temporary Life Expectancy 25-74 (years)	1990	41.77	±1.13	41.89
		2015	43.94	±1.05	44.03
		Change	2.17	±0.99	1.99
Smoking Related Diseases	COPD (deaths per 100,000)	1990	43.61	±12.01	41.43
		2015	36.44	±7.83	36.43
		Change	-7.17	±7.74	-6.23
	Lung Cancer (deaths per 100,000)	1990	13.30	±7.30	10.70
		2015	12.15	±2.9	11.89
		Change	-1.16	±6.26	-0.12
Socioeconomic Position Indicators	Illiteracy (%)	1990	4.36	±1.5	4.16
		2015	1.48	±0.59	1.65
		Change	-2.87	±1.00	-2.76
	No Primary School (%)	1990	17.53	±4.42	17.12
		2015	6.54	±1.99	7.15
		Change	-10.99	±2.77	-10.29
	No Sewer Nor Toilet (%)	1990	2.94	±3.6	1.44
		2015	0.05	±0.06	0.03
		Change	-2.89	±3.54	-1.42
	No Electricity (%)	1990	1.12	±1.33	0.69
		2015	0.06	±0.09	0.02
		Change	-1.06	±1.24	-0.66
	No Running Water (%)	1990	4.30	±4.95	2.13
		2015	1.70	±3.29	0.44
		Change	-2.60	±2.94	-1.69
	Overcrowding (%)	1990	47.98	±11.54	48.43
		2015	19.62	±6.59	20.08
		Change	-28.36	±6.29	-28.26
	Soil Floor (%)	1990	3.55	±3.81	2.69
		2015	0.58	±0.54	0.31
		Change	-2.97	±3.31	-2.28
	Small Villages (%)	1990	1.32	±2.76	0.00
		2015	1.83	±5.17	0.00
		Change	0.51	±3.11	0.00
	Low Income (%)	1990	61.73	±8.29	62.48
		2015	28.03	±7.76	27.63
		Change	-33.70	±4.12	-33.69

Broad Characteristics	Variable / Indicator		Mean	SD	Median
Air Pollution Related Diseases	Ischemic Heart Disease (deaths per 100,000)	1990	126.39	± 51.84	122.35
		2015	189.12	±26.96	195.99
		Change	62.72	±40.52	61.04
	Stroke (deaths per 100,000)	1990	21.69	± 6.9	20.30
		2015	23.38	± 3.82	22.60
		Change	1.69	± 4.01	1.14
	ALRI (deaths per 100,000)	1990	110.39	±70.98	91.80
		2015	24.05	± 10.07	25.04
		Change	-86.35	± 71.85	-65.65
Reference Diseases	Diabetes (deaths per 100,000)	1990	113.44	±26.06	111.15
		2015	170.42	±32.04	170.96
		Change	56.98	± 27.22	53.35
	Hypertension (deaths per 100,000)	1990	25.35	± 7.57	25.55
		2015	33.11	± 7.49	32.60
		Change	7.76	± 11.13	6.77
	Colon Cancer (deaths per 100,000)	1990	6.59	± 3.30	6.14
		2015	11.97	± 3.21	11.86
		Change	5.38	± 3.26	5.82
	Stomach Cancer (deaths per 100,000)	1990	11.39	± 4.23	12.62
		2015	11.01	± 1.93	10.93
		Change	-0.38	± 4.43	-0.52
	External (Assault) (deaths per 100,000)	1990	19.07	± 13.26	16.64
		2015	11.32	± 9.35	9.45
		Change	-7.75	± 5.93	-6.08

SD: Standard Deviation; COPD: Chronic Obstructive Pulmonary Disease; ALRI: Acute Lower Respiratory Infections.

APPENDIX IX. DISEASES ASSOCIATED WITH AIR POLLUTION AND REFERENCE DISEASES: ICD9 AND ICD10 MORTALITY CODES

Mortality data for causes associated with air pollution were obtained from the Secretary of Health of the Government of Mexico City (SEDESA). SEDESA, in turn, gets the data from annual registries of INEGI (Institute of Statistics Geography and Informatics). Data for the rest of mortality causes were retrieved directly from INEGI's webpage. Data include number of deaths by day for each year.

For the 1990-1997 period the International Classification of Diseases (ICD) ninth version codes are used (ICD-9) and from 1998 onwards those from the tenth version are applied (ICD-10).

Quality control for mortality data is described in detail in the report of Phase II. Phase II. Estimation of the Health Benefits of Air Pollution Improvements, Appendix III. Causes of death. Data management and quality control procedures. The process included cleaning the data to ensure we had valid registries for CDMX and corresponding *alcaldías*, causes and age-groups needed for our study, year of death, etc.

Relevant notes regarding causes of death included in Table IX.1.

Global Burden of Disease analyses have determined five causes of death to be causally associated with air pollution exposures; long-term exposures to fine particles have been causally associated with all five causes, and ozone only with COPD. These causes are included under the label "Causes of mortality associated with exposures to fine particles and ozone".

For ALRI, the under five years-old group includes only post-neonatal deaths –over 28 days--, since they are influenced by the external environment of the child. Scientific evidence points that deaths occurring during the first month after birth are mainly related with intrinsic causes, such as congenital anomalies and that is less likely to be influenced by ambient air pollutants.

For trachea, bronchus and lung cancer there were no deaths for codes related with carcinoma in situ of middle ear and respiratory system, carcinoma in situ of trachea, of bronchus and lung, unspecified bronchus and lung (D02, D02.1, D02.2), nor of neoplasm of uncertain behavior of middle ear and respiratory and intrathoracic organs (D38).

For diabetes codes, CIE-9 code 250.0 includes type I and type II diabetes mellitus. Codes 250.1 to 250.9 include diabetes with ketoacidosis, hyperosmolarity...and other complications or manifestations of related disorders. ICD-10 E10 and E11 codes includediabetes mellitus type I and II. We exclude codes that refer to diabetes mellitus due to underlying condition (E08), drug or chemical induced diabetes mellitus (E09), and other specified diabetes mellitus (E13).

Table IX.1. Codes for causes of mortality associated with air pollution and reference causes of mortality

Causes of Death		ICD9 Codes (1990-1997)	ICD10 Codes (1998-2014)	Age
Causes of mortality associated with exposures to fine particles and ozone				
Acute Lower Respiratory Infections		487, 481, 482.2, 480.1, 466, 480.0-480.9, 482.0- 4829, 483-486, 513, 770	J09-J11, J13, J14 J12.1, J12, J15- J22, J85, P23	<5
Trachea, Bronchus and Lung Cancer		162-162.9 231.1, 231.2, 231.8, 235.7	C33-C34, D02.1-D02.2, D38.1	≥25
Ischemic Heart Disease		410-414	I20-I25	
Cerebrovascular Stroke	Hemorrhagic	430-431	I60-I62	
	Ischemic	432	163	
Chronic Obstructive Pulmonary Disease		490-492.8, 494, 496	J40-J44 J47	
Reference Causes of Death				
Hypertension		401 - 405	I10 - I15	≥25
Colon Cancer		153.0 - 154.8	C18 - C20	
Stomach Cancer		151.0 - 151.9	C16	
Diabetes		250.0 – 250.9	E10 - E14	
External Causes (Assault)		960 - 968	X92 - Y09	

Sorces: IHME, 2015. Mapping revisions and variants of the ICD. Web Table 3 - List of International Classification of Diseases codes mapped to the Global Burden of Disease cause list. http://www.healthdata.org/sites/default/files/files/data_for_download/2012/IHME_GBD2010_CauseListandICD.pdf; http://www.icd9data.com; https://www.icd10data.com

HISTORICAL ANALYSIS OF AIR QUALITY-RELATED HEALTH BENEFITS IN THE POPULATION IN MEXICO CITY FROM 1990 TO 2012.

Phase IV. Public Policy and Economic Valuation of the Health Benefits of Air Quality Improvements. Emission Controls for In-Use Heavy Duty Vehicles.

Final Report
2017

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Contents

Executive Summary

Due to the potential health impacts of diesel emissions to the atmosphere, we chose to conduct a cost-effectiveness analysis on emission reductions from diesel heavy-duty vehicles that circulate in Mexico City. This control policy was selected in line with the Air Quality Management Plan *PROAIRE 2011-2020*, specifically with *Strategy 3. Quality and energy efficiency in all sources*. Within this strategy, Measure 21 refers to the renewal of diesel vehicles with motor substitution and by adopting emissions controls (better known as retrofit).

The cost-effectiveness analysis conducted for Mexico City in-use heavy-duty vehicles clearly shows that performing retrofit with control technologies, such as Diesel Oxidation Catalysts (DOCs) or Diesel Particulate Filters (DPFs), can reduce particulate matter emissions, lead to improvements in air quality, and produce public health benefits among the inhabitants of the Mexico City Metropolitan Area, at a cost that is acceptable relative to the health benefits.

Retrofit programs have been put in place in other countries and have been on the radar of policy makers in Mexico for decades. In the US, CARB and EPA have implemented retrofit programs for most heavy-duty diesel vehicles. EPA's benefit-cost analysis of the program (2009 to 2013) shows an estimate of 1,700 fewer deaths attributed to the reduction in pollutant emissions, with a total present value of up to $11 billion in monetized health benefits over the lifetime of the affected engines (ICCT, 2017).

Over ten years ago (2005-2006), a pilot retrofit project was conducted in Mexico City (EMBARQ-WRI, 2007). DOCs and DPFs-catalyzed were installed in 20 model years 1991 and 2001 buses and were followed-up for close to a year. Primary PM reductions were on the order of 20 to 30% for DOCs, and 80 to 90% for DPFs. More recently, the *Autorregulación* voluntary program put in place by SEDEMA in Mexico City, has succeeded in having retrofit devices installed in 27 heavy-duty trucks and 18 RTP buses.

Our analysis seeks to determine whether expanding retrofit programs to a wide variety of diesel-fueled heavy-duty vehicles might be cost-effective. Resulting expected net benefits (health benefits minus control costs) were calculated as follows:

(i) Uses Emission Inventory, 2014 data for fleet number, emissions, and activity;
(ii) Estimates the cost-effectiveness on a per-vehicle basis: emissions reductions (kg/veh-yr), attributable deaths avoided (#/1000 veh-yr), monetized benefits of the avoided deaths (1000 US$/veh-yr), control costs (1000 US$/veh-yr), and net benefits (1000 US$/veh-yr);

(iii) Evaluates the net benefits of three retrofit technologies: oxidation catalyst; diesel particulate filter, active regeneration; diesel particulate filter, catalyzed; and, an ideal control to provide an upper bound on the net benefits of any possible emission-control technology;

(iv) Uses intake fraction to estimate exposure, where intake fraction is defined as the ratio of the population intake (g/y) of a pollutant divided by the emissions (g/y) of the pollutant or its precursor;

(v) Applies the concentration-response functions from the GBD analysis for 2010 and 2013 (Burnett et al., 2014), which includes five causes of death: ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, and trachea, bronchus and lung cancers, in adults, and acute lower respiratory infections in children; and,

(vi) Estimates the monetary value of health benefits with the Value per Statistical Life (through benefits transfer, using values from the US to estimate those for Mexico), and uses a discount rate of 3%.

We evaluate 1985 to 2014 model-year vehicles[1] from ten vehicle classes and five model-year groups, that span the range of vehicle types, uses and model years in the heavy-duty fleet operating in Mexico City. The vehicle classes in our analysis are: bus RTP – local plate, bus – school and personnel – local plate; bus – concession – local plate; Metrobús – local plate; bus – tourism – federal plate; bus – passenger – federal plate; truck – local plate; truck – federal plate; long-haul trailer – local plate; and long-haul trailer – federal plate. The model-year groups are: 1985-1993 (pre-control); 1994-1997 (US 1991/Euro I); 1998-2006 (US 1994/Euro II); 2007-2010 (US 1889/Euro III); and 2011-2014 (US 2004/Euro IV).[2]

Long-haul tractor trailers make up almost half of the fleet, with virtually all having federal plates. Buses account for about one third of the fleet, with two thirds of these having federal plates serving as tourism or passenger buses. Trucks, split equally between those with local plates and federal plates, account for the remaining 20% of the fleet. The heavy-duty diesel fleet is relatively old. Roughly 60% of the vehicles are more than 10 years old, with two thirds of these having been on the road for over 20

[1] Model year 1984 and older vehicles are excluded from the cost-effectiveness analysis because they are grouped in one category in the Emissions Inventory, 2014. This grouping results in aggregate emissions for a wide range of technologies.

[2] Because the Metrobús System (MB) started operations in 2005, MB vehicles were assigned to three model-year groups based on their own model years: 1998-2006 (for MB model year vehicles 2005-2006), 2007-2010, and 2011-2014. The control technologies of MB vehicles are one generation newer for such model-year groups (SEDEMA, 2017a): 2005-2006 (US 1998/Euro III); 2007-10 (US 2004/Euro IV); and 2011-14 (US 2007/Euro V).

years. Only 20% of vehicles were between 3 and 7 years old by the time the Emissions Inventory was developed.

The estimated total annual emissions of primary particles of ~ 1000 metric tons, are attributed mainly (> 50%) to long-haul trailers with federal plates, another 25% is due to concession buses with local plates. The remaining 20-25% is roughly equally split between buses, tourism and passenger with federal plates, and trucks with both local and federal plates. Four categories of vehicles with local plates, RTP transportation buses, school & personnel buses, Metrobús vehicles, and long-haul trailers, make inconsequential contributions to primary particle emissions.

Among the two vehicle types which dominate emissions of primary particles, 1998-2006 model year vehicles (group III – EPA 1994/Euro II) contribute most substantially, followed by 2007-2010 model year vehicles (group IV – EPA 1998/Euro III), and then, almost equally, by 1985-1993 model year vehicles (group I – pre-control), and 2011-2014 (US 2004/Euro IV).

To illustrate the results of our analysis, we describe our results for two categories of vehicles (bus concession – local plate and long-haul tractor trailer – federal plate) which yield the largest share of primary PM emissions.

For the approximately four thousand concession buses with local plates from the model-year group 1998-2006 (EU 1994/Euro II), the catalyzed DPF (passive) retrofit is expected to reduce emissions by 35.6 kg per vehicle-year, and to reduce premature deaths attributable to air pollution by about 3 per 1000 vehicle-year; with benefits of US$ 9.2 thousand, and costs of only 1.4 thousand US$ per vehicle-year. For this category of vehicles, the catalyzed DPF is an option because these buses are driven only locally, where ultra-low sulfur fuel is available. The expected net benefits of this strategy are almost 8 thousand US$ per vehicle year.

Results for the long-haul tractor trailers with federal plates, also for model-year group 1998-2006 EU 1994/Euro II, show that for the nearly 16 thousand units, the largest net benefits (1.8 thousand US$ per vehicle-year) would be generated by choosing to retrofit with a catalyzed DPF. However, the catalyzed DPF (passive) is not an option because these long-haul tractor trailers which have federal plates are driven both in Mexico City, and outside of the city where ultra-low sulfur fuel is not widely available. Thus, among the remaining control options, the largest net benefits of close to 1.6 thousand US$ per vehicle-year, are generated by choosing to retrofit with an active regeneration DPF. This would be expected to reduce emissions by 10.2 kg per vehicle-year, and to reduce premature deaths attributable to air pollution by approximately 1 per 1000 vehicle-year; with benefits of over 2.6 thousand US$, and costs of slightly over 1 thousand US$ per vehicle-year. These are the same emissions reductions and health benefits as the

catalyzed DPF, but the costs are roughly 20% higher due to the larger fuel penalty associate with active regeneration of the filter.

Table I presents retrofit options that maximize expected net benefits for each vehicle type and model-year group. For all vehicle types and model years there is some retrofit that is cost-effective. In some cases, for example trucks with either local or federal plates, there are some model-year groups for which DPFs are not cost-effective, but oxidation catalysts are, with projected emissions reductions that range between 20 and 26%.

Table I. Retrofit options which maximize expected net benefits by vehicle type and model-year group in Mexico City, 2014, and estimated probability (%) that net benefits of indicated retrofit option are positive

Type of Vehicle & Plate		1985-93 Pre-Control	1994-97 US 1991/EURO I	1998-06 US 1994/EURO II	2007-10 US 1998/EURO III	2011-14 US 2004/EURO IV
Transportation Buses	RTP - Public Transport Local Plate	n.a.	n.a.	DPF-p 80	DOC 70	n.a.
	School & Personnel Local Plate	DPF-a 99	DPF-p 97	DPF-p 97	DPF-p 80	DPF-p 78
	Concession Local Plate	DPF-a 96	DPF-p 99	DPF-p 99	DPF-p 99	DPF-p 99
	Metrobús Local Plate	n.a.	n.a.	DPF-p 72	DPF-p 99	DPF-p 99
	Tourism Federal Plate	DPF-a 99	DPF-a 96	DPF-a 95	DPF-a 86	DPF-a 82
	Passenger Federal Plate	DPF-a 90	DPF-a 74	DPF-a 70	DOC 98	DOC 98
Delivery Trucks > 3.8 tons	Trucks Local Plate	DOC 99	DPF-p 80	DPF-p 80	DPF-p 80	DOC 96
	Trucks Federal Plate	DOC 99	DOC 99	DPF-a 65	DPF-a 74	DPF-a 58
Long-Haul Tractor Trailers > 27.2 tons	Trailers Local Plate	DOC 91	DOC 93	DPF-p 84	DPF-p 93	DPF-p 87
	Trailers Federal Plate	DPF-a 95	DPF-a 88	DPF-a 95	DPF-a 97	DPF-a 94

Notes: RTP public transportation and Metrobús vehicles have vehicles than belong to only two and three model-year groups, respectively. DOC stands for Diesel Oxidation Catalyst; DPF-p stands for Diesel Particulate Filter with catalyzed regeneration (passive), DPF-a stands for Diesel Particulate Filter with active regeneration, and n.a. stands for not applicable for vehicle categories and model-year groups for which there were no vehicles in the Emissions Inventory, 2014.

There is always uncertainty about the health benefits and costs of policies to reduce air pollution. By quantifying uncertainty about some of the most important parameters used in our analyses, we can estimate the probability that the net benefits of the identified retrofit options are positive, i.e., that benefits from reductions in mortality

risk exceed the cost of the specified retrofit technology. These probabilities are displayed in Table I for each vehicle type and model-year group.

In our analyses we quantified uncertainty about some of the most important inputs: the relationship between emission reductions and population exposure (summarized by the intake fraction), the relationship between mortality and air pollution (with the slope of the exposure-response functions for specific causes of death), the monetary value of reductions in mortality risk (summarized by the value per statistical life), as well as the control efficiency and cost of each of the control options.

For most vehicle types and model-year group categories, the probability that the identified retrofit option will yield benefits greater than its cost is about 80 percent or larger. For the two vehicle types accounting for the largest share of emissions, the estimated probability that the value of the mortality-risk reduction associated with retrofit with either active or passive DPF exceeds the cost of the retrofit is at least of 88 percent and 96 percent, respectively, for long-haul trailers with federal plates and concession buses with local plates.

The two exceptions are RTP buses with local plates and delivery trucks with federal plates, for which the most cost-effective technologies, for certain model-year groups, yield positive net benefits with probabilities of only 70 and 58 percent, respectively. Although the chance the identified controls for these vehicle types will yield benefits less than their costs is significant, the alternative of oxidation catalysts for delivery trucks with federal plates is very likely (almost 99%) to yield positive but smaller net benefits. For decisions like these, where one can never be certain about the benefits and costs of a decision, maximizing expected net benefits is an attractive criterion that balances the probability that benefits exceed costs with the likely sizes of the gain (if benefits do exceed costs) or loss (if benefits do not exceed costs).

Our analysis shows that a fully implemented program to retrofit every heavy-duty vehicle with the control which maximizes *expected* net benefits for that vehicle type and model-year group, has the potential to:

- Reduce annual emissions of primary fine particles by close to 950 metric tons; which would
- Cut the annual population-weighted mean concentration of $PM_{2.5}$ in Mexico City by slightly over 0.90 $\mu g/m^3$;

- Reduce the annual number of deaths attributable to air pollution by over 80[3]; and to
- Generate expected health benefits of almost 250 million US$ per year.

Also, it would have *expected* annual costs of close to 93 million US$ per year – consisting of 61 million US$ in 'amortization' of capital cost of retrofit devices; 19 million US$ in annual maintenance costs; and 11 million US$ in fuel use penalties. Net benefits, thus, would be in the order of 150 million US$ per year.

We close by noting that this one small step must be viewed from the wider perspective suggested by the Air Quality Management Program 2011 – 2020 (PROAIRE de la ZMVM), 2011-2020 and by the Mario Molina Center's 2016 position paper on air quality in the Mexico City Valley (CMM, 2016). In addition to reducing emissions from heavy-duty vehicles, many other programs -- such as the development of an integrated public transportation system, the promotion of the rational use of cars, the reduction of emissions from industrial sources and fires, and redesign of the MCMA area to reduce urban sprawl -- must be analyzed and implemented to make significant strides forward in the control of air pollution and its public health impacts.

[3] This is an estimate for the initial years after vehicles are retrofit. As lives cannot be saved by air pollution controls or any other public policy intervention but can merely be extended. Over time the reduction in mortality risk will change the size and age structure of the population exposed to air pollution -- increasing both the number of people at risk and their ages. Ultimately, the total number of deaths per year will rise to the level it would have been without retrofits, but these deaths will occur at older ages, on average.

Introduction

The Secretaries of Environment (SEDEMA) and Health (SEDESA) of the government of Mexico City[4] initiated a program of collaboration with the Harvard TH Chan School of Public Health in 2015. The focus of the program is on Mexico City's air quality, and its general objective is to analyze the health benefits for the inhabitants of Mexico City associated with the improvements in air quality achieved over the last 20 years through implementation of air quality policies. These benefits are being assessed through the interpretation and application of the most rigorous international and national scientific information in the fields of epidemiology and air quality, as well as the implementation of a risk assessment and policy analyses methodology.

The program consists of four phases, including two phases that were completed in 2016: a review of the state of knowledge relevant to Mexico City (Phase I), and an estimation of the health benefits of air quality improvements, through risk assessment methods (Phase II). Two more phases are being conducted in 2017: Phase III, that seeks to verify the results of the risk assessment through epidemiological methods, and Phase IV, that seeks to assess the health benefits to the population of Mexico City associated with the implementation of public policies to improve air quality and produce an economic valuation thereof. This document is the final report of Phase IV.

Due to the potential health impacts of diesel emissions to the atmosphere, the control policy selected for the analysis relates to the reduction of emissions from diesel heavy-duty vehicles that circulate in Mexico City. This control policy was selected in agreement with the Air Quality Management Plan *PROAIRE 2011-2020*, specifically with *Strategy 3. Quality and energy efficiency in all sources*. Within this strategy, Measure 21 refers to the renewal of diesel vehicles with motor substitution and by adopting emissions controls (better known as retrofit).

The evaluation of benefits and costs was carried out for several retrofit systems for control of emissions from diesel heavy-duty vehicles. These retrofit alternatives are aimed at reducing emissions of fine suspended particles ($PM_{2.5}$) from diesel heavy-duty vehicles, since this is the pollutant with the greatest health impacts.

This final report includes a description of the *status quo*, i.e., a characterization of the current state of diesel heavy-duty vehicles, describing their number, age, activity, and emissions, based on the data from the Mexico City Emissions Inventory, 2014. Also, the

[4] Previously called Federal District, and since 2016 called Mexico City. The Federal District and now Mexico City are formed by 16 counties (called *delegaciones* and will change to *alcaldías* starting in 2018).

retrofit control systems are defined and characterized, in terms of their emissions control efficiencies for the heavy-duty diesel vehicles that circulate in Mexico City.

This study aims at conducting a cost-effectiveness analysis of control alternatives to reduce fine particle emissions from heavy-duty vehicles (HDV) in Mexico City. For the study, emissions and activity data refer to those within Mexico City itself; thus, we count emission reductions within the city, and ignore those outside of the city boundaries. Buses, delivery trucks and long-haul trailers included in Mexico's City latest emissions inventory with base-year 2014 (SEDEMA, 2016), which may have plates from Mexico City or may have federal plates (see below for plate characteristics), are comprised in our analysis. Estimated health benefits accrue to the entire Mexico City Metropolitan Area population. The goal of this cost-effectiveness analysis is to provide information for decision makers to discriminate among control alternatives and implement the best possible controls in the most cost-efficient way.

1. Emissions Inventory, 2014 Heavy-Duty Fleet: *Status quo*

In the Mexico City Metropolitan Area (MCMA) there were 5.3 million vehicles in 2014 (SEDEMA, 2016). The fleet is almost evenly distributed between Mexico City and the State of Mexico. In Mexico City, there are almost 120,000 heavy-duty diesel-fueled vehicles (including vehicles of the Metrobús System), which account for close to 6% of the total vehicular fleet. The share of these vehicles is similar between the Mexico City and the State of Mexico. Examples of heavy-duty vehicles that circulate in Mexico City are shown in Appendix I.

Heavy-duty vehicles are grouped in three main categories –which are further divided in 10 classes (Table 1):

1. Buses, such as urban buses and inter-city buses
2. Trucks ≥ 3.8 tons, which include medium-sized delivery trucks with weights ranging from 4.6 to 27.2 tons for local plate vehicles, and from 11.8 to 14.9 tons for federal plate vehicles (See below for explanation on plate types).
3. Long-Haul Trailers, large vehicles, such as tractor trailers, and food supply vehicles weighing over 27.2 tons.

Heavy-duty vehicles are registered with either local or federal plates. This license plate system is regulated by the Secretary of Communications and Transportation (*Secretaría de Comunicaciones y Transportes*, SCT). Local license plates are issued at a state level or in Mexico City for vehicles that circulate within that specific state or within Mexico City. Federal license plates are reserved for vehicles that mainly circulate on highways that are under federal jurisdiction, regardless of the use of the vehicle --including vehicles for passengers, tourism and goods. Federal plates are issued within a state; therefore, vehicles circulating in the MCMA, in general, have plates issued by the Mexico City or by the State of Mexico authorities, but also from other states that make up the megalopolis. Table 1 shows that most HDV are registered under the federal plate system. One possible reason for the preponderance of federal plates is that vehicles with local plates undergo compulsory inspection and maintenance (I&M) and if they comply with certain emissions limits can circulate every day (SEDEMA, 2017). In contrast, I&M is voluntary for vehicles with federal plates.

Several factors lead to high emissions from HDV in Mexico City, including the age of the fleet. The oldest model year category is “1966 and older” for long-haul tractor trailers, which means that some of these vehicles are more than 50 years old (Fig. 1). The average

age for the whole fleet is 16 years.[5] Vehicles with local Mexico City plates are newer (mean 10 years old) than those with federal plates (mean 17 years old). Over 58% of the HDV fleet is more than 10 years old, which suggests that many vehicles may be approaching the end of their useful lifetime.

Table 1. Age Distribution of HDV in Mexico City (Years)

HDV Category	Transportation Buses						Delivery Trucks >3.8 tons		Long-Haul Tractor Trailer >27.2 tons	
	RTP Public Transport	School & Personnel	Concession	Metrobús	Tourism	Passenger	4.6-27.2 tons	11.8-14.9 tons		
Type of plate	Local				Federal		Local	Federal	Local	Federal
n	1,215	1,171	6,391	376	10,330	14,245	11,131	13,225	650	58,191
Mean	11	10	10	6	18	11	11	18	8	18
Std. Dev.	4	6	4	3	11	8	8	13	6	13
Q2-Median	13	9	10	6.5	15	10	9	15	7	15
Min = MY 2014	6	1	1	1	1	1	1	1	1	1
Q3-75%	14	14	13	10	24	14	16	27	10	27
Q1-25%	9	4	7	3.5	9	4	5	7	5	7
Max	14	39	34	10	41	40	48	49	38	49

Notes: The Emissions Inventory, 2014 includes vehicles from model year 1966 and older to model year 2014. The descriptive statistics presented in the table were computed with vehicles model year 1966 and older grouped together as model year 1966; and, vehicles model year 2014 were assumed to have been on the road for one year.
Source: SEDEMA, 2016.

The age distribution for the all categories is shown in Table 1 and Figure 1. The figure depicts a very skewed age distribution, with a long tail of old vehicles mostly composed of federal plate vehicles: long-haul trailers, delivery trucks (> 3.8 tons), and tourism transportation buses. These three federal plate categories are the oldest, with an average of 18 years; federal plate passenger buses are newer, with an average of 11 years. In contrast, the average age for local plate vehicles is between 6 and 11 years. This may be evidence of the effectiveness of programs led by Mexico City authorities such as the *Programa de Autorregulación*, which encourages private fleets to renew and retrofit their buses and thus be exempt from the *Hoy No Circula* program, as well as the efforts to renew the fleet used for public transport, such as Metrobús.

[5] Age of vehicles were estimated based on corresponding model years reported in the most recent Emissions Inventory for Mexico City (base-year 2014). Vehicles grouped in the oldest category in the Inventory (1966 and older) were taken as being 49 years old, and 2014 model year vehicles were assumed to have been in circulation for one year.

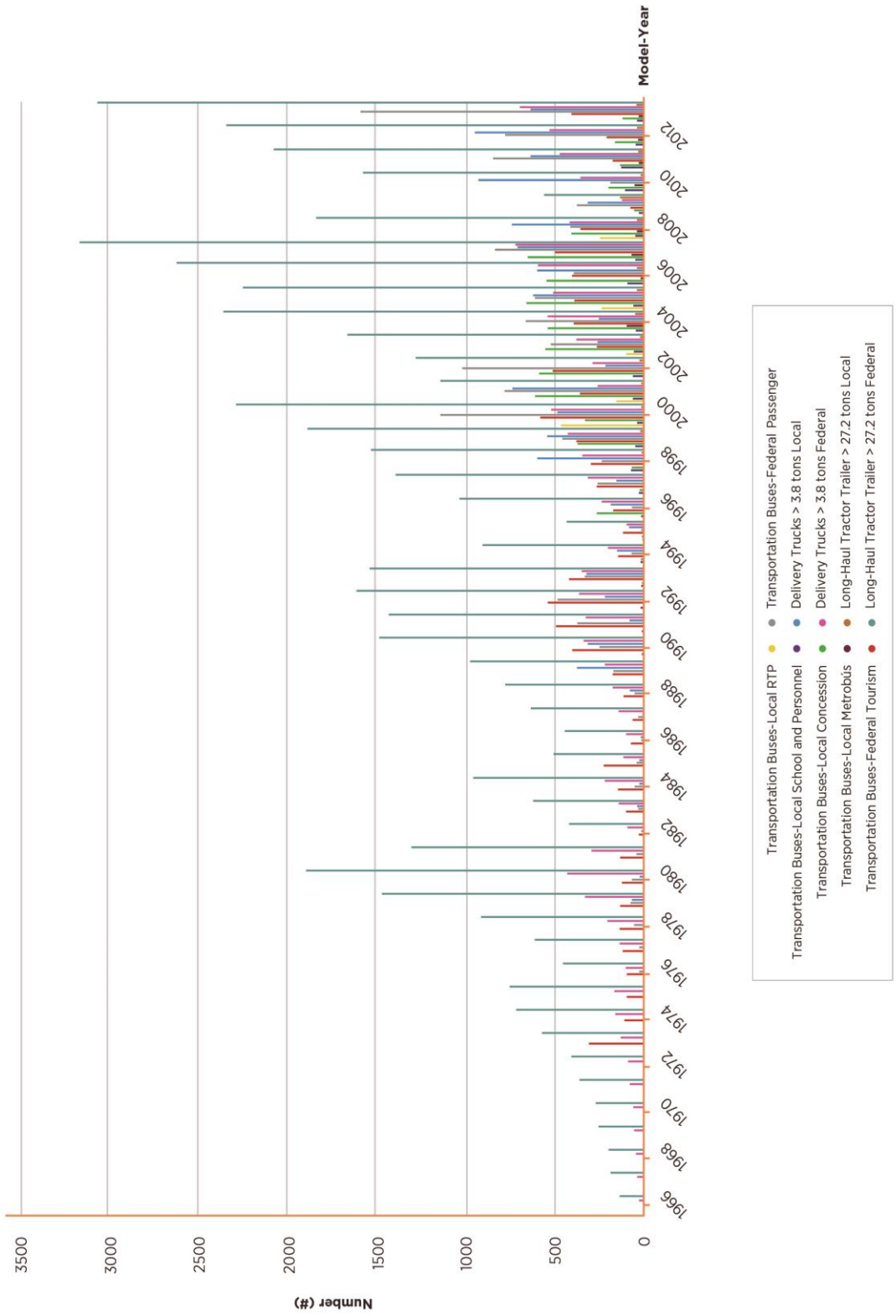


Figure 1. HDV Distribution by Model-Year and Category

Source: Elaborated by authors with data from SEDEMA, 2016; Dieselnet 2017; DOF, 1993; DOF, 2006

1.1. Emissions of Heavy-Duty Fleet

Diesel vehicles are a major source of air pollutant emissions, most importantly fine particles and nitrates, and, less so, sulfur dioxide and VOCs (Table 2).

The most recent emissions inventory conducted in Mexico City and in the MCMA (base-year 2014) indicates that for Mexico City mobile sources account for 33% of total primary $PM_{2.5}$ emissions, 79% of NOx emissions, 32% of SO_2 emissions, and 20% of VOC emissions. HD diesel vehicles, despite their small share of the vehicle fleet (less than 6%), are responsible for 24% of primary fine particle emissions, almost 40% of NOx emissions, and 3.5% and 1.3% of SO_2 and VOC emissions, respectively.

In the MCMA, the share of emissions is like that for Mexico City: mobile sources account for about one third of fine particle emissions, close to 80% of NOx emissions, and 20% of VOC emissions. Mobile sources contribute a lower share of sulfur dioxide emissions, 17% of total emissions. In the MCMA, the contribution of HD diesel vehicles shows a lower share than for Mexico City for fine particle emissions (slightly over 15%), for NOx (close to 30%), and almost the same contribution for SO_2 and VOC emissions.

Table 2. Mobile Source Emissions. Contribution of Heavy-Duty Vehicles, Mexico City and Mexico City Metropolitan Area, 2014

Vehicular Category	MCMA (tons/year)				CDMX (tons/year)			
	$PM_{2.5}$	NOx	SO_2	COV	$PM_{2.5}$	NOx	SO_2	COV
Mobile Sources	3,660	108,685	279	72,041	1,753	50,728	142	30,845
(%)	(100)	(100)	(100)	(100)	(100)	(100)	(100)	(100)
HDV+Metrobús	2,165.6	44,262.9	41.7	5,949.4	1,266.1	24,909.6	15.6	1,936.8
(%)	(59.2)	(40.7)	(14.9)	(8.3)	(72.2)	(49.1)	(11.0)	(6.3)
Long-Haul Trailers	841.1	16,013.0	8.2	627.0	605.6	11,550.6	6.0	447.1
(%)	(23.0)	(14.7)	(2.9)	(0.9)	(34.5)	(22.8)	(4.2)	(1.4)
Buses	796.8	15,534.1	8.8	1,289.4	480.3	9,379.8	4.2	599.4
(%)	(21.8)	(14.3)	(3.2)	(1.8)	(27.4)	(18.45)	(3.0)	(1.9)
Vehicles > 3.8 tons	493.8	12,152.8	24.3	4,007.3	155.6	3,546.7	5.1	871.0
(%)	(13.5)	(11.2)	(8.7)	(5.6)	(8.9)	(7.0)	(3.6)	(2.8)
Metrobús/Mexibús	33.9	563.0	0.4	25.7	24.6	432.5	0.3	19.3
(%)	(0.9)	(0.52)	(0.14)	(0.04)	(1.40)	(0.85)	(0.21)	(0.06)

Source: SEDEMA, 2016.

In Mexico City, and in the MCMA, among heavy-duty diesel vehicles, the main contributors to primary particle emissions are trailers, followed by buses, and then by trucks. The Metrobús fleet is a small contributor to the overall emissions in the Mexico City, not only because the vehicles are newer (mean 6 years old), but also because their technologies are one generation newer than for the rest of the fleet. For instance, Metrobús vehicles model years 2007 to 2010 are equipped with US 2004/EURO IV

technologies, whereas for the rest of the HDV categories, these model years roughly correspond to US 1998/EURO III technologies.

1.2. Activity of Heavy-Duty Fleet

For this analysis, we used activity levels (vehicle kilometers travelled, VKT) by buses, trucks and trailers that SEDEMA had used for the calculations of the Emissions Inventory, 2014 (SEDEMA, 2016). The description of activity for the HDV fleet will only include vehicles model year 1985 to 2014 (Table 3). Vehicles from model year 1984 and older are excluded from the cost-effectiveness of control alternatives analysis because the Emissions Inventory, 2014 groups them in one category of vehicles of 30 years and older, which results in aggregate emissions for a wide range of technologies.

Table 3. Activity Level (VKT) by Vehicle Category for Heavy-Duty Vehicles (Model Years 1985 to 2014)

Vehicle Category	Transportation Buses-Local				Transportation Buses		Delivery Trucks > 3.8 tons		Long-Haul Tractor Trailer > 27.2 tons	
	RTP Public	School & Personnel	Concession	Metrobús	Tourism	Passenger				
Type of Plate	Local				Federal		Local	Federal	Local	Federal
Activity Level (VKT)										
n	1,215	1,171	6,391	376	10,330	14,245	11,131	13,225	650	58,191
Mean	8,947,884	779,217	16,696,907	3,396,089	2,204,225	4,463,486	4,192,822	3,785,620	435,889	16,657,765
Std. Dev.	5,512,843	857,365	18,301,222	2,222,817	1,359,436	4,864,202	4,629,770	2,506,490	627,397	11,015,498
Median	7,046,243	477,373	10,319,452	2,848,478	1,679,734	3,089,503	2,614,615	3,095,161	154,080	13,676,710
MAX	18,190,893	3,261,721	54,180,192	6,978,687	5,126,689	19,965,836	18,950,654	10,134,790	3,107,658	44,477,095
Q3	9,388,106	1,184,521	34,036,953	4,984,888	3,420,709	6,865,214	7,265,505	5,165,991	785,079	22,734,793
Q1	6,068,507	74,465	632,812	2,204,799	1,084,828	538,940	325,728	1,771,400	35,294	7,867,647
MIN	4,045,671	16,860	63,281	71,211	279,956	25,067	17,647	450,025	17,647	1,972,611

Notes: The Emissions Inventory, 2014 estimated pollutant emissions for vehicles older than 1985 pooled into one group per vehicle category. We excluded vehicles from such year group from our analysis (n=17,193). Delivery Trucks > 3.8 tons with local plates weigh between 4.6 to 27.2 tons, those with federal plates weigh from 11.8 to 14.9 tons.
Source: SEDEMA, 2016.

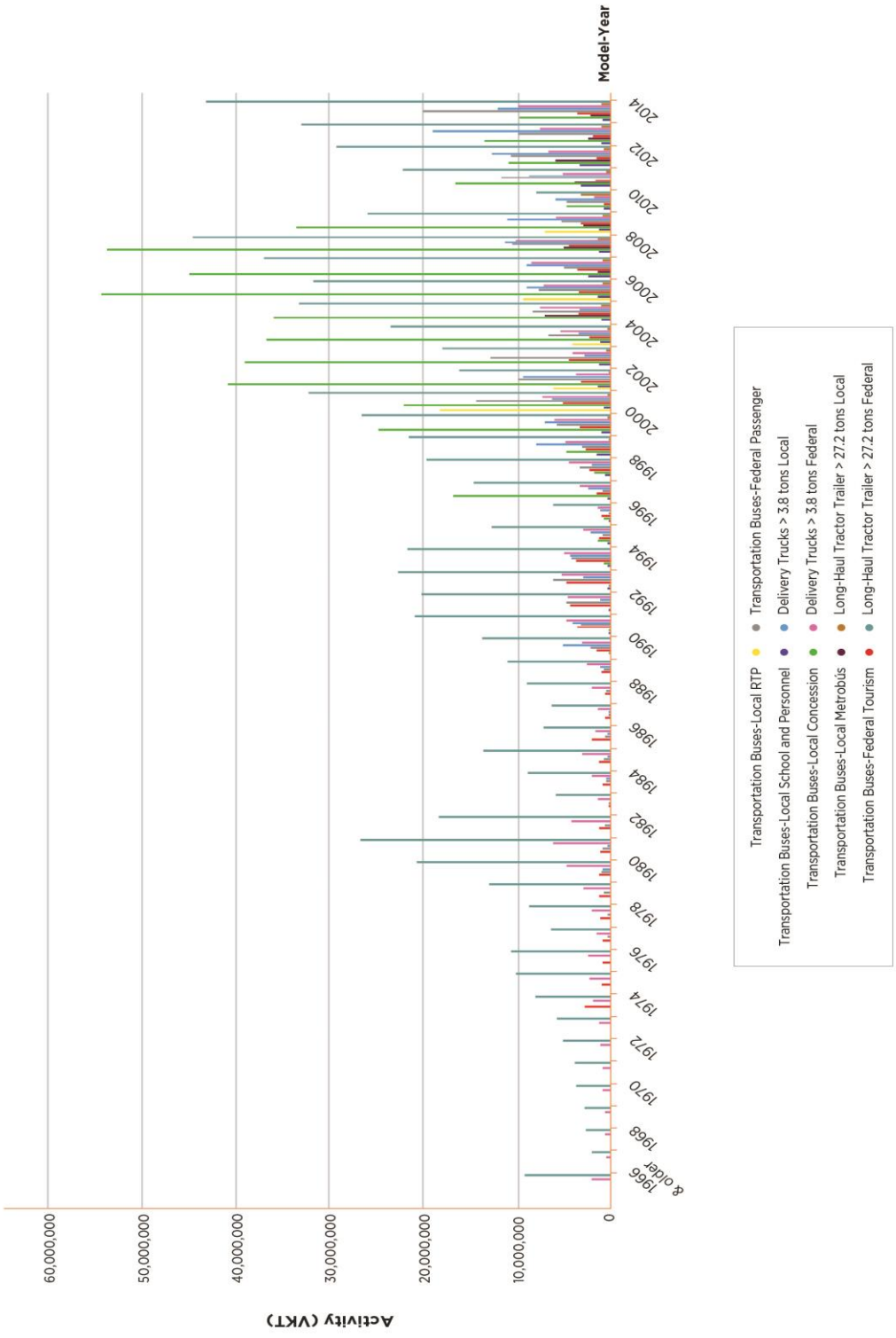


Figure 2. HDV Activity by Category and Model-Years 1985-2014 (VKT)

Source: Elaborated by authors with data from SEDEMA, 2016.

SEDEMA is currently conducting a study to improve the quality of input activity and emissions data to have better estimates and less uncertainty associated with the emissions inventory results (MOVES). This will be achieved by using observations, including activity and emissions estimates from remote sensing measuring methods, and satellite imagery. Future emissions inventories will profit from these improvements.

Annual kilometers traveled within Mexico City vary considerably between the three vehicle broad categories, and within these categories. From Table 3 and Figure 2 we find that concession buses-local plate and long-haul trailers-federal plate account for the most VKT, with averages exceeding 16 million VKT. RTP buses-local followed in activity levels, with an average of almost 9 million VKT. In sharp contrast, the average activity levels for school & personnel buses-local plate and for long-haul trailers-local plate were only 800,000 and 440,000 VKT, respectively.

2. Alternatives to Control Emissions of Air Pollutants: Retrofit Technologies

Emissions control technologies, including diesel particulate filters (DPFs) and diesel oxidation catalysts (DOCs), can significantly reduce diesel particulate matter emissions. In this analysis, we consider three main technologies: a catalyzed or passive DPF, an active regeneration DPF, and a DOC.

Diesel particulate filters reduce particulate matter, hydrocarbon and carbon monoxide (CO) emissions more than oxidation catalysts. DPF can reduce PM emissions by 85 to 90% or more, while also significantly reducing hydrocarbon (75-80%) and CO (75-80%) emissions. In contrast, DOC reduce PM emissions by only 20 to 26%, hydrocarbon emissions by 50 to 66%, and CO emissions by 40 to 50%. As these control devices mainly control primary particles, our analyses will focus in the cost-effectiveness analysis of diesel particulate matter emission reductions.

All DPFs trap particulate matter and must undergo a process called “filter regeneration” to burn it off (releasing carbon dioxide and water). This process cleans the trap and avoids clogging, which would result in high back-pressure affecting the engine performance. There are two different technologies to regenerate the filter – catalyzed or passive regeneration and active regeneration.

Catalyzed filters, such as the DCL International Inc. TITAN DPF, or the Mobiclean R of HUG Engineering, Inc., use a catalyst to facilitate regeneration. The catalyst allows the particulate matter to ignite at certain exhaust temperatures, typically greater than 235º C for a certain fraction of time of the applicable duty cycle. Efficiency of several brands and models of this type of trap have been verified by EPA and CARB and have been used in retrofit applications, with positive results. However, a catalyzed DPF is not compatible with pre-1994 Mexican diesel technologies. Passive DPFs require ultra-low sulfur (ULS) fuel for reliable regeneration and optimal function. ULS diesel (≤ 15 ppm) has been available in Mexico City since 2009 but is not yet available in a large portion of the country. Therefore, vehicles that drive outside of the city are not candidates for DPFs with catalytic regeneration because the fuel available outside of Mexico City may contain as much as 500 ppm sulfur.

Actively regenerating DPFs use a fuel burner or electric burner to heat the exhaust, allowing particulate matter to burn off. The ESW CleanTech Incorporated Horizon DPF and the Vista Electric Particulate Filter are examples of active DPFs that include a silicon carbide wall-flow filter and either an electric heater (ESW CleanTech Horizon) or a diesel-fueled burner assembly (Vista Electric) for regeneration of the filter. Active regeneration DPFs do not require ULS diesel. Most active DPF models are suitable for 1993/1994 and

newer vehicles, and to our knowledge only one model can be used in pre-1993 vehicles (the ESW CleanTech Horizon DPF).

Diesel oxidation catalysts are easy to retrofit and maintain. Although DOCs are less expensive, they are much less effective at removing solid PM. In fact, DOCs remove only about 25% of the fine particulate mass. DOCs achieve this by oxidizing the soluble organic fraction of the particulate matter.

These systems, DPFs or DOCs, are likely to remain effective for the life of the vehicle, generally five to ten years or 10,000 or more hours of operation. These figures are roughly comparable to the warranty duration (200,000 km). Some experts report that performance can be maintained for as much as 10 to 15 years, depending on the vehicle or equipment application (Jim Blubaugh, 2017). Anecdotal information points to even longer lifetimes; tests with buses in long distance operations show that DOCs and DPFs performed well and were still meeting emission-reduction requirements after more than 600 000 km (Lennart Erlandsson, 2017).

3. Emissions Limits and Model Year Heavy Duty Vehicles in Mexico

This section presents an overview of the standards that limit pollutant emissions in heavy-duty engines for the US, and their equivalent standards in Mexico. In response to these increasingly tight standards, technologies have been developed to reduce emissions. Standards are enacted and enter into force with an established lag time to allow industry to adjust their production. So, depending on when the standard enters into force it may be possible to equate which model year vehicles have introduced certain technologies to be able to comply with certain emission limits. This is relevant to our analyses, since emissions limits are used to estimate the contribution of vehicles in emission inventories.

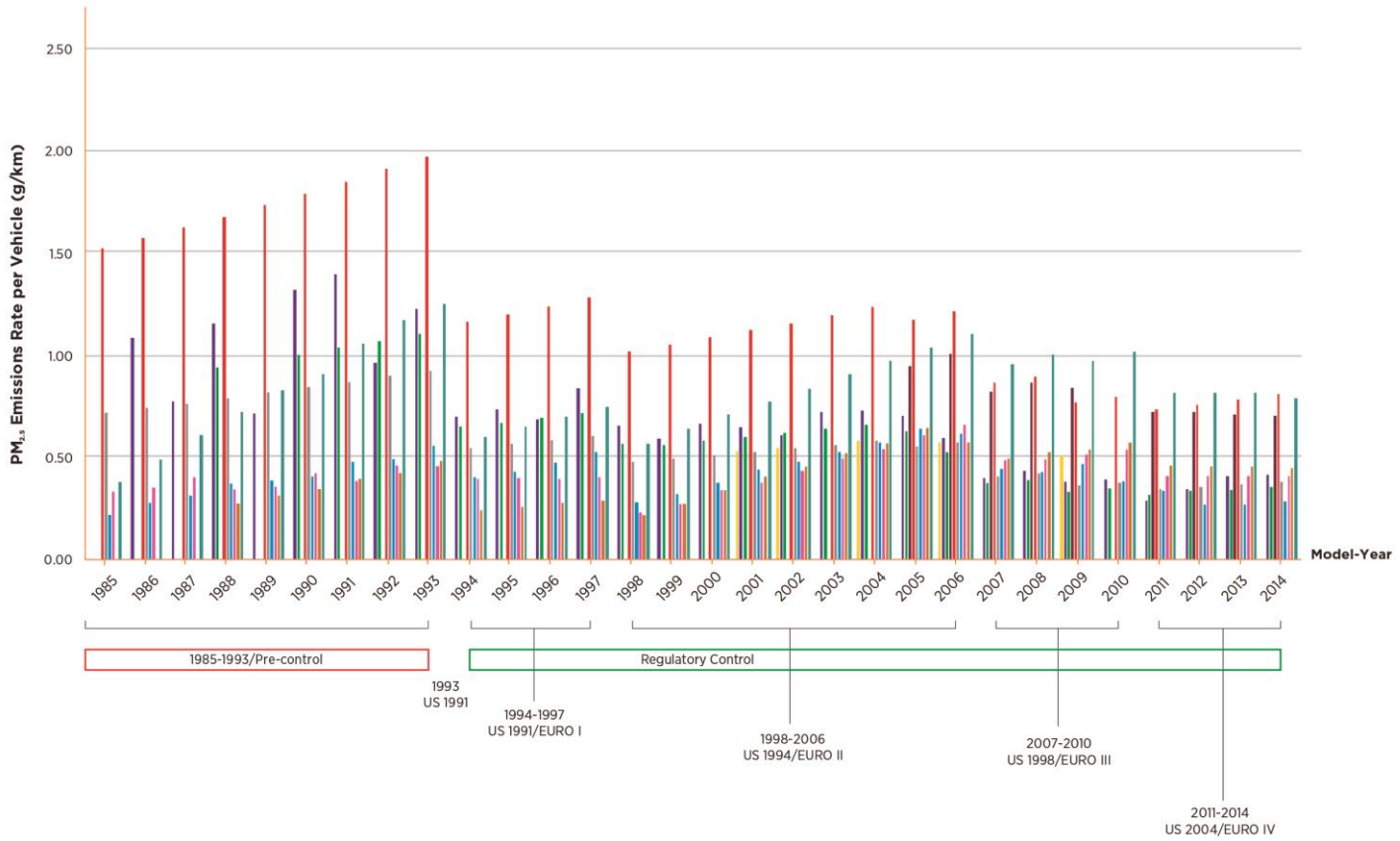
As has happened in other countries, in Mexico heavy-duty vehicle emissions have decreased over the years in response to regulations to limit exhaust emissions of gases and particles. The US 1991 standard, with which emissions controls were required for the first time, applied to model year 1992 vehicles. In México, an equivalent standard was enacted in 1993. Since then, tighter emission limits have been published in both countries, although in Mexico with delays.

Table 4. US and Mexican Model Years and Emission Standards for Heavy-Duty Engines

US Model-Years	Equivalent US Standard	Required in Mexico by NOM-044
1980-1991	Pre-control (Before 1991)	Pre-control (Before 1993)
1991-1993	US 1991	1993 (NOM-044-1993)
1994-1997	US 1994	1994-1997 (NOM-044-1993)
1998-2003	US 1998	1998-2005 (NOM-044-1993) 2006-2008 (NOM-044-2006 A)
2004-2006	US 2004	2009-2017 (NOM-044-2006 B)
2007-2019	US 2007	2018 and newer (NOM-044-2017)
2010 and newer	US 2010	Not yet required

Table 4 shows emissions regulations in the US and the equivalent standards for Mexico. The Emissions Inventories for Mexico City are estimated by SEDEMA; emission rates from the US are adjusted by model year to account for differences between the US and Mexico's heavy-duty engine standards. All vehicles included in the Mexico City's Emissions Inventory, 2014 have emissions rates that correspond to US model-year vehicles before DPFs were required by US corresponding emissions standards.

Figure 3. $PM_{2.5}$ Emissions Rates per Category and Model-Year Groups, 1985-2014



Transportation Buses-Local RTP	Transportation Buses-Federal Passenger
Transportation Buses-Local School and Personnel	Delivery Trucks > 3.8 tons Local
Transportation Buses-Local Concession	Delivery Trucks > 3.8 tons Federal
Transportation Buses-Local Metrobús	Long-Haul Tractor Trailer > 27.2 tons Local
Transportation Buses-Federal Tourism	Long-Haul Tractor Trailer > 27.2 tons Federal

Equivalent US European Standards	Equivalent Mexican Standard	Model-Year Groups for Cost-Effectiveness Analysis
Pre-control	Pre-control	1985-1993
US 1991/EURO I	NOM-044-1993	1994-1997
US 1994/EURO II	NOM-044-1993	1998-2006
US 1998/EURO III	NOM-044-2006	2007-2010
US 2004/EURO IV	NOM-044-2006	2011-2014

Source: Elaborated by authors with data from SEDEMA, 2016; Dieselnet 2017; DOF, 1993; DOF, 2006

4. Cost-Effectiveness of Potential Controls

This section of the report explores the cost-effectiveness of the technologies to control emissions of primary fine particles from heavy-duty diesel vehicles in Mexico City. The benefits of such controls are the expected improvements in ambient air quality and the associated reductions in mortality.

The costs of control include both capital costs (i.e., the cost of the equipment and its installation) and annual operating and maintenance costs (i.e., costs associated with reduced fuel economy and periodic inspection and maintenance of the equipment).

Our analysis, conducted using the software Analytica, involves five major elements: (i) analysis of the efficiency of each potential control for reducing emissions of primary fine particles; (ii) analysis of the costs of each potential control; (iii) characterization of the impacts of emissions reductions on ambient PM concentrations; (iv) analysis of the reductions in mortality expected to result from these improvements in ambient air quality; and (v) monetization of these health benefits and comparison of benefits and costs.

The unit of analysis is a single vehicle. In the current analyses, we include vehicles from model years 1985 to 2014.[6] We evaluate representative vehicles from each of ten vehicle classes and five model-year groups – intended to span the range of vehicle types, uses and model years in the heavy-duty fleet operating in Mexico City. The vehicle classes are: bus RTP public transportation – local plate; bus school and personnel – local plate; bus concession – local plate; Metrobús – local plate; bus tourism – federal plate; bus passenger – federal plate; truck – local plate; truck – federal plate; long-haul trailer – local plate; and long-haul trailer – federal plate. The model-year groups are: 1985-1993 (pre-control); 1994-1997 (US 1991/Euro I); 1998-2006 (US 1994/Euro II); 2007-2010 (US 1889/Euro III); and 2011-2014 (US 2004/Euro IV).

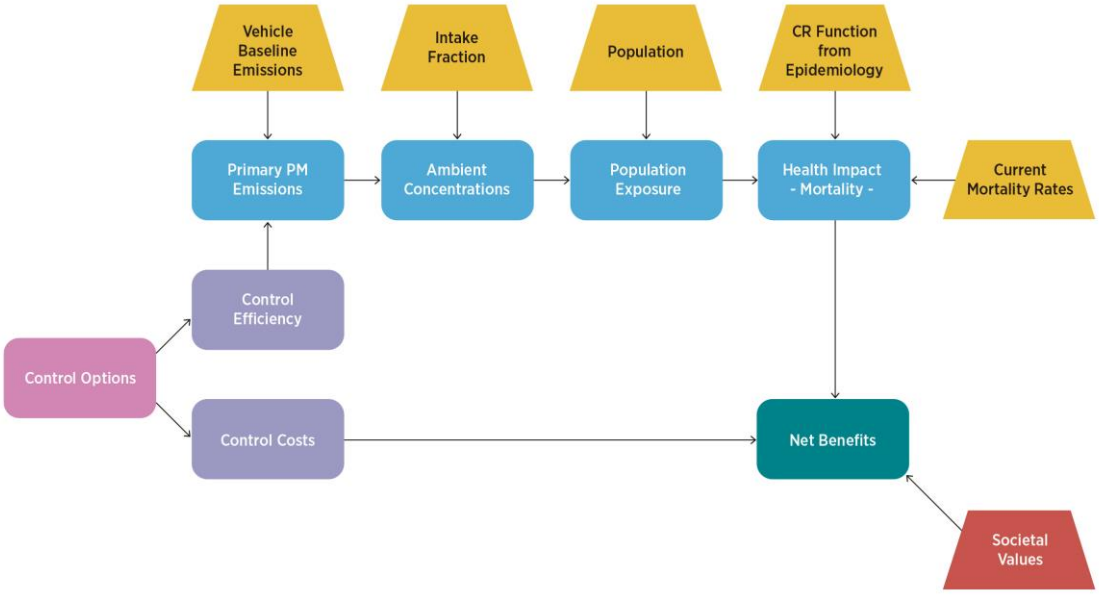
Based on their model years, RTP public transportation buses and Metrobús vehicles were assigned to the corresponding model-year groups that were formed for the rest of the heavy-duty fleet. RTP buses belong to 1998-2006 and 2007-2010 model-year groups. The Metrobús System started operations in 2005, so vehicles were assigned to the three

[6] Vehicles from model year 1984 and older are excluded from the cost-effectiveness analysis because the Emissions Inventory, 2014 groups them in one category, which results in aggregate emissions for a wide range of technologies. Also excluded are vehicles that have been retrofitted under the *Autorregulación* Program: 16 RTP buses, 2 school and personnel buses, 24 trucks with local plates, and 3 trucks with federal plates.

newest model-year groups: 1998-2006 (for model year vehicles 2005-2006), 2007-2010, and 2011-2014.[7]

Figure 4 is a schematic representation of the model and its major elements, which relate emissions from vehicles, pollutant concentrations in the atmosphere, population exposures to air pollutants, health impacts, the benefits from control options (i.e., effect on emissions reductions), and their estimated societal values in monetary units.

Figure 4. Conceptual Model for the Cost-Effectiveness Analysis to Retrofit Heavy-Duty Vehicles in Mexico City, 2014



4.1. Activity and Emissions Per Vehicle

The analysis begins by characterizing each vehicle in terms of its nature (bus, truck, tractor trailer) and age (model-year group), its activity level (vehicle km travelled each year), its baseline emissions rates (g/km travelled) and fuel economy (km/L), and its remaining useful lifetime (yr). Data on age, activity and baseline emissions rates come from the official emissions inventory for 2014 (SEDEMA, 2016). Data on fuel economy comes from U.S. Department of Energy (2015). Using this information, baseline annual

[7] The technologies of Metrobús vehicles are one generation newer than those of the rest of the heavy-duty fleet (SEDEMA, 2017a): 2005-2006 vehicles correspond to US 1998/Euro III, 2007-2010 to US 2004/Euro IV, and 2011-2014 to US 2007/Euro V. As the cost-effectiveness analysis is based on the Emissions Inventory, 2014 emissions data for each vehicle type, for simplicity our report labels the model-year groups with the technologies (corresponding US and Euro standards) of most of the fleet.

emissions (g/yr) for primary PM are computed. Appendix II provides a complete set of tables giving the properties of vehicles of each type from each model-year group.

Table 5, shown below, provides information on the size and composition of the heavy-duty diesel fleet operating in Mexico City, by vehicle type and model-year group.

Table 5. Heavy-Duty Fleet in Mexico City Included in Cost-Effectiveness Analysis. Number and Composition by Vehicle Type and Model-year group, 2014

Vehicle Type		1985-93 Pre-Control	1994-97 US 1991/ EURO I	1998-06 US 1994/ EURO II	2007-10 US 1998/ EURO III	2011-14 US 2004/ EURO IV	All Model Years
Transportation Buses	RTP Public Transport Local Plate	0	0	949	250	0	1,199
	School & Personnel Local Plate	55	65	490	227	324	1,161
	Concession Local Plate	12	310	3,770	1,669	626	6,387
	Metrobús Local Plate	0	0	99	129	148	376
	Tourism Federal Plate	2,250	861	3,465	1,343	992	8,911
	Passenger Federal Plate	1,491	491	5,722	2,028	4,155	13,887
Delivery Trucks >3.8 tons	Trucks Local Plate	1,162	750	3,893	2,367	2,763	10,935
	Trucks Federal Plate	2,013	893	3,603	1,862	2,065	10,436
Long-Haul Tractor Trailers >27.2 tons	Trailers Local Plate	13	9	206	273	139	640
	Trailers Federal Plate	8,864	3,929	15,839	8,207	9,088	45,927
All Vehicle Types		15,860	7,308	38,036	18,355	20,300	99,859

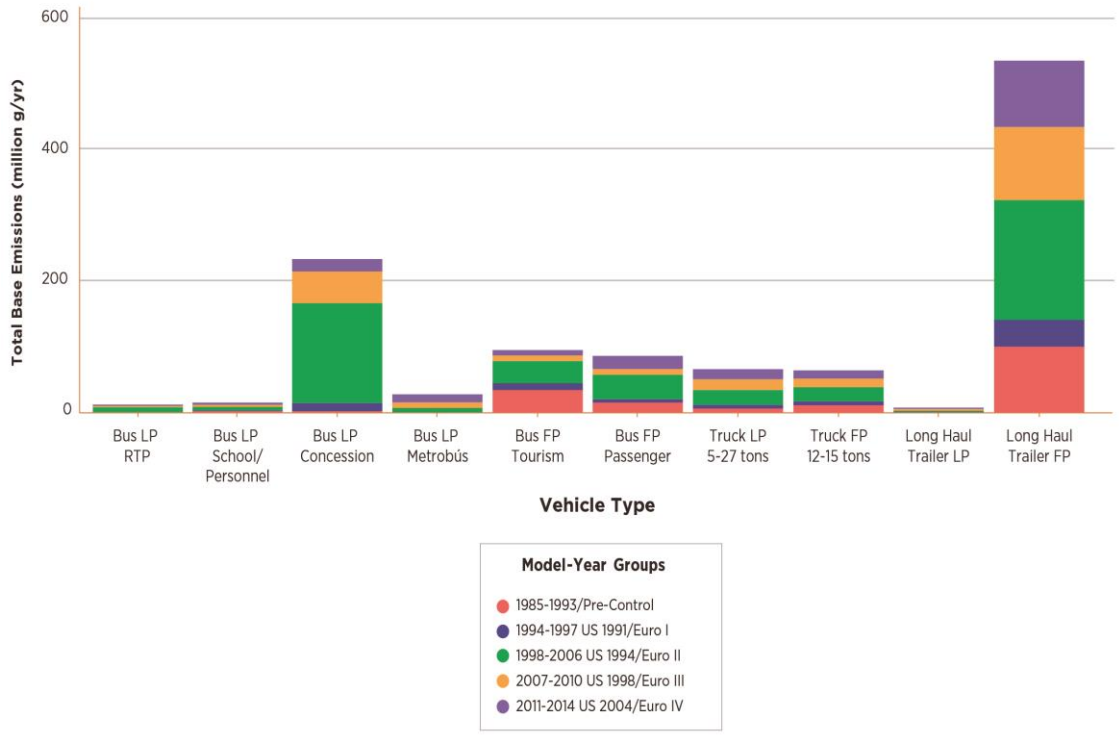
Notes: Vehicles from model year 1984 and older are excluded from the cost-effectiveness analysis because the Emissions Inventory, 2014 groups them in one category, which results in aggregate emissions for a wide range of technologies. Also excluded are vehicles that have been retrofitted under the *Autorregulación* Program: 16 RTP buses, 2 school and personnel buses, 24 trucks with local plates, and 3 trucks with federal plates. RTP buses belong to two model-year groups, 1998-2006 and 2007-2010, and Metrobús System vehicles to three model-year groups, 1998-2006, 2007-2010, and 2011-2014. Delivery Trucks > 3.8 tons with local plates weigh between 4.6 and 27.2 tons, those with federal plates weigh from 11.8 to 14.9 tons.

There are roughly 100,000 heavy-duty diesel trucks and buses from model years 1985 to 2014 that are still in operation. Long-haul tractor trailers make up almost half of the fleet, with virtually all having federal plates. Buses account for about one third of the fleet, with two thirds of these having federal plates serving as tourism or passenger buses. Trucks, split equally between those with local plates and federal plates, account for the remaining 20% of the fleet. The heavy-duty diesel fleet is relatively old. Roughly

60% of the vehicles are more than 10 years old, with two thirds of these more than 20 years old. Only 20% of vehicles are in the most recent model-year group – between 3 and 7 years old.

Estimated annual emissions of primary particles by each type of vehicle and model-year group are depicted in Figure 5.

Figure 5. Annual Emissions of Primary Particles by Vehicle Type and Model-Year Group



Note that of the estimated total annual emissions of primary particles of ~ 1000 metric tons, more than 50% is due to long-haul trailers with federal plates, another 25% is due to concession buses with local plates. The remaining 20-25% of primary particle emissions is roughly equally split between buses (both tourism and passenger) with federal plates, and trucks (with both local and federal plates). Two categories of vehicles – school & personnel buses with local plates, and long-haul trailers with local plates make inconsequential contributions to primary particle emissions.

Among the two vehicle types which dominate emissions of primary particles, 1998-2006 EPA 1994/Euro II model year vehicles contribute most substantially, followed by 2007-2010 EPA 1998/Euro III model year vehicles, and then, almost equally, by 1985-1993 pre-control model year vehicles, and 2011-2014 US 2004/Euro IV.

4.2. Controls: Efficiency and Cost

Once the vehicle is characterized, attention turns to determining which controls are potentially applicable and then to estimating their costs. Our analysis considers four possible controls – (i) oxidation catalyst, (ii) diesel particulate filter, active regeneration, (iii) diesel particulate filter, catalyzed, and (iv) an ideal control – i.e., one which is 100% efficient in reducing emissions of primary PM and which has no cost. The ideal control provides an upper bound on the net benefits of any possible emission-control technology.

Information on the efficiency of each control for reducing primary PM emissions came from CARB Diesel Certification & Verification Procedure, and technology-specific corresponding Executive Orders (CARB, 2013, 2014, 2015a and 2015b). We have assumed that since ultra-low sulfur fuel is the only type of diesel fuel available in Mexico City that the introduction of retrofit technology has no impact on SO_2 emissions. Similarly, we assume that oxidation catalysts and diesel particulate have no impact on NOx emissions.

Estimates of the capital costs are taken from recent SEDEMA bids for diesel retrofit devices (SEDEMA, 2017b).[8] Annual maintenance costs are from a SEDEMA quote from HUG Engineering (SEDEMA,2017), and estimates of the fuel use penalties for each control device came from MECA (1999) in Stevens et al. (2005).

Table 6 below, summarizes the data on the costs and efficiency of the control devices considered in our current analysis.

Table 6. Costs and Efficiency of Control Retrofit Technologies for Heavy-Duty Diesel Vehicles

		Diesel Oxidation Catalyst	Diesel Particulate Filter Active Regeneration	Diesel Particulate Filter Catalyzed	Hypothetical Perfect Control
Capital Cost (1000 US$/veh)		0.5 - 1.5	7.0 - 9.0	6.0 - 8.0	--
Lifetime of Equipment (y)		10[9]	10	10	--
Annual Maintenance Cost (US$/veh-y)		--	220	220	--
Fuel Use Penalty (fractional)		--	0.02	0.004	--
Control Efficiency (fractional)	PM	0.20 - 0.26	0.8 - 0.9	0.8 - 0.9	1.00

[8] The bids submitted to SEDEMA were for retrofit equipment that combined diesel particulate filters with oxidation catalysts. We subtracted the median estimate of the cost of an oxidation catalyst, $1000, from each bid to estimate the cost of diesel particulate filters for application in Mexico City.

The equivalent annual control cost for each device was computed by converting the capital cost to an equivalent annual cost stream using the capital recovery factor and adding the result to the annual maintenance cost and any additional cost related to the decreased fuel economy of vehicles equipped with DPFs. The discount rate used in our analysis was 3% per year. The cost of (ultra-low sulfur) fuel used to compute the fuel use penalty was taken as 1.01 US$ per Liter (INPC, 2017).

$$EAC = C * crf + M + CIFU$$

where EAC is the equivalent annual cost (US$/veh-y); C is the capital cost (US$/veh); crf is the capital recovery factor which depends on the lifetime of the equipment, L (y), and the discount rate, r (fraction/y); M is the annual maintenance cost (US$/veh-yr); and CIFU is the cost of increased fuel use (US$/veh-yr).

The capital recovery factor is given by:

$$crf = \frac{r*(1+r)^L}{(1+r)^L - 1}.$$

The cost of increased fuel use is given by:

$$CIFU = P * F * A/E$$

where P is the price (US$/L) of fuel; F is the fuel use penalty (fractional increase); A is the activity level (km/veh-y); and E is the baseline fuel economy (km/L).

The emissions $E_{i,j,k}$ (g/y) of the j^{th} pollutant from the i^{th} vehicle type expected after implementation of the k^{th} control are given by:

$$E_{i,j,k} = \left(1 - \varepsilon_{j,k}\right) * Eo_{i,j}$$

Where $\varepsilon_{j,k}$ is the control efficiency (fractional) of the k^{th} control for the j^{th} pollutant, and $Eo_{i,j}$ (g/y) represents the uncontrolled emissions of the j^{th} pollutant from the i^{th} vehicle type.

4.3. Population Exposure. Intake Fraction and Primary Particle Concentrations

Once the uncontrolled emissions, and the emissions with the implementation of each control device, are known, the vehicle's contribution to population exposure may be estimated using the concept of intake fraction.

Intake fraction, which is the simplest measure of the relationship between emissions and exposure, is defined as the ratio of the population intake of a pollutant (g/y) divided

by the emissions (g/y) of the pollutant or a precursor. Intake fractions depend on all the factors which influence the relationship between emissions and exposure. These include the nature and location of the source (whether it is ground level or elevated; whether it is located in a densely populated city or in a rural area); the pollutant (whether it is conservative – i.e., has a low deposition velocity, does not react chemically with other pollutants – or reactive – i.e., has a short atmospheric half-life) and the atmosphere to which it is emitted (e.g., the wind speed, the mixing height); and the receptors (for example, the population density).

Intake fractions may be estimated using atmospheric fate and transport models or by combining results from source-receptor analysis with information from emissions inventories. Because our emission controls do not affect NOx or SO_2 we only care about the primary $PM_{2.5}$ intake fractions. Intake fractions may be greater for emissions within the city than for emissions outside of the city – especially for primary $PM_{2.5}$ emitted by vehicles.

Table 7 summarizes the estimates of intake fractions used in our central analysis for exposures within the Mexico City Metropolitan Area. For primary $PM_{2.5}$ our central analysis relies on the intake fraction estimates of Stevens et al., 2007. Their estimates of intake fractions for emissions within Mexico City reflect the entire Mexico City Metropolitan Area population of 18 million and use a nominal breathing rate of 20 m^3/day.

Table 7. Primary $PM_{2.5}$ Intake Fraction Estimates for Emissions within Mexico City

Pollutant	Intake Fraction (ppm)
Primary $PM_{2.5}$	Triangular distribution Mode = 60 Range = 30 to 120

Source: Stevens et al., 2007.

Stevens et al. (2007) applied four approaches (a static box model, a dynamic box model, a regression approach, and a method which estimated iF using source apportionment) that gave iF estimates varying from 30 ppm (regression) to 120 ppm (static box and source apportionment), geometrically centered at 60 ppm. Stevens and coauthors indicated that their estimate was good to within a factor of 2. Our analysis relies on a triangular distribution with a mode of 60 ppm, a minimum of 30 ppm and a maximum of 120 ppm to reflect their results.

Using these estimates of intake fraction and the emissions estimates discussed previously, the city-wide average annual concentration change, $\Delta C_{i,j}$ (μg/m^3), due to the emissions, $E_{i,j}$, of the pollutant from the ith vehicle type under the jth control are given by:

$$\Delta C_{i,j} = iF_j * E_{i,j} / (P * B * 365)$$

where iF is the intake fraction, P is the population (persons), B is the nominal breathing rate (m^3/person-day) and 365 is the constant needed to convert the daily breathing rate to an annual breathing rate.

4.4. Health Impact: Concentration-Response Function

The impact on mortality of the incremental air pollution exposure caused by emissions from a representative vehicle is computed using the integrated exposure response function (IER) developed to support the 2010 and 2013 Global Burden of Disease analysis (Burnett et al., 2014). The nature of the IER and the rationale for using it in this analysis were described in detail in a previous report (*Phase II. Estimation of the Health Benefits of Air Pollution Improvements*).

Current evidence suggests that, among adults, mortality rates from four causes of disease – Ischemic Heart disease (IHD), Cerebrovascular Stroke (STK), Chronic Obstructive Pulmonary Disease (COPD), and Trachea, Bronchus and Lung Cancers (LC) – are elevated by chronic exposure to airborne $PM_{2.5}$. In addition, among young children, mortality rates from Acute Lower Respiratory Infections are elevated among those with chronic $PM_{2.5}$ exposure. The IER gives the relative risk (i.e., the mortality rate among the exposed divided by the mortality rate among the unexposed), RR (dimensionless), as:

$$for\ C \geq Xo, \qquad RR = 1 + \alpha * (1 - \exp(-\beta * (C - Xo)^{\delta}))$$

where α is the asymptotic limit of RR as $PM_{2.5}$ approaches infinity, β indicates the rate of increase per unit increase in $PM_{2.5}$, Xo is the counterfactual level (the $PM_{2.5}$ concentration below which there is no known increase in risk), δ (dimensionless) is the power, and C is the annual average concentration (μg/m^3) of $PM_{2.5}$.

Values of the parameters – i.e., α, β, Xo, and δ – for each disease and age-group of interest have been estimated by Burnett (2014).[9] Burnett et al. have carefully analyzed

[9] For ischemic heart disease and for stroke, parameters have been estimated for each of 12 five-year age groups (25-29, 30-34, …, 75-79, and ≥ 80). For chronic obstructive pulmonary disease and for lung cancer, a single set of non-age specific parameters applies to all deaths of persons 25 or more years of age. For acute lower respiratory infections in young children, a single set of parameters applies to all children younger than 5 years of age.

the uncertainty in the parameters and provide a set of 1000 equally-likely sets of the parameter values for each disease and age-group.

Here we rely on a linear approximation to the IER. Rather than computing the RR using the IER for each of the 1000 equally likely sets of parameter values for each of the five diseases and twelve age groups of interest, we assume that for small increments or decrements in $PM_{2.5}$ the change in relative risk can be approximated well by the product of the slope of the tangent to the IER evaluated at current levels of $PM_{2.5}$ in Mexico City.

As a part of our Phase II analysis, we probabilistically characterized the slope (% increase in RR per $\mu g/m^3$) of the IER for each of the five diseases of interest at two levels of $PM_{2.5}$: 20 $\mu g/m^3$ and 50 $\mu g/m^3$. This was done numerically by evaluating:

$$\text{Slope of RR @ Co} = [\text{RR (Co + 1)} - \text{RR (Co)}] \,/\, [(\text{Co + 1}) - (\text{Co})]$$

The slope was characterized for each of the five diseases of interest using each of the 1000 equally likely sets of disease-and-age-specific parameters – α, β, Xo, δ. Summary slopes for application in Mexico City and in the Mexico City Metropolitan Area (excluding the city) were then computed by weighting the disease-and-age-specific slopes obtained above by the disease-and-age-specific mortality rates in CDMX and in the MCMA (minus CDMX). Fifteen parameters (min, 1%, 2.5%, 5%, 10%, 25%, 33%, 50%, 67%, 75%, 90%, 95%, 97.5%, 99% and max) of each of these distributions were extracted and were used to probabilistically characterize the summary slopes for CDMX and MCMA (minus CDMX) in our calculations of the impacts of control options.

The annual average $PM_{2.5}$ level in Mexico City in 2014 was 22.8 $\mu g/m^3$ (SEDEMA, 2015). As a result, we use the disease-and-age-weighted slope @ 20 $\mu g/m^3$ in our calculations.

Table 8. Relative Risk of Mortality and Slope of the Integrated Exposure-Response Function in Mexico City and in the Mexico City Metropolitan Area (outside of the City) at 20 µg/m³

Parameter	RR @ 20 µg/m³		Slope @ 20 µg/m³ (% increase in RR per µg/m³ $PM_{2.5}$)	
	CDMX	MCMA (minus CDMX)	CDMX	MCMA (minus CDMX)
2.5%	1.209	1.218	0.753	0.678
25%	1.229	1.236	0.880	0.792
50%	1.239	1.247	0.962	0.880
75%	1.250	1.257	1.042	0.974
97.5%	1.274	1.278	1.234	1.182

Table 8 provides probabilistic characterizations (median, 25% and 75%, 2.5% and 97.5%) of the disease-and age-weighted relative risk (RR) of mortality and slope of the function, across all diseases and age groups of interest, for CDMX and MCMA (minus CDMX) given by the integrated exposure-response function at an ambient $PM_{2.5}$ concentration of 20 $\mu g/m^3$.[10] Table 9 provides the mortality rates used in computing the summary slope.

Finally, we introduced a cessation lag[11] in our benefit calculation. When reducing $PM_{2.5}$ emissions via retrofitting heavy-duty vehicles we aim at reducing the risk of the diseases that are associated with $PM_{2.5}$ chronic exposures. The reduction of risk may start immediately after the emissions are reduced and may continue for some time. In practice, the $PM_{2.5}$ cessation lag effect is estimated by assigning a fraction of avoided deaths attributable to the $PM_{2.5}$ exposure (i.e., the benefits) every year after cessation (or reduction) of the exposure. In our analysis, the lag structure allocates 20% of the benefits in the first year, 50% equally divided in the following four years, and an even distribution of the remaining 30% in the following 15 years (HES, 2004).

Table 9. Cause-Specific Mortality Rates in Mexico City and in the Mexico City Metropolitan Area (excluding the city), 2014

Disease	Mortality Rate in 2014 (deaths / year)	
	CDMX	MCMA (minus CDMX)
Ischemic Heart Disease	9,851	6,376
Cerebrovascular Stroke	1,195	1,069
Chronic Obstructive Pulmonary Disease	2,012	1,953
Trachea, Bronchus and Lung Cancers	667	491
Acute Lower Respiratory Infections	168	258
All Diseases of Interest	13,893	24,045

4.5. Economic Impact: Monetization of Health Impact

The monetary value of the reduction in mortality risk is calculated by multiplying the population risk reduction (i.e., the reduction in deaths attributed to PM) times the rate at which mortality risk is valued, the Value per Statistical Life (VSL). We estimate VSL

[10] Our Analytica calculations use a more complete parameterization of the distribution of slopes – including the minimum, 1%, 2.5%, 5%, 10%, 25%, 33%, 50%, 67%, 75%, 90%, 95%, 97.5%, 99% and maximum.

[11] This concept refers to the reductions over time in the risks of mortality that are expected after the exposure to ambient $PM_{2.5}$ is reduced (HES, 2004).

following recommendations developed for conducting benefit-cost analysis (BCA) in low- and middle-income countries supported by the Gates Foundation. Robinson, Hammitt and O'Keefe (2018) suggest that, when high-quality direct estimates of VSL are not available, analysts should extrapolate from values estimated for the United States, adjusting for the difference in average income between the US and the target country. They recommend (i) using purchasing-power-parity (PPP) rather than market exchange rates to compare incomes; (ii) to use one of two values of income elasticity (1.0 or 1.5), chosen based on the ratio of incomes; and to extrapolate from ratios of VSL to income of 160 and 100 (based on US and OECD values) or from a ratio of 160.

We apply these methods to both Mexico City and the MCMA outside of the city boundaries and assume the lowest and highest estimates for each region span an 80 percent credibility interval.

The extrapolated ratios are calculated as follows:

$$VSL_M/y_M = (y_M/y_{US})^{h-1}\, VSL_{US}/y_{US}$$

Where y is income, and h is the income elasticity.

For income, we use GDP per capita in Mexico City itself and in the MCMA outside of the city proper. We adjust to US dollars using PPP and obtain US $37,500 for Mexico City and US $14,600 and in the MCMA outside of the city proper (INEGI, 2017).

Table 10. Estimates of the Value of Statistical Life for Mexico City and for the MCMA (minus CDMX)

Value per Statistical Life (million US$ / unit change in mortality risk)		
Lognormal Distribution Parameters	**CDMX**	**MCMA (minus CDMX)**
Median	4.7	1.7
GSD	1.4	1.7
Mean	5.1	1.9

Note: GSD=Geometric Standard Deviation. Computations used GDP per capita for CDMX = 37,500 US$ and for MCMA (minus CDMX) = 14,600 US$.

For Mexico City we characterize VSL using a lognormal distribution with a median of 4.7 million US$ and a geometric standard deviation of 1.4 (implying a mean of 5.1 million US$) (Table 10). For the area in the MCMA outside of the city proper, we characterize VSL using a lognormal distribution with a median of 1.7 million US$ and a geometric standard deviation of 1.7 (implying a mean of 1.9 million US$).[12]

For comparison, the only study estimating VSL in Mexico of which we are aware is Hammitt and Ibarrarán (2006). They estimated VSL in Mexico as $230,000 and $310,000 for an average income of $4100. Table 10 summarizes the estimates of the VSL used to monetize mortality impacts.

[12] The population weighted mean VSL for the entire MCMA is 3.3 million US$.

5. Results: Costs, Emissions Reductions & Health Benefits

Air pollution ignores political boundaries. Emissions within the City lead to exposures and health risks in the City and throughout the MCMA metropolitan area. Because of this the results presented in this analysis consider the benefits in the Mexico City Metropolitan Area, that is, benefits are extended outside of Mexico City. For each type of vehicle and model-year group tables of results have been constructed. These include the emissions reductions (kg/veh-yr), the attributable deaths avoided (#/1000 veh-yr), the monetized benefits of the avoided deaths (1000 US$/veh-yr), the control costs (1000 US$/veh-yr), and the net benefits (1000 US$/veh-yr) for the *status quo*, each of the three control technologies, and an ideal control.

Tables 11 and 12 give illustrative results for the two most important categories of vehicles in terms of emissions (bus concession – local plate –and long-haul trailer – federal plate) for one model-year group (1998-2006 EU 1994/Euro II). Appendix III provides a complete set of results for all vehicle types and model-year groups.

For the approximately 4 thousand concession buses with local plates, the largest *expected* net benefits are generated by choosing to retrofit with a catalyzed DPF (Table 11). These vehicles are heavily used, each traveling roughly 70 thousand km per year. The catalyzed DPF retrofit is expected to reduce emissions by 35.6 kg per vehicle-year; and to reduce premature deaths attributable to air pollution by about 3 per 1000 vehicle-year; with benefits of US$ 9.2 thousand and costs of only 1.4 thousand US$ per vehicle-year. The catalyzed DPF is an option because these buses are driven only locally, where ultra-low sulfur fuel is available. The expected net benefits of this strategy (health benefits minus control costs) are almost 8 thousand US$ per vehicle year.

Table 11. Results for Bus Concession – Local Plate
Model Years 1998 to 2006 US 1994/Euro II

	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/veh-yr)	Control Cost (1000 USD/veh-yr)	Net Benefits (1000 USD/veh-yr
Status Quo	0.00	0.00	0.00	0.00	0.00
DOC	9.35	0.83	2.41	0.14	2.27
DPF -- Active	35.56	3.14	9.17	2.42	6.75
DPF -- Passive	35.56	3.14	9.17	1.43	7.74
Ideal Control	40.64	3.59	10.48	0.00	10.48

Notes: DOC stands for Diesel Oxidation Catalyst; DFP-p stands for Diesel Particulate Filter with catalyzed regeneration (passive), and DFP-a stands for Diesel Particulate Filter with active regeneration. Rows in green highlight the retrofit technology that maximizes the expected net benefits.

For the approximately 16 thousand long-haul trailers with federal plates the largest *expected* net benefits (almost 1.8 thousand US$ per veh-year) would be generated by choosing to retrofit with a catalyzed DPF (Table 12). This would be expected to reduce

emissions by 10.2 kg per vehicle-year; and to reduce premature deaths attributable to air pollution by approximately 1 per 1000 vehicle-year; with benefits of over 2.6 thousand US$ and costs of less than 0.9 thousand US$ per vehicle-year. Unfortunately, the catalyzed DPF is not an option because these long-haul trailers, with federal plates, are driven both in Mexico City and outside of the city, where ultra-low sulfur fuel is not widely available. The use of these trailers *within the city* is on average only 14 thousand km per vehicle-year.

Of the remaining options, the largest *expected* net benefits of close to 1.6 thousand US$ per veh-year are generated by choosing to retrofit with an active regeneration DPF. This generates the same emissions reductions and health benefits as the catalyzed DPF but has costs which are roughly 20% higher due to the larger fuel penalty associated with active regeneration of the filter.

Table 12. Results for Long-Haul Tractor Trailer – Federal Plate
Model Years 1998 - 2006 EU 1994/Euro II

	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/veh-yr)	Control Cost (1000 USD/veh-yr)	Net Benefits (1000 USD/veh-yr
Status Quo	0.00	0.00	0.00	0.00	0.00
DOC	2.68	0.24	0.69	0.09	0.60
DPF -- Active	10.18	0.90	2.63	1.06	1.56
DPF -- Passive	10.18	0.90	2.63	0.86	1.77
Ideal Control	11.64	1.03	3.00	0.00	3.00

Notes: DOC stands for Diesel Oxidation Catalyst; DPF-p stands for Diesel Particulate Filter with catalyzed regeneration (passive), and DPF-a stands for Diesel Particulate Filter with active regeneration. Rows in light gray highlight retrofit technologies that are not adequate for such vehicle type. Rows in green highlight the retrofit technology that maximizes the expected net benefits.

Following this approach, together with a careful review of the cost-effectiveness and the applicability of available control technologies for each vehicle type and model-year group, control options that maximize expected net benefits were identified. Table 13, below, provides the results.

Note that for the two categories of vehicles, bus concession - local plate and long-haul trailer - federal plate, which are responsible for the greatest share of primary PM emissions, DPF retrofits are cost-effective – providing the maximum possible expected net benefits with expected emissions reductions between 80 and 90%. Comparable results are shown for the third largest primary PM emitter, bus tourism – federal plate, for which DPF retrofits are cost-effective for all year groups.

Also note that there is no category or model year of vehicle for which some retrofit is not cost-effective. In some cases, for example trucks with local or federal plates, DPFs are not cost-effective for some model-year groups, but oxidation catalysts are, for which projected emissions reductions range between 20 and 26%. A similar result is found for

bus passenger - federal plate –the fourth largest primary PM emitters--, either DPF or DOC are cost-effective for all model-year groups.

Table 13. Retrofit options which maximize expected net benefits by vehicle type and model-year group in Mexico City, 2014, and estimated probability (%) that net benefits of indicated retrofit option are positive

Type of Vehicle & Plate		1985-93 Pre-Control	1994-97 US 1991/EURO I	1998-06 US 1994/EURO II	2007-10 US 1998/EURO III	2011-14 US 2004/EURO IV
Transportation Buses	RTP - Public Transport Local Plate	n.a.	n.a.	DPF-p 80	DOC 70	n.a.
	School & Personnel Local Plate	DPF-a 99	DPF-p 97	DPF-p 97	DPF-p 80	DPF-p 78
	Concession Local Plate	DPF-a 96	DPF-p 99	DPF-p 99	DPF-p 99	DPF-p 99
	Metrobús Local Plate	n.a.	n.a.	DPF-p 72	DPF-p 99	DPF-p 99
	Tourism Federal Plate	DPF-a 99	DPF-a 96	DPF-a 95	DPF-a 86	DPF-a 82
	Passenger Federal Plate	DPF-a 90	DPF-a 74	DPF-a 70	DOC 98	DOC 98
Delivery Trucks > 3.8 tons	Trucks Local Plate	DOC 99	DPF-p 80	DPF-p 80	DPF-p 80	DOC 96
	Trucks Federal Plate	DOC 99	DOC 99	DPF-a 65	DPF-a 74	DPF-a 58
Long-Haul Tractor Trailers > 27.2 tons	Trailers Local Plate	DOC 91	DOC 93	DPF-p 84	DPF-p 93	DPF-p 87
	Trailers Federal Plate	DPF-a 95	DPF-a 88	DPF-a 95	DPF-a 97	DPF-a 94

Notes: RTP public transportation and Metrobús vehicles have vehicles than belong to only two and three model-year groups, respectively. DOC stands for Diesel Oxidation Catalyst; DPF-p stands for Diesel Particulate Filter with catalyzed regeneration (passive), DPF-a stands for Diesel Particulate Filter with active regeneration, and n.a. stands for not applicable for vehicle categories and model-year groups for which there were no vehicles in the Emissions Inventory, 2014.

It is reasonable to ask what the aggregate benefits and costs of such a strategy would be. If fully implemented, our analysis indicates that the strategy consisting of retrofitting every vehicle with the control which maximizes *expected* net benefits for that vehicle type and model-year group, has the potential to:

- Reduce annual emissions of primary fine particles by 950 metric tons; which would
- Cut the annual population-weighted mean concentration of $PM_{2.5}$ in the Mexico City Metropolitan Area by close to 0.90 $\mu g/m^3$

- Reduce the annual number of deaths attributable to air pollution by over to 80[13]; and to
- Generate expected health benefits on the order of 250 million US$ per year.

It has *expected* annual costs of less than 93 million US$ per year – consisting of 61 million US$ in 'amortization' of capital cost of retrofit devices; 19 million US$ in annual maintenance costs; and 11 million US$ in fuel use penalties. This results in close to 150 million US$ net benefits for a fully implemented strategy of retrofitting every vehicle.

The primary results presented above for our cost-effectiveness analysis have come from the following parameters and assumptions:

(i) Uses Emission Inventory, 2014 data for fleet number, emissions, and activity;
(ii) Estimates the cost-effectiveness on a per-vehicle basis: emissions reductions (kg/veh-yr), attributable deaths avoided (#/1000 veh-yr), monetized benefits of the avoided deaths (1000 US$/veh-yr), control costs (1000 US$/veh-yr), and net benefits (1000 US$/veh-yr);
(iii) Evaluates the net benefits of three retrofit technologies, DOC, DPF-catalyzed, and DPF-active regeneration, plus an ideal control;
(iv) Uses intake fraction (iF) from Stevens et al., 2007 to estimate exposure, where intake fraction is defined as the ratio of the population intake (g/y) of a pollutant divided by the emissions (g/y) of the pollutant or its precursor;
(v) Applies the concentration-response functions from the GBD-2010 and 2013, which includes five causes of death – ischemic heart disease, cerebrovascular stroke, chronic obstructive pulmonary disease, and trachea, bronchus and lung cancers, in adults, and acute lower respiratory infections in children; and,
(vi) Estimates the monetary value of health benefits with the Value per Statistical Life (VSL) --through benefits transfer, using values from the US to estimate those for Mexico City and the MCMA, using purchasing-power-parity to compute Mexico City's and MCMA (minus CDMX) GDP in US dollars--, and uses a discount rate of 3%.

There is always uncertainty about the health benefits and costs of policies to reduce air pollution. Our analysis quantifies uncertainty about some of the most important inputs, including the relationship between emissions (in this case emission reductions) and population exposure (summarized by the intake fraction), the slope of the exposure-

[13] This is an estimate for the initial years after vehicles are retrofit. Over time, the reduction in mortality risk will change the population exposed to air pollution, increasing both the number of people at risk and their average age. Ultimately, the total number of deaths per year will rise to the level it would be without retrofits, but these deaths will occur at older ages, on average.

response functions relating mortality to air pollution, the monetary value of reductions in mortality risk (summarized by the value per statistical life), as well as the efficiency and cost of each control option.

By quantifying uncertainty about some of the most important parameters, we can estimate the probability that the net benefits of the identified retrofit program are positive, that is, that the benefits of the reduction in mortality risk exceed the cost of the specified retrofit technology. These probabilities are displayed in Table 13 for each vehicle type and model-year group. As shown there, for the vehicle type accounting for the largest share of emissions --long-haul trailers with federal plates--, the estimated probability that the value of the mortality-risk reduction associated with retrofit with active DPF exceeds the cost of the retrofit is between 88 percent and 97 percent depending on the specific model-year group. For the vehicle type accounting for the second largest share of emissions --concession buses with local plates--, the probability that retrofitting vehicles of model year 1994 or later with passive DPFs is cost effective is 99 percent and the probability that retrofitting older vehicles with active DPFs is cost effective is 96 percent.

Overall, for most vehicle type/model-year group categories, the probability that the identified retrofit option will yield benefits greater than its cost is about 80 percent or larger. The two exceptions are RTP buses with local plates, and delivery trucks with federal plates. For the first group, the probability that retrofitting 2007-2010 vehicles with oxidation catalysts yields net benefits is only 70 percent. For the second group, the probability that retrofitting newer vehicles (model years 1998 and after) with active DPF yields net benefits is estimated as 58 to 74 percent. However, this does not imply that these vehicles should not be controlled, because the probability that retrofitting these vehicles with oxidation catalysts --a less costly control technology--, yields positive net benefits is 99 percent.

In evaluating uncertainty about the net benefits of different control options, we find that uncertainty about the net benefits of DPFs is greater than about the net benefits of oxidation catalysts. This result because the DPFs reduce primary PM emissions substantially more than do DOCs, and hence uncertainty about the effect of emissions on mortality or about the monetary value of mortality risk have a larger effect on estimated benefits. Moreover, because oxidation catalysts are much less costly than DPFs, the range of benefits that is less than their costs are very small, and it is unlikely that benefits fall in this narrow range. It should be iterated that the net benefits of the passive DPF always exceed those of the active DPF, because they produce the same benefits but are less pricy, partly because the active DPFs impose a larger fuel-efficiency penalty.

6. Discussion and Conclusions

The cost-effectiveness analysis conducted for Mexico City heavy-duty vehicles clearly shows that performing retrofit with DOCs or DPFs can reduce particulate matter emissions, lead to improvements in air quality, and have public health benefits among the inhabitants of the Mexico City Metropolitan Area.

Retrofit programs have been put in place in other countries and have been on the radar of policy makers in Mexico for decades. Their success comes from the fact that diesel retrofit technologies, such as DOCs and DPFs, that can reduce diesel particulate matter, are similar in control efficiency to emission control technologies from newer diesel vehicles (ICCT, 2017). In the US, CARB implemented a mandatory retrofit program for most in-use heavy-duty diesel vehicles, and EPA, in turn, established a voluntary retrofit program. EPA's benefit-cost analysis of the program for the years 2009 to 2013 shows an estimate of 1,700 fewer deaths attributed to the reduction in pollutant emissions, with a total present value of up to $11 billion in monetized health benefits over the lifetime of the affected engines (ICCT, 2017).

Over ten years ago (2005-2006), a pilot retrofit project was conducted in Mexico City, by EMBARQ in partnership with EPA, and Mexican environmental federal and local authorities (EMBARQ-WRI, 2007). DOCs and DPFs-catalyzed were installed in 20 urban passenger buses and followed-up for 11 months; DOCs were installed in model year 1991 buses, and DPFs in model year 2001 buses. Emission reduction efficiencies were as expected; primary $PM_{2.5}$ reductions were on the order of 20 to 30% for DOCs, and 80 to 90% for DPFs. Two fundamental lessons were learned. One key to the success of the program was selecting appropriate buses for retrofitting through previous careful testing. A second essential element for success was training operators on how the emissions control devices worked, how they were installed, and driving techniques for best performance of the equipment.

More recently, the *Autorregulación* Program was put in place by SEDEMA in Mexico City. This voluntary program has succeeded in having retrofit devices installed in 45 vehicles – 27 heavy-duty trucks and 18 RTP buses.

Our current analysis seeks to determine whether expanding retrofit programs to a wide variety of diesel-fueled heavy-duty vehicles might be cost-effective. In Mexico City, there are over 100,000 heavy-duty vehicles that are used intensively, that stay on the road for long periods of time, and that are significant sources of particle emissions.

Our analysis suggests that one attractive target for retrofit might be concession buses-local plates for the model-year group 1994-97 (US 1991/EURO I). If these buses were retrofitted with catalyzed DPFs, emissions reductions would be on the order of 38 kg per vehicle-year. Such a reduction would be expected to reduce the annual number of deaths attributable to ambient particulate matter by 3.5 per 1000 vehicle-year, leading to health benefits of US$ 6 thousand per vehicle year, with costs of less than 1.5 thousand US$ per vehicle-year, and net benefits of over 4.5 thousand $US per vehicle-year.

The benefits of controlling concession bus emissions are not limited to the 1994-97 model-year group. Positive net benefits are generated by retrofitting concession buses from all model-year groups (from pre-control (1985-93) to Euro III (2011-2014) and would yield more benefits than any other vehicle type in the heavy-duty diesel fleet.

Long-haul trailers-federal are also important targets for retrofit, especially those for the model-year group 2007-10 (US 1998/EURO III). If retrofitted with the most cost-effective and adequate technology –DPF-active regeneration--, emissions would be reduced by 12 kg per vehicle-year. Such a reduction would be expected to decrease the annual number of deaths attributable to ambient particulate matter by 1.1 per 1000 vehicle-year, leading to health benefits of more than US$ 3 thousand per vehicle-year. The costs would be only approximately of 1 thousand US$ per vehicle-year. This would yield net benefits of over 2 thousand US$ per vehicle-year.

The importance of cleaning the heavy-duty fleet in Mexico City has been recognized by experts and authorities in Mexico and Mexico City. Mexico's City and MCMA Air Quality Management Plan, *PROAIRE 2011-2020*, SEDEMA's Institutional Program, and scientists from the Center of Atmospheric Sciences of UNAM, have outlined the importance of cleaning heavy-duty diesel vehicle emissions, either by substitution of engines of by retrofitting control technologies.

Retrofitting the heavy-duty diesel vehicle fleet would represent a small, but important, step towards further improvement of air quality in Mexico City. We encourage authorities in Mexico City to consider moving forward with the design and implementation of such a program.[14]

[14] In the early stages of program design, it will be important – (i) to heed the lessons learned from previous pilot programs, and (ii) to involve air pollution engineers. Engineers with such experience have the knowledge needed to determine which are the best retrofit technologies for each vehicle type and model year – as this depends on the vehicle's age, duty cycle, and PM baseline emission levels, as well as on the maintenance of the vehicle, and on whether the

Table 14. Strategic Priorities for Air Quality Management in the MCMA: PROAIRE 2010-2020 and Mario Molina Center

ProAire, 2011-2020 Strategy 3. Energy Quality and Efficiency in all Sources	Strategy Priorities
Measure 21. Renewal of diesel vehicles with motor substitution and by adapting emissions controls. *Action 21.1. Design a program to replace diesel motors that have been in use for 10 years or more, and adapt emissions control equipments.*	▪ SEDEMA's Institutional Program, 2012-2018 ▪ Mario Molina Center for Strategic Studies on Energy and Environment ▪ Scientific Advisory Board of the Environmental Commission of the Megalópolis ▪ Center of Atmospheric Sciences, UNAM

We close by noting that this one small step must be viewed from the wider perspective suggested by the Mario Molina Center's 2016 position paper on air quality in the Mexico City Valley (CMM, 2016) (Table 14). As the Molina Center report suggests, in addition to reducing emissions from heavy-duty vehicles, many other programs -- such as the development of an integrated public transportation system, the promotion of the rational use of cars, the reduction of emissions from industrial sources and fires, and redesign of the MCMA area to reduce urban sprawl -- must be analyzed and implemented to make significant strides forward in the control of air pollution and its public health impacts.

vehicle has had engine modifications from the original equipment manufacturer (OEM). They also realize the importance of training vehicle operators about the nature, operation and maintenance of retrofit devices.

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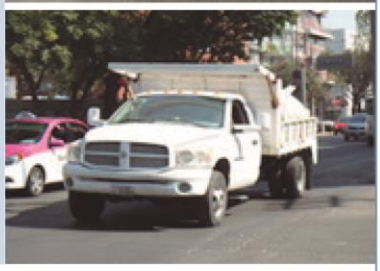
Appendix I. Examples of Heavy-Duty Vehicles from Mexico City (Photographs by Pedro Enrique Armendares)

	Vehicle Category from Emissions Inventory, 2014	Vehicle Description	Examples of HDV Circulating in Mexico City
BUSES	RTP Local Plate	Carry between 20 and 45 passengers	
	School and Personnel Local Plate	Carry between 20 and 45 passengers	 
	Concession Local Plate	Carry between 20 and 45 passengers	 

	Vehicle Category from Emissions Inventory, 2014	Vehicle Description	Examples of HDV Circulating in Mexico City
BUSES	Tourism Federal Plate	Carry more than 45 passengers	
	Passenger Federal Plate	Carry more than 45 passengers	
	Metrobús	Bus rapid transit system with articulated / bi-articulated buses	

	Vehicle Category from Emissions Inventory, 2014	Vehicle Description	Examples from Mexico City
Delivery Trucks > 3.8 tons	Trucks Local Plate 4.6 to 27.2 tons	Light commercial truck, single unit and combination trucks Two axels and 4 to at least 6 tires	
	Trucks Federal Plate 11.8 to 14.9 tons		
Long-Haul Tractor Trailers	Trailers Local and Federal Plate >27.2 tons	Usually with a double rear axle and may have more than one trailer	
			

Appendix II. Properties of Vehicles Per Model-Year Group

Tables II.1. to II.5. present properties for our three main categories and ten types of heavy-duty vehicles:

- Buses: RTP public Transportation – Local Plate, School & Personnel - Local Plate, Concession - Local Plate, Metrobús – Local Plate, Tourism - Federal Plate, Passenger - Federal Plate;
- Delivery Trucks >3.8 tons: Local and Federal Plate; and
- Long-Haul Tractor Trailers: Local and Federal Plate.

Vehicles are grouped in five model-year groups, except for RTP public transportation buses and Metrobús vehicles, which were assigned to two and three groups, respectively. The Metrobús system started operations in 2005, so vehicles were grouped in the corresponding model-year group, based on information provided by SEDEMA (2017a).

Notes for Tables II.1. to II.5.

- Delivery Trucks > 3.8 tons with local plates weigh between 4.6 to 27.2 tons, those with federal plates weigh from 11.8 to 14.9 tons; local and federal plate long-haul tractor trailers weigh >27.2 tons.

Table II.1. Model-Year Group I - 1985-93 Pre-Control

		Bus School and Personnel Local Plate	Bus Concession Local Plate	Bus Tourism Federal Plate	Bus Passenger Federal Plate
Activity Level (km/veh-y)		19,721	63,281	8,749	12,533
Number of Vehicles		55	12	2,250	1,491
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.95	0.57	1.73	0.81
	SO_2	0.0033	0.0019	0.0059	0.0028
	NOx	13.7	8.21	24.9	11.7
Remaining Useful Life (y)		-	-	-	-

		Truck >3.8 tons Local Plate	Truck >3.8 tons Federal Plate	Long-Haul Tractor Trailer Local Plate	Long-Haul Tractor Trailer Federal Plate
Activity Level (km/veh-y)		13,053	14,013	17,647	14,028
Number of Vehicles		1,162	2,013	13	8.864
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.39	0.39	0.25	0.82
	SO_2	0.0013	0.0014	0.0009	0.0027
	NOx	4.18	4.40	2.90	9.32
Remaining Useful Life (y)		-	-	-	-

Table II.2. Model-Year Group II - 1994-97 US 1991/EURO I

		Bus School and Personnel Local Plate	Bus Concession Local Plate	Bus Tourism Federal Plate	Bus Passenger Federal Plate
Activity Level (km/veh-y)		19,617	63,281	8,749	12,533
Number of Vehicles		65	310	861	491
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.73	0.68	1.21	0.57
	SO_2	0.0044	0.0041	0.0073	0.0035
	NOx	13.5	12.5	22.2	10.5
Remaining Useful Life (y)		-	-	-	-

		Truck >3.8 tons Local Plate	Truck >3.8 tons Federal Plate	Long-Haul Tractor Trailer Local Plate	Long-Haul Tractor Trailer Federal Plate
Activity Level (km/veh-y)		13,369	14,004	17,647	14,026
Number of Vehicles		750	893	9	3,929
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.46	0.40	0.27	0.67
	SO_2	0.0022	0.0020	0.0020	0.0051
	NOx	5.21	4.51	4.80	12.6
Remaining Useful Life (y)		-	-	-	-

Table II.3. Model-Year Group III - 1998-06 US 1994/EURO II

		RTP Public Transportation Local Plate	Bus School and Personnel Local Plate	Bus Concession Local Plate	Metrobús Local Plate	Bus Tourism Federal Plate	Bus Passenger Federal Plate
Activity Level (km/veh-y)		39,060	20,364	68,889	77,211	8,749	12,533
Number of Vehicles		949	490	3,770	99	3,465	5,722
Fuel Economy (km/L)		-	-	-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.25	0.65	0.59	0.97	1.13	0.53
	SO_2	0.002	0.0052	0.0047	0.0041	0.0090	0.0043
	NOx	4.8	12.6	11.5	16.9	21.8	10.2
Remaining Useful Life (y)		-	-	-	-	-	-

		Truck >3.8 tons Local Plate	Truck >3.8 tons Federal Plate	Long-Haul Tractor Trailer Local Plate	Long-Haul Tractor Trailer Federal Plate
Activity Level (km/veh-y)		13,089	14,028	19,216	14,024
Number of Vehicles		3,893	3,603	206	15,839
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.47	0.44	0.44	0.83
	SO_2	0.0047	0.0042	0.0049	0.0088
	NOx	10.0	8.8	10.8	19.5
Remaining Useful Life (y)		-	-	-	-

Table II.4. Model-Year Group IV - 2007-10 US 1998/Euro III

		RTP Public Transportation Local Plate	Bus School and Personnel Local Plate	Bus Concession Local Plate	Metrobús Local Plate	Bus Tourism Federal Plate	Bus Passenger Federal Plate
Activity Level (km/veh-y)		28,185	24,561	81,835	71,212	8,749	12,533
Number of Vehicles		250	227	1,669	129	1,343	2,028
Fuel Economy (km/L)		-	-	-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.13	0.40	0.36	0.84	0.83	0.39
	SO_2	0.002	0.0050	0.0041	0.0040	0.0103	0.0049
	NOx	2.1	9.4	8.5	16.18	19.5	9.2
Remaining Useful Life (y)		-	-	-	-	-	-

		Truck >3.8 tons Local Plate	Truck >3.8 tons Federal Plate	Long-Haul Tractor Trailer Local Plate	Long-Haul Tractor Trailer Federal Plate
Activity Level (km/veh-y)		15,596	14,026	22,870	14,024
Number of Vehicles		2,367	1,862	273	8,207
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.43	0.50	0.53	0.98
	SO_2	0.0061	0.0069	0.0072	0.0124
	NOx	10.4	11.5	11.9	20.6
Remaining Useful Life (y)		-	-	-	-

Table II.5. Model-Year Group V - 2011-14 US 2005/EURO IV

		Bus School and Personnel Local Plate	Bus Concession Local Plate	Metrobús Local Plate	Bus Tourism Federal Plate	Bus Passenger Federal Plate
Activity Level (km/veh-y)		25,913	80,815	103,963	8,749	12,533
Number of Vehicles		324	626	148	992	4,155
Fuel Economy (km/L)		-	-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.36	0.34	0.71	0.77	0.36
	SO_2	0.0055	0.0040	0.0054	0.0116	0.0055
	NOx	6.9	6.4	11.4	14.5	6.8
Remaining Useful Life (y)		-	-	-	-	-

		Truck >3.8 tons Local Plate	Truck >3.8 tons Federal Plate	Long-Haul Tractor Trailer Local Plate	Long-Haul Tractor Trailer Federal Plate
Activity Level (km/veh-y)		18,870	14,031	22,890	14,020
Number of Vehicles		2,763	2,065	139	9,088
Fuel Economy (km/L)		-	-	-	-
Baseline Emissions Rate (g/km)	PM	0.29	0.41	0.45	0.80
	SO_2	0.0061	0.0080	0.0084	0.0138
	NOx	6.8	9.0	8.4	13.8
Remaining Useful Life (y)		-	-	-	-

Appendix III. Summary of Results per Model-Year Group

Tables III.1. to III.5. present the results (mean value estimates) from the cost-effectiveness analysis per model-year group. Three categories and 10 types of heavy-duty vehicles are included:

- Buses: School & Personnel - Local Plate, Concession - Local Plate, Metrobús – Local Plate, Tourism - Federal Plate, Passenger - Federal Plate
- Delivery Trucks >3.8 tons: Local and Federal Plate
- Long-Haul Tractor Trailers: Local and Federal Plate

Vehicles are grouped in five model-year groups, except for RTP public transportation buses and Metrobús vehicles, which were assigned to two and three groups, respectively. The Metrobús system started operations in 2005, so vehicles were grouped in the corresponding model-year group, based on information provided by SEDEMA (2017a).

Rows in green highlight the retrofit technology that maximizes the expected net benefits.

Rows in light gray highlight retrofit technologies that are not adequate for such model year or vehicle type. For example, model year vehicles pre-1994 may only be retrofitted with active diesel particulate filters or diesel oxidation catalysts. In turn, federal plate vehicles are only suitable for diesel particulate filters with active regeneration, as this type of filter requires ultralow sulfur diesel (ULSD) and such vehicles may fuel in regions where ULSD is not available.

Notes for Tables III.1. to III.5.

- Delivery Trucks > 3.8 tons with local plates weigh between 4.6 to 27.2 tons, those with federal plates weigh from 11.8 to 14.9 tons; local and federal plate long-haul tractor trailers weigh >27.2 tons.
- Ox Catalyst stands for Diesel Oxidation Catalyst; DPF-Passive stands for Diesel Particulate Filter with catalyzed regeneration (passive), and DPF-Active stands for Diesel Particulate Filter with active regeneration.

Table III.1. 1985-1993 Pre-Control

Bus - School and Personnel Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	4.31	0.38	1.11	0.12	1.00
DPF -- Active	16.39	1.45	4.23	1.45	2.78
DPF -- Passive	16.39	1.45	4.23	1.09	3.13
Ideal Control	18.73	1.66	4.83	0.00	4.83

Bus - Concession Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	8.30	0.73	2.14	0.14	2.00
DPF -- Active	31.56	2.79	8.13	2.33	5.80
DPF -- Passive	31.56	2.79	8.14	1.41	6.72
Ideal Control	36.07	3.19	9.30	0.00	9.30

Bus - Tourism Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	3.48	0.31	0.90	0.06	0.84
DPF -- Active	13.24	1.17	3.41	0.84	2.57
DPF -- Passive	13.24	1.17	3.41	0.67	2.74
Ideal Control	15.14	1.34	3.90	0.00	3.90

Bus - Passenger Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.33	0.21	0.60	0.08	0.52
DPF -- Active	8.88	0.78	2.29	1.05	1.24
DPF -- Passive	8.88	0.79	2.29	0.82	1.47
Ideal Control	10.15	0.90	2.62	0.00	2.62

Truck > 3.8 tons Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.17	0.10	0.30	0.08	0.22
DPF -- Active	4.45	0.39	1.15	0.99	0.16
DPF -- Passive	4.45	0.39	1.15	0.82	0.33
Ideal Control	5.09	0.45	1.32	0.00	1.32

Truck > 3.8 tons Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.26	0.11	0.32	0.09	0.24
DPF -- Active	4.78	0.42	1.23	1.04	0.20
DPF -- Passive	4.78	0.42	1.23	0.85	0.38
Ideal Control	5.47	0.48	1.41	0.00	1.41

Long-Haul Tractor Trailer Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.01	0.09	0.26	0.11	0.16
DPF -- Active	3.86	0.34	1.00	1.25	-0.25
DPF -- Passive	3.86	0.34	1.00	0.99	0.00
Ideal Control	4.41	0.39	1.14	0.00	1.14

Long-Haul Tractor Trailer Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.65	0.23	0.68	0.09	0.60
DPF -- Active	10.07	0.89	2.59	1.06	1.53
DPF -- Passive	10.07	0.89	2.59	0.86	1.73
Ideal Control	11.50	1.02	2.96	0.00	2.96

Table III.2. 1994-1997 US 1991/EURO I

Bus - School and Personnel Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	3.29	0.29	0.85	0.12	0.73
DPF -- Active	12.53	1.11	3.23	1.44	1.79
DPF -- Passive	12.53	1.11	3.23	1.09	2.14
Ideal Control	14.32	1.27	3.69	0.00	3.69

Bus - Concession Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	9.90	0.88	2.55	0.14	2.41
DPF -- Active	37.65	3.33	9.70	2.33	7.37
DPF -- Passive	37.65	3.33	9.71	1.41	8.29
Ideal Control	43.03	3.80	11.09	0.00	11.09

Bus - Tourism Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.43	0.22	0.63	0.06	0.57
DPF -- Active	9.26	0.82	2.39	0.84	1.54
DPF -- Passive	9.26	0.82	2.39	0.67	1.71
Ideal Control	10.59	0.94	2.73	0.00	2.73

Bus - Passenger Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.64	0.15	0.42	0.08	0.34
DPF -- Active	6.25	0.55	1.61	1.05	0.56
DPF -- Passive	6.25	0.55	1.61	0.82	0.79
Ideal Control	7.14	0.63	1.84	0.00	1.84

Truck > 3.8 tons Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.41	0.13	0.36	0.08	0.28
DPF -- Active	5.38	0.48	1.39	1.01	0.38
DPF -- Passive	5.38	0.48	1.39	0.83	0.56
Ideal Control	6.15	0.54	1.59	0.00	1.59

Truck > 3.8 tons Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.29	0.11	0.33	0.09	0.25
DPF -- Active	4.90	0.43	1.27	1.04	0.23
DPF -- Passive	4.90	0.43	1.27	0.85	0.41
Ideal Control	5.60	0.50	1.45	0.00	1.45

Long-Haul Tractor Trailer Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.10	0.10	0.28	0.11	0.18
DPF -- Active	4.17	0.37	1.08	1.25	-0.17
DPF -- Passive	4.17	0.37	1.08	0.99	0.08
Ideal Control	4.76	0.42	1.23	0.00	1.23

Long-Haul Tractor Trailer Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.16	0.19	0.56	0.09	0.47
DPF -- Active	8.22	0.73	2.12	1.06	1.05
DPF -- Passive	8.22	0.73	2.12	0.86	1.26
Ideal Control	9.40	0.83	2.42	0.00	2.42

Table III.3. 1998-2006 US 1994/EURO II

Bus - RTP - Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.25	0.20	0.58	0.14	0.44
DPF -- Active	8.54	0.76	2.20	1.89	0.32
DPF -- Passive	8.54	0.76	2.20	1.32	0.88
Ideal Control	9.77	0.86	2.52	0.00	2.52

Bus - School and Personnel Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	3.04	0.27	0.79	0.12	0.67
DPF -- Active	11.58	1.02	2.99	1.49	1.50
DPF -- Passive	11.58	1.02	2.99	1.12	1.87
Ideal Control	13.24	1.17	3.41	0.00	3.41

Bus - Concession Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	9.35	0.83	2.41	0.14	2.27
DPF -- Active	35.56	3.14	9.17	2.42	6.75
DPF -- Passive	35.56	3.14	9.17	1.43	7.74
Ideal Control	40.64	3.59	10.48	0.00	10.48

Metrobús Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	17.23	1.52	4.44	0.14	4.30
DPF -- Active	65.53	5.79	16.89	2.40	14.49
DPF -- Passive	65.53	5.79	16.89	1.42	15.47
Ideal Control	74.89	6.62	19.30	0.00	19.30

Bus - Tourism Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.27	0.20	0.59	0.06	0.53
DPF -- Active	8.65	0.76	2.23	0.84	1.39
DPF -- Passive	8.65	0.76	2.23	0.67	1.56
Ideal Control	9.89	0.87	2.55	0.00	2.55

Bus - Passenger Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.53	0.14	0.39	0.08	0.31
DPF -- Active	5.81	0.51	1.50	1.05	0.45
DPF -- Passive	5.81	0.51	1.50	0.82	0.68
Ideal Control	6.64	0.59	1.71	0.00	1.71

Truck > 3.8 tons Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.41	0.13	0.37	0.08	0.28
DPF -- Active	5.38	0.48	1.39	0.99	0.40
DPF -- Passive	5.38	0.48	1.39	0.82	0.57
Ideal Control	6.15	0.54	1.59	0.00	1.59

Truck > 3.8 tons Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.42	0.13	0.37	0.09	0.28
DPF -- Active	5.40	0.48	1.40	1.04	0.36
DPF -- Passive	5.40	0.48	1.40	0.85	0.54
Ideal Control	6.17	0.55	1.60	0.00	1.60

Long-Haul Tractor Trailer Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.94	0.17	0.50	0.11	0.39
DPF -- Active	7.40	0.65	1.91	1.33	0.58
DPF -- Passive	7.40	0.65	1.91	1.05	0.86
Ideal Control	8.46	0.75	2.19	0.00	2.19

Long-Haul Tractor Trailer Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.68	0.24	0.69	0.09	0.60
DPF -- Active	10.18	0.90	2.63	1.06	1.56
DPF -- Passive	10.18	0.90	2.63	0.86	1.77
Ideal Control	11.64	1.03	3.00	0.00	3.00

Table III.4. 2007-2010 US 1998/EURO III

Bus - RTP - Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	0.84	0.07	0.22	0.14	0.07
DPF -- Active	3.21	0.28	0.83	1.74	-0.91
DPF -- Passive	3.21	0.28	0.83	1.29	-0.47
Ideal Control	3.66	0.32	0.94	0.00	0.94

Bus - School and Personnel Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.26	0.20	0.58	0.14	0.44
DPF -- Active	8.60	0.76	2.22	1.72	0.50
DPF -- Passive	8.60	0.76	2.22	1.28	0.94
Ideal Control	9.82	0.87	2.53	0.00	2.53

Bus - Concession Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	6.78	0.60	1.75	0.14	1.61
DPF -- Active	25.78	2.28	6.64	2.62	4.03
DPF -- Passive	25.78	2.28	6.64	1.47	5.18
Ideal Control	29.46	2.60	7.59	0.00	7.59

Metrobús Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	13.76	1.22	3.55	0.14	3.41
DPF -- Active	52.34	4.63	13.49	2.32	11.17
DPF -- Passive	52.34	4.63	13.49	1.41	12.08
Ideal Control	59.82	5.29	15.42	0.00	15.42

Bus - Tourism Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.67	0.15	0.43	0.06	0.37
DPF -- Active	6.35	0.56	1.64	0.84	0.79
DPF -- Passive	6.35	0.56	1.64	0.67	0.96
Ideal Control	7.26	0.64	1.87	0.00	1.87

Bus - Passenger Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.12	0.10	0.29	0.08	0.21
DPF -- Active	4.28	0.38	1.10	1.05	0.05
DPF -- Passive	4.28	0.38	1.10	0.82	0.29
Ideal Control	4.89	0.43	1.26	0.00	1.26

Truck > 3.8 tons Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.54	0.14	0.40	0.10	0.30
DPF -- Active	5.87	0.52	1.52	1.11	0.40
DPF -- Passive	5.87	0.52	1.52	0.91	0.61
Ideal Control	6.71	0.59	1.73	0.00	1.73

Truck > 3.8 tons Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.61	0.14	0.42	0.09	0.33
DPF -- Active	6.14	0.54	1.59	1.04	0.55
DPF -- Passive	6.14	0.54	1.59	0.85	0.73
Ideal Control	7.01	0.62	1.81	0.00	1.81

Long-Haul Tractor Trailer Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.79	0.25	0.72	0.13	0.59
DPF -- Active	10.61	0.94	2.74	1.51	1.23
DPF -- Passive	10.61	0.94	2.74	1.19	1.55
Ideal Control	12.12	1.07	3.13	0.00	3.13

Long-Haul Tractor Trailer Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	3.16	0.28	0.82	0.09	0.73
DPF -- Active	12.03	1.06	3.10	1.06	2.03
DPF -- Passive	12.03	1.06	3.10	0.86	2.24
Ideal Control	13.74	1.21	3.54	0.00	3.54

Table III.5. 2011-2014 US 2004/EURO IV

Bus - School and Personnel Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.15	0.19	0.55	0.14	0.41
DPF -- Active	8.16	0.72	2.10	1.76	0.35
DPF -- Passive	8.16	0.72	2.10	1.30	0.81
Ideal Control	9.33	0.82	2.40	0.00	2.40

Bus - Concession Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	6.32	0.56	1.63	0.14	1.49
DPF -- Active	24.04	2.12	6.20	2.60	3.60
DPF -- Passive	24.04	2.12	6.20	1.47	4.73
Ideal Control	27.48	2.43	7.08	0.00	7.08

Metrobús Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	16.98	1.50	4.38	0.14	4.24
DPF -- Active	64.59	5.71	16.65	2.76	13.89
DPF -- Passive	64.59	5.71	16.65	1.50	15.15
Ideal Control	73.81	6.52	19.03	0.00	19.03

Bus - Tourism Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.55	0.14	0.40	0.06	0.34
DPF -- Active	5.89	0.52	1.52	0.84	0.68
DPF -- Passive	5.89	0.52	1.52	0.67	0.84
Ideal Control	6.74	0.60	1.74	0.00	1.74

Bus - Passenger Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.04	0.09	0.27	0.08	0.19
DPF -- Active	3.95	0.35	1.02	1.05	-0.03
DPF -- Passive	3.95	0.35	1.02	0.82	0.20
Ideal Control	4.51	0.40	1.16	0.00	1.16

Truck > 3.8 tons Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.26	0.11	0.32	0.11	0.213
DPF -- Active	4.79	0.42	1.24	1.27	-0.04
DPF -- Passive	4.79	0.42	1.24	1.03	0.205
Ideal Control	5.47	0.48	1.41	0.00	1.41

Truck > 3.8 tons Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	1.32	0.12	0.34	0.09	0.25
DPF -- Active	5.03	0.45	1.30	1.04	0.26
DPF -- Passive	5.03	0.45	1.30	0.85	0.45
Ideal Control	5.75	0.51	1.49	0.00	1.49

Long-Haul Tractor Trailer Local Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.37	0.21	0.61	0.13	0.48
DPF -- Active	9.01	0.80	2.33	1.51	0.81
DPF -- Passive	9.01	0.80	2.33	1.19	1.14
Ideal Control	10.30	0.91	2.66	0.00	2.66

Long-Haul Tractor Trailer Federal Plate	Emissions Reduction (kg/veh-yr)	Deaths Avoided (#/1000 veh-yr)	Benefits (1000 USD/ veh-yr)	Control Cost (1000 USD/ veh-yr)	Net Benefits (1000 USD/ veh-yr)
Status Quo	0.00	0.00	0.00	0.00	0.00
Ox Catalyst	2.58	0.23	0.67	0.09	0.58
DPF -- Active	9.81	0.87	2.53	1.06	1.47
DPF -- Passive	9.81	0.87	2.53	0.86	1.67
Ideal Control	11.22	0.99	2.89	0.00	2.89