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Particulate air pollution and daily mortality: Can results be generalized to Latin American countries?*

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Abstract

Objective. Recently, a series of reports, based on ecological analyses of routinely collected data, have shown positive associations between measures of particle concentration and daily mortality counts in various cities of the US and Europe. **Material and methods.** We reviewed the process of generalization of these results to Latin American countries addressing possible differences in air pollution mixtures, exposure profiles, and population susceptibility. **Results.** A limitation to the process of generalization is the lack of a well-established biological mechanism by which particles may act on daily mortality. Also, sources and levels of ambient air pollution as well as population characteristics and habits vary widely between Northern communities of Europe and the US, and Latin American countries, which impairs the process of generalization. However, results of studies conducted in Latin American countries suggest a similar effect to that observed in Northern countries of Europe and the US. **Conclusions.** Despite uncertainty about the mechanism, there is sufficient evidence that particles are harmful for health. Control measures of particle emission are urgently needed in Latin American countries. Given the potential of misclassification of exposure, the dose-response relationship observed in Northern Europe and the US may not be adequate for Latin American populations. There is a need for a new generation of epidemiological studies including a specific assessment of exposure to fine particles and of events surrounding death.

Key words: air pollution; particles; mortality; Latin America

Romieu I, Borja-Aburto VH.
Contaminación atmosférica por partículas
y mortalidad diaria: ¿se pueden generalizar
los resultados a los países de Latinoamérica?
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Resumen

Objetivo. Recientemente una serie de reportes basados en análisis ecológicos de datos recolectados rutinariamente ha mostrado asociaciones positivas de la concentración diaria de partículas y el número de muertes diarias en diferentes ciudades de los Estados Unidos y Europa. **Material y métodos.** Se revisaron los procesos de generalización de los resultados a América Latina con énfasis en posibles diferencias en las mezclas de contaminantes, perfiles de exposición y susceptibilidad de las poblaciones. **Resultados.** Una limitante del proceso de generalización es la falta de un mecanismo biológico bien establecido por el cual las partículas pueden actuar sobre la mortalidad diaria. Además, las fuentes y niveles de la contaminación ambiental, así como las características de las poblaciones y sus hábitos varían considerablemente entre los países occidentales y Latinoamérica, lo que impide la extrapolación directa de los resultados. Sin embargo, los resultados de los estudios llevados a cabo en América Latina sugieren un efecto similar al observado en los países occidentales. **Conclusiones.** A pesar de las incertidumbres en el mecanismo, existe suficiente evidencia de que las partículas son nocivas para la salud y se requiere urgentemente de medidas de control de emisiones en los países latinoamericanos. Debido al potencial problema de inadecuada medición de la exposición, la relación de dosis-respuesta observada en países del norte puede no ser adecuada para las poblaciones latinoamericanas. Existe la necesidad de una nueva generación de estudios epidemiológicos incluyendo una evaluación de exposición específica a partículas finas en la fracción respirable y de los eventos ocurridos alrededor de la muerte.

Palabras clave: contaminación atmosférica; partículas; mortalidad; América Latina

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Earlier in the twentieth century, a series of episodes of excess mortality occurring concomitantly to extremely high levels of air pollution produced by fossil fuel combustion documented that air pollution can cause death.¹ As the levels of air pollution were reduced in the northern countries (NC), concentration of air pollution sufficiently elevated to cause short-term and readily evident excess mortality were infrequently observed. Air pollution was not widely viewed as an important cause of morbidity and mortality.² However, in the past 20 years, large particles from uncontrolled coal burning have been replaced, in the urban environment, by fine particles (commonly $\leq 0.2 \mu\text{m}$) from motor vehicle emission and secondary aerosols arising from atmospheric reactions between gases.^{1,3} Recently, a series of reports based on ecological analysis of routinely collected data have shown positive associations between measurements of particle concentration—primarily total suspended particles (TSP) and particulate matter less than $10 \mu\text{m}$ —and daily mortality counts from different cities in the US⁴ and Europe.⁵ In general, these studies report that a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} pollution is associated with an increase of 1% in total mortality, ranging from 0.5% to 1.5%.^{3,5} Results also suggest the absence of a threshold and at levels of 24-hour exposure (0 to $200 \mu\text{g}/\text{m}^3$) the exposure-response curve fits a straight line reasonably well. Although most of the new evidence of the relation between particulate matter and mortality is based on ecological data, two recent reports based on longitudinal data^{6,7} have observed an increase in mortality among subjects residing in cities with higher fine particle air pollution levels.

In Latin America, particle levels still exceed the standards in many urban areas.⁸ The population exposed to these levels is large and increasing steadily given the large migration from rural to urban areas and the high fertility rate.⁸ Major concerns have been raised regarding the generalizability of the association observed in NC to Latin American countries (LAC). This is important for risk evaluation and prioritization of pollution control measures, especially given their large economic cost. This paper discusses different issues that need to be considered in the generalization process and the importance of such an attempt at the public health level.

Material and methods

Based on the epidemiological definition of generalization,⁹ relevant issues to consider in the relation between particulate pollution (PM) and daily mortality are: the identification of agent(s) responsible for such an asso-

ciation and its biological mechanism, the conditions of exposure to this agent, and the characterization of susceptible groups. Therefore, in the process of generalization of this relation we need to analyze potential similarities or discordances between NC and LAC of three major factors: 1) air pollution mixtures, 2) exposure profiles, and 3) population characteristics. In this paper, we first present the scientific evidence of the relation of particle air pollution and mortality; then we discuss the role of the three previously mentioned major factors in the generalization process; finally, we present the results of studies conducted in Latin America, and conclude on the implications of generalization of the results for governments of LAC.

Results

Scientific evidence of the relationship between mortality and particles

Most of the scientific evidence of the relationship of PM and mortality is based on the consistency of the results of epidemiological studies across study locations, and coherence with other health endpoints. However, the biological mechanisms by which particulate air pollution causes mortality in relation to acute exposure is still unclear. The effect of inhaled particles seems to be determined by their physical properties, their sites of deposition, and their chemical composition. Exposure to particulate air pollution can induce alveolar inflammation and exacerbate severe preexisting cardiac respiratory diseases, in particular ischemic heart diseases and chronic obstructive pulmonary diseases (COPD), leading eventually to the death of susceptible subjects.¹⁰

Experimental toxicology suggests that the most relevant size fraction for health effects are combustion fine and ultrafine particles (including sulfates (SO_4^{2-}), nitrates (NO_3^-), strong acidity (H^+) and trace elements) because they penetrate deep into the lung and can cause inflammatory reactions.¹¹ The injuries caused to the lung by ultrafine particles with a potential inflammatory reaction, would exacerbate lung disease and increase blood coagulability.¹² Oberdoster *et al*¹³ have shown, in animal models, that ultrafine particles cause greater inflammation than large particle of the same substances. This is related to their high deposition efficiency in the lower respiratory tract, their large number per unit mass, and their increased surface areas available for interaction with cells.

For this reason the most susceptible individuals are likely to be subjects with pre-existing chronic cardiovascular or pulmonary conditions. Recent studies

have used various animal models of human cardiopulmonary diseases to demonstrate that impaired animals show increased sensitivity to inhalation of particles as do individuals with pre-existing diseases in exposed human population. Godleski *et al*¹⁴ exposed normal rats and rats with induced pulmonary inflammation or chronic bronchitis to concentrated ambient air particles (230-270 $\mu\text{g}/\text{m}^3$) for 6 hours on 3 consecutive days. No mortality occurred in the normal animals, but mortality was 19% in the inflammation group, and 37% in the chronic bronchitis group. Other studies¹⁵ have shown that rats with induced pulmonary hypertension (PHT), exposed for 72-hour to residual oil fly ash (ROFA) (an acid-metal rich emission source of particles PM that serves as a $\text{PM}_{2.5}$ surrogate) had enhanced lung damage and significant mortality. Among rats with induced emphysema or lung fibrosis no enhancement was present.

The composition of particles may also be an important element in the toxicity. Particles of both natural and anthropogenic origin can include soluble metal salts and also contain metal complexes at the surface of an insoluble particle. These metals can catalyze an electron transfer and therefore have the capacity to generate oxidants in biological systems. Thus, pulmonary effects of exposure to such particles may resemble those produced by oxidant gas including neutrophilic alveolitis, airway hyperactivity and increased virulence of pulmonary infection leading to enhanced mortality.¹⁶ A study conducted by Prichard *et al*¹⁷ investigated the pulmonary response to ten aerosols relative to their content of the first row transition metals: titanium (Ti), vanadium (V), chromium (Cr), manganese (Mn), iron (Fe), cobalt (Co), nickel (Ni), and copper (Cu). The concentration of soluble metals was the lowest in volcanic ash (natural source), intermediate in the ambient air sample and the highest in the oil fly ash. The ambient air sample and the oil fly ash increased mortality due to subsequent bacterial challenge in mice. Other acute rat toxicity studies¹⁸ demonstrated that the level of total soluble metals correlate with the degree of acute injury. More specifically, soluble nickel and sulfate accounted for protein and lactate dehydrogenase (LDH) leakage in the broncho-alveolar lavage fluid, whereas cellular inflammation correlated best with vanadium containing particles. Rats with systemic hypertension were more severely impacted by this PM, but mortality did not occur.

The biological plausibility of the relationship between PM and mortality is enhanced by the observation of the coherence of cardiopulmonary health effects in epidemiologic studies, and by the fact that non-cardiopulmonary health effects are not typically associ-

ated with particulate pollution.¹⁹ There is a growing body of animal experimental data that provide hypotheses on the pathophysiologic mechanisms that could explain the relationship between inhaled particles and mortality. However, human toxicologic studies are sparse and fail to replicate ambient particle mixtures. There is a need for a better understanding of the mechanisms of injury including the identification of neurotransmitters (such as cytokines), and of immune suppression.²⁰

Potential for generalization

The main factors that need to be considered to determine if a similar relation of mortality and PM, such as that observed in the NC, could be expected in LAC include: 1) the characteristics and chemical composition of particles and air mixture in different locations; 2) the assessment of the population exposure to ambient and indoor air pollutants; 3) the differences in sociodemographic factors and the health status of the exposed population.

Characteristics of particles and air mixtures in different locations

Particulate matter in the air is a mixture of many subclasses of pollutants. The size and chemical composition depends on formation mechanisms, the atmospheric composition, and climatic variables. This variation may be observed within and between large cities, and between urban and rural areas.²¹ Fine ($\leq 2.5 \mu\text{m}$) and ultrafine particles are emitted into the atmosphere by combustion processes including burning of coal and oil, smelting metals, diesel exhaust and exhaust from automobiles equipped with catalytic converters. Major components of the fine fraction ($\leq 2.5 \mu\text{m}$) are: sulfate, nitrate, strong acid, ammonium, organic compounds, trace elements (including metals), elemental carbon, and water. The ratio of total suspended particles (TSP) to particles less than $2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) varies widely depending on the sources and atmospheric conditions such as humidity and sunlight. The coarse fraction (TSP minus PM_{10}), is generally formed by abrasion.²¹

There is no available data on the major sources and composition of fine particles in Latin America. However, data on the sources of TSP and PM_{10} emission from three large cities in Latin America suggest that in Mexico, the larger part of TSP is from natural origin (94.2%), and 4.2% from vehicular exhaust of which 55% is from personal vehicles, and 1.4% from industry.²² In Santiago de Chile, 74.2% of the PM_{10} is

from natural origin and emission of indoor wood burning, 10.6% is produced by mobile sources and 15.2% from industry.²³ In Sao Paulo, Brazil 41% of TSP is related to mobile sources and 59% to industry.²⁴ It is important to mention that 3 types of fuel are currently used in Brazil: 100% ethanol, gasoline with 22% ethanol, and diesel; therefore, the composition of emitted particles could be substantially different from that of oil fly ash. These data contrast with data from the US. Receptor modeling studies in the western United States have found that fugitive dust, motor vehicles, and wood smoke are the major contributors to ambient PM samples there, while results from eastern United States sites indicate that stationary combustion and fugitive dust are major contributors to ambient PM samples in the East. Sulfate and organic carbon are the major secondary components in the Eastern, US while nitrates and organic carbon are the major secondary components in the West. Primary motor vehicle exhaust contributions account for up to 40% of average PM_{10} at many of the sample sites. Emissions of surface dust, organic debris, and sea spray are concentrated mainly in the coarse fraction of PM_{10} ($> 2.5 \mu m$ aero. diam.). A small fraction of this material is in the $PM_{2.5}$ size range ($\leq 2.5 \mu m$). Emission from combustion sources (mobile and stationary sources, biomass burning) are predominantly in the $PM_{2.5}$ size range.²⁵ Given the diversity of sources, one would expect differences in particle composition, and consequently different health effects. Recent data from Mexico City have shown that samples of PM_{10} from the northern part of the city, the focus of industrial activity, and central and southern areas where motor vehicles, pollen and soil are the main pollution sources, have a different composition. Total soluble transition metals were highest in the sample from the northern industrial area (Cu, V, Zn, Pb) which produces a measurable concentration-dependent cytotoxic effect on human fibroblast ($EC50 = 30 \mu g/cm^2$) whereas the other two dusts caused negligible cytotoxicity at similar or higher doses.²⁶

The atmosphere is a complex mixture with other major air pollutants, unmeasured inorganic or organic compounds that could act in synergy with particles (or be highly correlated with particles) and be partly responsible for the health effects observed. For example, in Mexico City, the atmosphere presents substantial levels of particles, ozone and hydrocarbons in particular during the dry season (winter),²² whereas in Santiago particles are high and ozone low during the winter period.²³ In Sao Paulo, due to the use of ethanol, aldehydes and alcohols are also present in the atmosphere.²⁴ Since smog formation increases with

sunlight and temperature, secondary PM peaks during the summer in most US areas.²⁵

Based on the large variability in the atmospheric composition, one would expect that the effect of PM on mortality would vary across cities with different atmospheric and climatic conditions, in particular when the emission source varies. Further analysis of the Philadelphia data,² suggests that the effect of particles varies according to the season due to a change in particle source contribution (in summertime aerosols both sulfate and nitrate components are predominant). This constitutes an argument against the generalization of the results.

Exposure profiles

Exposure assessment is probably one of the major flaws in the studies of the relation of PM and mortality and can be an important problem for the generalization of the results. The ecological analysis of routinely collected data including the use of outdoor monitors, to estimate a population level index of exposure, has raised many concerns because of uncertainty and possible bias. Even in studies where outdoor particle levels near population centers are well represented by monitor, the extent to which fluctuations in outdoor concentrations are found to affect indoor concentrations and personal exposure to particles of outdoor origin remain important. It has been mentioned that in a time series analysis of mortality and particles, if we can assume a day-to-day consistency within individual activity patterns and indoor sources, the ranking of individual daily exposure could be adequate. It would result in similar regression slopes, with different intercepts. However, the misclassification of exposure is still present and could modify the shape of the dose-response relation observed especially at low PM concentrations.¹¹

The difficulty to accurately determine individual exposure impairs the generalization process in particular because: 1) the number of monitoring stations and their distribution vary within and between cities and therefore the validity of the average level as representative of the population exposure will also vary widely; 2) a good correlation between measurements at different monitoring stations does not insure similar levels; 3) personal exposure depends on geographic, climatic and atmospheric factors, time activity patterns, housing characteristics, and indoor sources; all factors that also vary from place to place. For example, several surveys have shown that the population in Mexico City spends in average 20 hours

indoors, 2.76 hours outdoors, and 1.22 hours in transport each day.^{27,28} This is in contrast to 1 hour or less spent outdoors among residents of US cities.²⁹ The reported indoor/outdoor ratio of PM₁₀ in downtown Mexico City is 1.05.²⁷ In the United States, at low outdoor levels of fine particulates mean indoor concentrations have been found to be twice as high as outdoor levels. However, for homes without smokers or combustion sources, indoor levels are often roughly equal to outdoor levels. At high outdoor levels, mean indoor concentrations have been about 10% lower than the mean outdoor concentration.²⁵ In Santiago, Chile, because of the low temperature observed in the winter period, indoor sources of heating (wood burning, kerosene) will largely contribute to personal exposure. Therefore, under similar outdoor levels, an individual residing in Philadelphia, Mexico or Santiago would be exposed to different doses of particles and it would be difficult to use a similar dose-response curve to determine the health effect. Finally, an additional difficulty is related to the estimate of exposure to concurrent pollutants, which in turn can act as confounders or effect modifiers.

Population characteristics

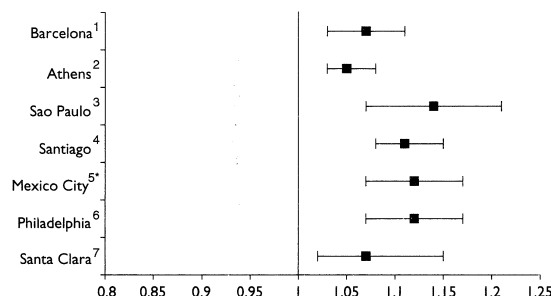
Although most people would agree that the population of different US cities can be compared, there are several differences between these populations and those of LAC including the age structure, the underlying disease pattern, the prevalence of disease cofactors (smoking, nutrition), the access and quality of medical care, and life style in general. Latin American populations tend to be younger with lower crude death rates.⁸ In Brazil, Chile and Mexico, the population of children less than 5 years old is close to 12% whereas in the US this population corresponds to 7%. The population of adults older than 55 years is close to 9% in Brazil and Mexico, 12% in Chile and reaches 21% in the US.^{30,31} Disease pattern is also different. For example, in Mexico non-respiratory related infectious and parasitic diseases in children less than 5 years still represent close to 25% of hospitalizations, and low birth weight is more prevalent.³² The major causes of death among children 1 to 4 years of age include accidents (0.5%), pneumonia and influenza (0.3%), gastrointestinal infections (0.3%) and congenital abnormalities (0.2%).³³ In adults over 65 years, 43% of hospitalizations are related to cardiovascular diseases and 16% to respiratory diseases. The major causes of mortality are cardiovascular diseases (11%), malignant tumors (6%), diabetes (5%), cerebrovascular disease (4%), pneumonia (2%), malnutrition (9.5%) and chronic

respiratory diseases (1.5%).³³ Smoking habits show a direct correlation with education, smoking being more prevalent in higher social classes;³⁴ in contrast, in the US population smoking is more prevalent in lower social classes.³⁵ In the US, accidents and congenital anomalies are the main causes of death in children under five. In adults over 65 the leading causes of death are heart disease (36%), malignant neoplasms (22%), cerebrovascular diseases (8%), chronic obstructive pulmonary diseases (5%), pneumonia and influenza (4%), diabetes mellitus (3%) and accidents (2%).³⁶ Therefore, the pool of susceptible individuals as well as competing risk of death differ between LAC and NC. When considering the relation of PM and mortality we can expect a smaller risk among LAC populations given the smaller pool of susceptible individuals and the fact that the most susceptible individuals may have died from other causes. The generalization process would need to consider subgroups of population such as individuals 65 years of age or over with chronic pulmonary or cardiovascular diseases, given that there is no evidence of differential susceptibility in relation to their country of origin. One interesting observation in the Philadelphia data³⁷ is that the strength of the association between PM and mortality increases when specific age stratified mortality is considered. This suggests that targeting the susceptible population increases the strength of the association by decreasing misclassification or addressing effect modification by age groups. A similar observation has been reported in other studies.³ We can then conclude that at the global level, we might expect different findings in LAC than those observed in NC, but assuming similar physical and chemical properties of PM in different countries, similar estimates could be found if stratification by age group and relevant pathology was considered.

Latin American studies

To date, three studies have examined the relation of air pollution and daily mortality in large Latin American cities (Mexico City, Santiago, and Sao Paulo).³⁸⁻⁴¹ In these studies, PM levels were considerably higher than those observed in previous studies conducted in the US; however, the associations observed were similar to those reported previously (Figure 1).^{5,37,42-44}

In the study conducted in Mexico, Borja *et al*³⁸ studied the relation between exposure to air pollutants, in particular ozone and TSP, and daily mortality from 1990 to 1992. Air pollutant levels were averaged over Mexico City using 9 monitoring stations providing information on daily ambient levels of sulfur dioxide (SO₂), carbon monoxide (CO), and ozone (O₃). During the



Source: 1. Sunyer et al.,⁴² 2. Touloumi et al.,³ 3. Saldiva et al.,⁴¹ 4. Ostro et al.,⁴⁰ 5. Borja et al.,³⁸ 6. Schwartz et al.,³⁷ 7. Fairley et al.⁴³

*Converted from TSP to PM₁₀ assuming a ratio of 0.5

FIGURE 1. RELATIVE RISK OF DAILY MORTALITY IN DIFFERENT CITIES RELATED TO A 100 µg/m³ INCREASE IN PM₁₀

study period, ozone 1-hour maximum ranged from 25 to 285 ppb with a median of 155 ppb (n= 1072). TSP were measured every 6 days (n= 216) and the median 24-hour TSP averaged 204 µg/m³ with a range of 66 to 456 µg/m³. Total mortality, cardiovascular mortality, and mortality for those over 65 years were associated with ozone concentration after adjusting for minimum temperature (2.4%, 2.3% and 3.9% increase for each 100 ppb change in daily maximum ozone level, respectively). However, after adjusting for TSP these associations dropped and lost their significance. In a model adjusting for temperature and long time trend, ozone, and SO₂, the authors observed an increase of 6% (95% CI= 3%-8%) in daily mortality associated with an increase of 100 µg/m³ of TSP; similar estimates were observed among subjects aged 65 years or more and the population as a whole. The air pollution levels in Mexico City is being reported from five different areas (north east, north west, south east, south west, and center) given the large difference in the daily air pollution levels observed in this megacity.²² The effect of TSP was consistent across several areas of the city.³⁹

The study from Santiago reviewed data from 1989 to 1991, extracting daily deaths of residents of metropolitan Santiago.⁴⁰ Total mortality and mortality from respiratory and cardiovascular diseases were studied separately as well as all-cause mortality for males and females and subjects over 65 years. Exposure to PM₁₀ and other pollutants were determined through the monitoring network of Santiago using 4 stations located in the center of the city. The authors correlated historical data of the downtown monitoring stations and five monitors around the city (correlation ranging from 0.69 to 0.92) and concluded that the downtown

monitors were representative of the entire metropolitan area of Santiago. During the study period, PM₁₀ was measured on a daily basis (n= 790); the 24-hour average PM₁₀ was 115.4 µg/m³ with a range of 30 to 367 µg/m³. The average highest daily reading was 141.5 µg/m³ ranging from 35 to 500 µg/m³. After adjusting for minimum temperature, hottest and coldest 10% of the days, quarter and year, a 10 µg/m³ increase in PM₁₀ was related to a 0.6% increase in total mortality (0.4% to 0.7%). For respiratory death, the estimate reached 1% (95% CI= 0.6% to 1.5%). Among older subjects the risk was lower (0.3%, 95% CI= 0.2% to 0.7%) than among the total population. These authors report a stronger effect during the winter period (RR= 1.07, p= 0.0002) than during the summer (RR= 1.06, p= 0.10) and a 27% increase in mortality associated with a change of 10 °C in minimum temperature. This suggests that low temperature and indoor exposure to biomass or fossil fuel during the winter period may play an important role in the total mortality observed in this study. Data from the monitoring stations of Santiago show that the ratio of PM_{2.5} to PM₁₀ is higher during the winter months (0.60 vs 0.40)⁴⁴ which could also explain part of the increased risk observed during this period.

In the study from Sao Paulo, Saldiva et al. studied the relation between air pollution and mortality among subjects 65 years and over between May 1990 and April 1991.⁴¹ Air pollution levels were averaged over 10 monitoring stations to determine 24-hour averages of PM₁₀, SO₂ and CO, over 4 stations for O₃ and 3 stations for nitrogen dioxide (NO₂). After adjusting for month, weather variable, day of the week and other air pollutants, an increase of 100 mg/m³ in 24-hour average PM₁₀ ambient levels was related to a 13% (95% CI= 7% to 18%) increase in total mortality with a significant linear trend. Cold weather was also a strong predictor of total mortality. In this study specific mortality for respiratory or cardio-vascular diseases was not examined.

Results from these preliminary reports suggest that a similar relation between PM and daily mortality as that observed in the NC is observed in LAC, although some inconsistencies exist within the studies, such as the lack of higher mortality risk in older subjects. In addition, morbidity studies conducted in LAC have also reported an adverse effect of PM exposure such as increases in respiratory-related emergency visits related to PM₁₀ and PM_{2.5} levels,⁴⁵ decrease in lung function, and increase in respiratory symptoms. Stronger adverse effects have been observed with fine particles (PM_{2.5}).⁴⁶ This consistency across different health endpoints suggests that the health effects of

PM are real and that generalization of results could be justified.

Conclusion

Acute air pollution episodes occurring earlier in this century have shown that particles at high concentration could cause mortality. Later studies have reported associations between daily mortality and particles at much lower levels. Although the biological mechanism of action of particulates on mortality is still uncertain, the lack of a known mechanism does not necessarily mean that the relation observed is not causal. It is important to remember that biological plausibility at any given time depends on the current state of knowledge.⁴⁷ It seems quite clear that particles have an adverse health effect and there is no reason to believe that physiopathological mechanisms would be different among Latin American subjects. Studies conducted in NC suggest a linear relation between mortality and particulate exposure. However, there is still uncertainty on the shape of the dose response relationship at low concentration (under $50 \mu\text{g}/\text{m}^3$ of PM_{10}) given that error measurements at low levels of exposure may have a large impact on this relation.

To generalize these results to LAC, the major issue remains the extrapolation of the dose-response relationship of PM and mortality. Is it possible to assume a similar slope in different locations where sources of air pollutant emission as well as climatic and atmospheric conditions vary, and consequently air pollution mixtures are different. Similarly, should we expect a similar effect among populations with such differences in exposure pattern, susceptible groups, disease cofactors and competing risks of death?

Based on the major points previously presented, we conclude that we cannot generalize the results and that the dose-response relationship is likely to be different. However, epidemiological studies conducted in Latin America provide estimates similar to those observed in studies conducted in the western world, thus supporting generalization. One possible explanation of these similarities could be linked to the fact that time series studies are using very crude estimates of exposure leading to misclassification and consequently to an underestimation of the effects, in general.

Because of this large misclassification of exposure, the influence of other factors such as physical and chemical composition of particles, co-pollutants in the atmosphere, temperature and relative humidity, and population characteristics may not be readily observed. The fact that similar results have been observed in studies using different particle measurements (TSP,

PM_{10} and "Coefficient of Haze")⁴ converted to similar units using constant converting factors, tends to support this hypothesis. It is very unlikely that a similar relation of TSP to PM_{10} or $\text{PM}_{2.5}$ be observed in such different locations. For example in the northern part of Mexico City, a large proportion of PM_{10} corresponds to coarse particles and the ratio of $\text{PM}_{2.5}$ to PM_{10} vary between the northern and southern part of the city (0.5 and 0.70 respectively Romieu, unpublished. In Santiago, Chile, the ratio of $\text{PM}_{2.5}$ to PM_{10} tends to vary between seasons.⁴⁵ Therefore, different measures of particles should lead to different estimates.

To compare the dose-response relationship of particles and mortality between NC and LAC, we need a new generation of epidemiological studies. These studies should focus on the most susceptible individuals and include a better understanding of the events surrounding death to assign an appropriate exposure level to each individual. Exposure assessment should focus on fine particles as well as other pollutants, and climatic variables, to control for model interaction. In addition, we need a better understanding of the biological mechanism in order to focus exposure assessment on relevant factors, in particular PM composition and acidity should be determined in different areas and results compared.

Uncertainty about the true dose-response relationship of PM and mortality should not delay the implementation of control measures, in particular because the true association is likely to be stronger than that observed in epidemiological studies. Even a small effect such as a 1% increase in total mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase in daily PM_{10} (24-hour average) would have a large impact at the population level, given the increasing prevalence of cardiovascular and respiratory diseases in LAC.³² In Latin America more than 80 million people are exposed to TSP levels exceeding yearly averages of $75 \mu\text{g}/\text{m}^3$ ⁸ but environmental air pollution is not considered a priority in most of those countries. It is important to stress the adverse effect of particle pollution even at levels previously considered safe (the current norm for PM_{10} in many LAC is $150 \mu\text{g}/\text{m}^3$ (24-hour average)) to promote long term control policies.

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