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Health impacts of air pollution

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The Art & Science of Risk

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Abstract

Air, water and food, in this order, are the core metabolic requirements for human life. A reduction in either the quality or quantity of any of these essential resources constitutes a major threat to human health.

Due to the globalization of industrial production and energy needs, and to an increasing number of acute air pollution episodes in many cities worldwide, air pollution has become a growing concern over the past decade, with mounting evidence of its dramatic health impacts.

Air pollution is today considered as the world's most serious environmental health risk by the World Health Organisation (WHO). Numerous studies have consistently shown its deleterious effect on human health. It is estimated that polluted air was responsible for over 6 million deaths worldwide in 2016[1], twice as many as AIDS, tuberculosis and malaria combined. Ambient particulate matter, nitrogen dioxides and other pollutants have been associated with increased prevalence of a number of respiratory and cardiovascular diseases[2], cancers and even appear to be correlated with neurodevelopmental disorders in children and neurodegenerative diseases in adult. Data on air quality indicators is becoming increasingly available and the science underlying the related health impact is also evolving rapidly[3].

As a global reinsurer with significant exposure to mortality risks, SCOR is monitoring the consequences of emerging risks that might have an impact on human life, such as air pollution, endocrine disruptors and anti-microbial resistance, to name a few. The (re)insurance industry has recognized that air pollution should be considered as an important emerging risk[4].

This paper presents the issue of air pollution and discusses the latest findings of fundamental research with regard to its consequences on human health. It is organized as follows: in the first section, the nature and dynamics of air pollution are described. In the second section, the consequences of air pollution on health are documented. In the last section, the consequences of air pollution on the (re)insurance industry are discussed.

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[1] *Global Burden of Disease Study, 2016 [1]*
[2] *Beelen et al., 2014 [2], Carey et al., 2013 [3], Cesaroni et al., 2013 [4], Turner et al., 2017 [5], Garcia et al., 2016 [6], Gakidou Emmanuela et al., 2017 [7]*
[3] *Beelen et al., 2008 [8]*
[4] *Risk Radar, CRO Forum* https://www.thecroforum.org/wpcontent/uploads/2018/05/CRO-ERI_Emerging-Risk-RadarTrends_Apr2018_FINAL.pdf

I. Air pollution: components and dynamics

1. The different categories of air pollutants

The Earth's atmosphere is composed mainly of dinitrogen (N_2 : 78% by volume) and dioxygen (O_2 : 21% by volume). It can be polluted by gaseous, liquid and solid pollutants either from natural sources or discharged in the atmosphere by human activities. Natural sources include emissions from plants, from the biomass of the ocean, volcanic gas and the re-suspension of dust in arid areas such as deserts. Anthropogenic sources include combustion engines (both diesel and petrol), household and industry solid-fuel combustion for energy production (coal, lignite, heavy oil and biomass), other industrial activities (building, mining, manufacture of cement, smelting), agriculture, with the use of entrants, and the erosion of roads by vehicles and abrasion of brakes and tyres.

Man-made and natural discharge in the atmosphere can lead to both primary and secondary pollutants.

Primary pollutants are directly released in the air, and include the following components:

- Particulate matter (PM_{10} and $PM_{2.5}$);
- Carbon oxides (e.g. carbon monoxide);
- Oxides of sulphur;
- Ammonia;
- Light hydrocarbons;
- Volatile organic compounds;
- Metals (lead, mercury, cadmium).

By contrast, secondary pollutants are formed in the atmosphere as a result of a chemical reaction between gaseous precursors such as sulphur dioxide, oxides of nitrogen, ammonia and non-methane volatile organic compounds. They include the following elements:

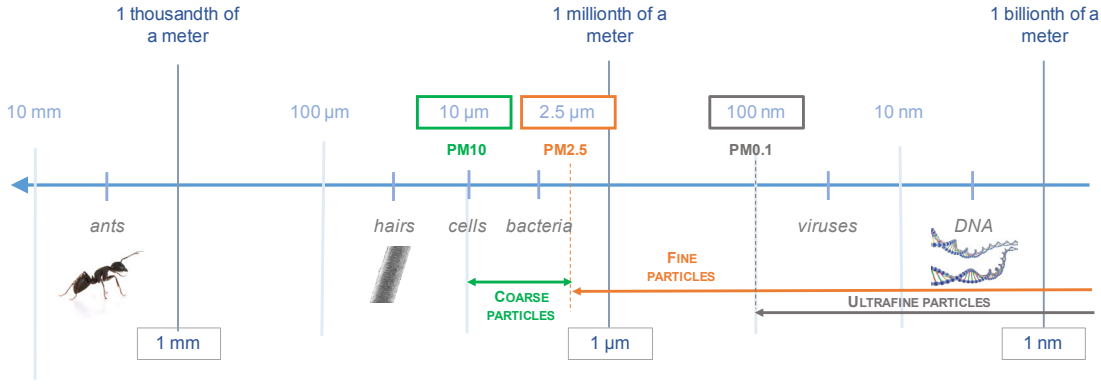
- Oxides of nitrogen[5],
- Ozone[6].

a. Particulate matter (PM)

PM, or coarse particles[7], consist of invisible solid and liquid particles with diameters of either less than 10µm[8] (PM_{10}), or 2,5µm ($PM_{2.5}$). They affect more people than any other pollutant[9], and can penetrate into the respiratory tract. $PM_{2.5}$, being even smaller, can reach the deepest areas of the breathing apparatus, such as the pulmonary alveoli[10].

They include ultrafine particles having a diameter of less than 0.1µm. The chemical compounds of PM include sulfates, nitrates, ammonium and other inorganic ions such as sodium, potassium, calcium or magnesium, metals such as cadmium, copper, nickel and zinc and biological components such as allergens or microbes[11].

Figure 1: Schematic overview of the relative size of PM_{10}, $PM_{2.5}$ and ultrafine particles.



PM can be generated by industry, transport and agriculture, and due to their light weight, can also be carried on air currents from one country to another. As an example, two-thirds of the PM_{10} recorded in the Netherlands is estimated to have originated in foreign countries[12].

[5] *Amount of the loss divided by the value of the insured property*
[6] *See paragraph c. Ozone.*
[7] *WHO, 2013 [10]*
[8] *One millionth of a metre.*
[9] *WHO, 2018 [9]*
[10] *Deng, X., Rui, W., Zhang, F. et al., 2013 [11]*
[11] *WHO, 2013 [10]*
[12] *Hendriks et al., [12]*

The WHO 2005 Air Quality Guidelines (see Appendice, page 30) recommend maintaining PM concentrations below the following levels:

› $PM_{2.5}$: 10 µg.m^{-3} annual mean; 25 µg.m^{-3} 24-hour mean;
› PM_{10}: 20 µg.m^{-3} annual mean; 50 µg.m^{-3} 24-hour mean.

According to the WHO, in 2016, 91% of the world population was living in places where the air quality guidelines levels were not met.

b. Nitric oxides (NOx)

Nitric oxides include nitric oxide (NO) and nitrogen dioxide (NO_2), the result of the oxidation of nitric oxide by ozone. Nitrogen dioxide is a by-product of combustion reactions, and typically appears during the burning of fossil fuels in power plants. In cities, where it contributes to the formation of smog events[13], most of the nitrogen dioxide comes from motor vehicle exhaust.

Nitric oxide is an important molecule in human cells, but has a limited toxicity in the concentrations at which it is found in the atmosphere. However, exposure to nitrogen dioxide can decrease lung function and increase the risk of respiratory symptoms[14].

c. Ammonia (NH_3)

Ammonia is the most abundant alkaline gas in the atmosphere and the most commonly produced chemical. It is a precursor for the nitrogen reaction chain and is produced naturally from decomposition of organic matter, including plants, animals and wastes. The largest source of NH_3 emissions is agriculture, with both animal husbandry and the use of fertilizers.
Being lighter than air, this gas usually rises and does not typically lead to immediate impact on health. At high concentrations however, or in moist areas, ammonia leads to throat and respiratory tract irritation.

d. Ozone

Ozone is known as the high altitude shield of the Earth, where it protects the atmosphere against the harmful ultraviolet radiation emitted by the sun. However, at lower altitudes ozone is a secondary pollutant resulting from a reaction between nitric oxides and organic volatile compounds (as hydrocarbons present in petrol). This photochemical process can only occur under the radiation of the sun, which explains the summer-seasonality of ozone pollution events.

Ozone peaks have documented consequences on lungs[15] and the respiratory tract[16].

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[13] *Wang et al., 2016 [13]*
[14] *WHO, 2003 [14]*
[15] *WHO, 2003 [14]*
[16] *Anderson, H.R. et al., 2003 [15]*

e. Sulphur dioxide

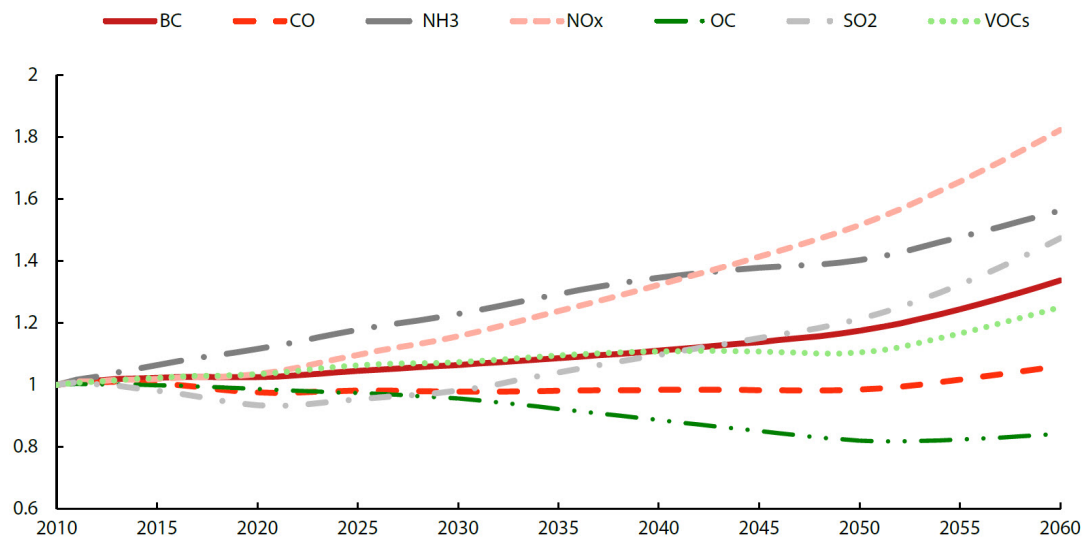
Sulphur dioxide (SO2) is a corrosive gas produced by the consumption of fuel containing sulphur, such as coal and oil. It can also be discharged in the atmosphere through natural processes, such as organic decomposition or volcanic eruptions. Sulphur dioxide irritates the skin and mucous membranes (eyes, nose, throat and lungs), and can affect the respiratory system.

Sulphur dioxide reacts with water in the air to form sulphuric acid, an important component of the phenomenon of acid rain.

2. Dynamics and trends of air pollution

Air pollution is considered an emerging risk, and its dynamics depend on a broad range of factors. A WHO global comparative analysis of air pollution[17] concluded that ambient air pollution increased by 8% between 2008 and 2013. In the absence of more stringent policies, or disruptive technological changes, increasing economic activity and energy demand will lead to a significant increase in global emissions of air pollutants in the coming decades. This is because air pollution mirrors the underlying baseline assumptions of economic growth, but at a slower pace. The OECD has developed projections of emissions of pollutants over time based on environmental-economic models (ENV Linkage models) where some pollutants, in particular nitrous oxides, are expected to almost double by 2060. The figure below summarises the projected trends in emissions of the most common pollutants, in particular black carbon (a component of fine particulate matter – $PM_{2.5}$), carbon monoxide, nitric oxides (NOx) and sulphur dioxide (SO2).

Figure 2: Emission projections over time indexed with respect to 2010[18]



[17] *WHO, 2016 [18]*
[18] *OECD, 2016 [16]*

Pollution in ancient Rome.

Economic activities have always been associated with the discharge of pollutants into the atmosphere. Residents of ancient Rome were already concerned about air pollution, calling the city's smoke cloud grave caelum ("heavy heaven") and infamis aer ("infamous air")[19]. Philosopher and statesman Seneca even wrote, in A.D. 61 "No sooner had I left behind the oppressive atmosphere of the city and that reek of smoking cookers which pour out, along with clouds of ashes, all the poisonous fumes they've accumulated in their interiors whenever they're started up, than I noticed the change in my condition".
The analysis of ancient Greenland ice provides evidence for the presence of lead in the air at the time of the Roman Empire, due to a process of silver extraction, making it the oldest report of international atmospheric pollution.

Finally, there are geographical discrepancies in both levels and trends of air pollution, due to the uneven level of development and population concentration around the globe. The concentration of pollutants, and in particular of PM, are already above the levels recommended by the WHO Air quality guidelines in a number of regions, such as in South and East Asia. Several studies have demonstrated that air pollution negatively correlates with the level of income of countries, as Europe, the Americas and the Western Pacific Region face low levels of pollution, while urban air pollution averages in the Eastern Mediterranean and South East Asia can exceed WHO limits by up to 5-10 times[20].

[19] *Frontinus, De Aquis Urbis Romae, 1st Century AC. [17]*
[20] *Beelen et al., 2008 [8]*

Figure 3: Trend in PM2.5 or PM10 based on cities available in several versions of the database, by region1 (Source: WHO, WHO's urban ambient air pollution database-update 2016)[21]

Region	Trend over the mean period 2008-2013[2]
Africa (Sub-Saharan)	NA
America, LMI	→
America, HI	↘
Eastern Mediterranean, LMI	↗
Eastern Mediterranean, HI	↗
Europe, LMI	↘
Europe, HI	↘
South-East Asia	↗
Western Pacific, LMI	↗
Western Pacific, HI	↘
World[3]	↗

[1] Criteria for inclusion: cities with measured $PM_{2.5}$ or PM_{10} values in the three database versions covering a period of 3 years or more, or in two versions and covering a period of 4 years or more.

[2] → : No more than 5% change over the five-year period;
↘ : More than 5% decrease over the five year period;
↗ : More than 5% increase over the five-year period.

[3] The mean for the World is based on weighting by regional urban population.

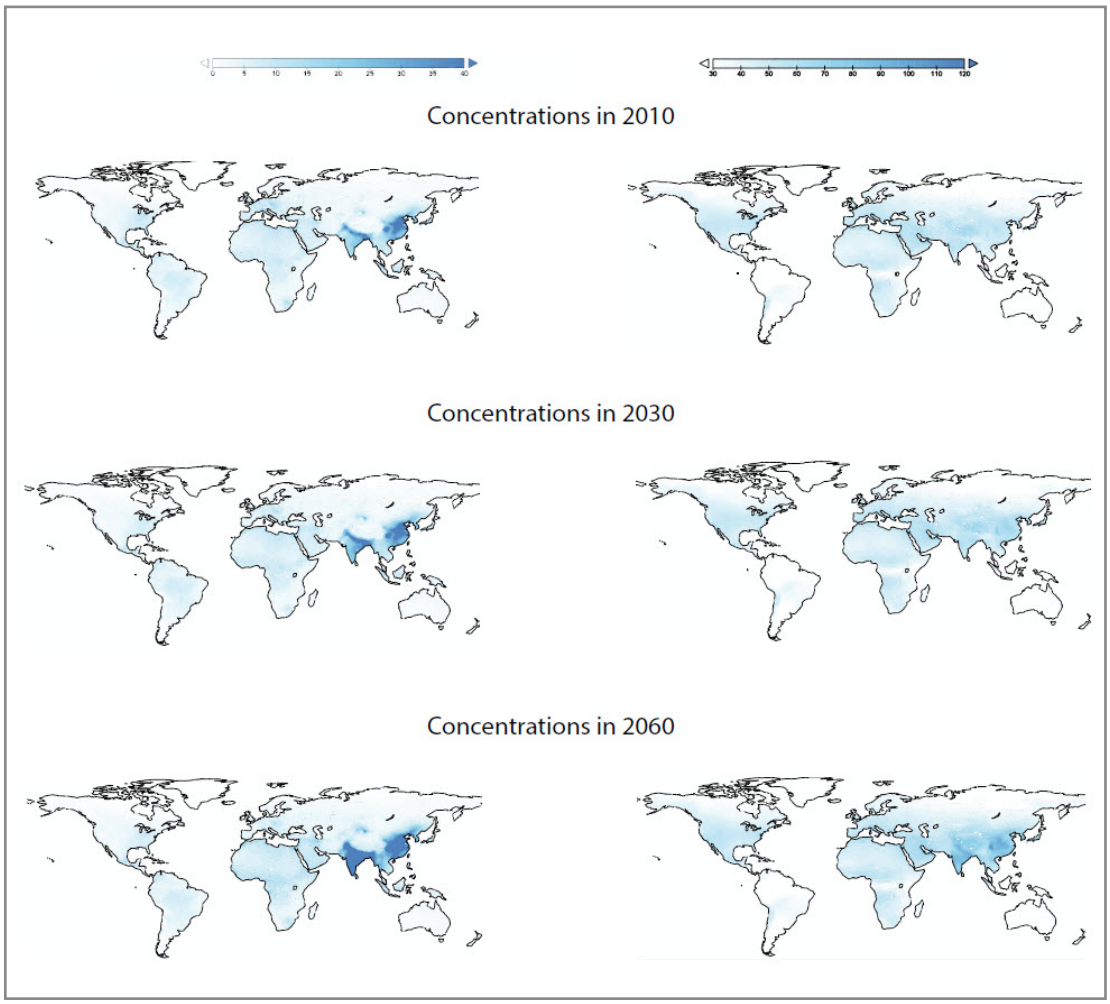
LMI: Low- and middle-income countries; HI: High-income; NA: Not available.

Results are based on 795 cities and are to be interpreted with caution, as 1) cities included might not ensure representativeness, 2) yearly variations due for example to climatic changes can be important and 3) a 5-year comparison does not necessarily represent trends, in particular when changes are limited.

[21] *WHO, 2016 [19]*

The figure below illustrates the expected disparities in projected pollution levels. While concentrations are expected to grow overall, this growth should be more rapid in South and East Asia. It is expected that in some high concentration areas, such as large cities, pollution levels will permanently remain above recommended levels.

Figure 4: Projection of emissions of particulate matter and ozone.
Projected annual average $PM_{2.5}$ (left) and maximal 6 month mean of daily maximal hourly ozone (right)[22]



[22] *OECD, 2016 [16]*

II. Health effects of pollution

Numerous studies have found an association between air pollution and several adverse health effects in the general population. These effects range from subclinical effects to premature death, and include notably the following consequences:

- Increased respiratory ailments (bronchiolitis, rhinopharyngitis, bronchial hypersecretions);
- Degradated ventilator function (lower breathing capacity, asthma, coughing);
- Eye irritation;
- Increased cardiovascular morbidity;
- Depleted immune system;
- Impact on short-term mortality due to respiratory and cardiovascular diseases;
- Impact on long-term mortality linked to the carcinogenic effect of pollutants.

Air pollution is a major cause of non-communicable diseases. It is estimated that at least 3% of cardiopulmonary and 5% of lung cancer deaths are attributable to PM globally. The most recent study on the Global Burden of Disease estimates that 7.5% of deaths globally were attributable to ambient air pollution in 2016. In the same year, 27.5% of deaths due to Lower Respiratory Tract Infections and 26.8% of deaths due to Chronic Obstructive Pulmonary Diseases were linked to air pollution. This chapter will review the impact of air pollution on the major sources of deaths globally[23].

1. Short-term or long-term exposure

The impact of environmental risk factors changes depending on the type of exposure. Short-term peak and long-term chronic exposure do not follow the same dynamics and do not lead to the same consequences. This section describes the differences between the health effects of short-and long-term exposure to air pollution. Overall, risk estimates of long-term exposure studies are higher than those of short-term exposure studies.

[23] *See Appendix 1 for a chart of the top 10 causes of deaths globally in 2015.*

a. Short-term exposure

Scientific studies have consistently observed associations between adverse mortality and short-term elevations in ambient air pollution.

The level of pollution varies on a daily basis following patterns which depend on human activities, meteorological conditions and the seasons. Several recent epidemiological studies have compared daily mortality to pollution exposure, in particular to PM. As an example, particle concentrations and mortality in six eastern US cities have been measured for eight years[24]. The strongest association was found with $PM_{2.5}$: a 10 $\mu g.m^{-3}$ increase in the two-day mean of $PM_{2.5}$ concentration was associated with an average 1.5%[25] increase in total daily mortality. Larger increases were found for deaths caused by chronic obstructive pulmonary disease (+3.3%) and ischaemic heart disease (+2.1%). Over the last decade, daily measures of ambient PM_{10} have been collected in Bangkok. The analysis indicates a statistically significant association between PM_{10} concentration and all of the alternative measures of mortality. The results suggest a 10 $\mu g.m^{-3}$ increase in daily PM_{10} is associated with a 1-2% increase in natural mortality, a 1-2% increase in mortality associated with cardiovascular affections, and a 3-6% increase in respiratory mortality[26].

The association between daily pollution and daily mortality has been confirmed by hospital admission data. A US study has demonstrated an association between short-term increases in hospital admission rates and $PM_{2.5}$ for all health outcomes except injuries[27]. The strongest association was for heart failure, which had a 1.28%[28] increase in risk per 10 $\mu g.m^{-3}$ increase in same-day $PM_{2.5}$.

This daily sensitivity of mortality has been confirmed for almost all pollutants. Mustafic et al.[29] assessed and quantified the association between short-term exposure to major air pollutants (ozone, carbon monoxide, nitrogen dioxide, sulphur dioxide, and PM) on myocardial infarction risk[30].

In addition to daily mortality analysis, it has been demonstrated that, as in the case for the influenza virus, mortality associated with air pollution varies seasonally. This is because seasonal variation in meteorological conditions, sources of pollution and human behavioural patterns lead to seasonal differences in personal exposure[31]. In Europe and in the US, the effects of air pollution appear to be more apparent during the summer[32]. This sensitivity can be analysed at country level: while in China, mortality associated with air pollution is higher in summer and winter, autumn is the worst season in South Korea, and spring and autumn have the highest death rates associated with air pollution in Japan[33].

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[24] *Joel Schwartz et al., 1996 [20]*
[25] *1.1% to 1.9% with a 95% confidence interval (CI).*
[26] *Bart Ostro et al.,1999 [21]*
[27] *Francesca Dominici et al., 2006 [22]*
[28] *0.78%-1.78% with a 95% CI*
[29] *Hazrije Mustafic et al., 2012 [23]*
[30] *Resp., carbon monoxide: 1.048; 95% CI, 1.026-1.070; nitrogen dioxide: 1.011; 95% CI, 1.006-1.016; sulphur dioxide: 1.010; 95% CI, 1.003-1.017; PM10:1.006; 95% CI, 1.002-1.009; and PM2.5: 1.025; 95% CI, 1.015-1.036*
[31] *Wan-Hsiang Hsu et al., 2017 [24]*
[32] *Stafoggia et al., 2008 [25], Moolgavkar S. H.,et al., 1996 [26]*
[33] *Kim et al, 2017 [27]*

Under specific circumstances, the concentration of pollutants can increase more dramatically and lead to possibly even more severe consequences on human health. Historically, the London Smog of 1952 was the first catastrophic pollution event in Western Europe, causing thousands of deaths and disrupting the city's transport system for several days. More recently, in November 2017, concentrations of $PM_{2.5}$ in Mumbai reached the extreme level of 1,010 µg.m-3 during a smog episode that lasted several days. As pollutant concentrations are expected to rise globally in the future, the frequency of high-concentration events could increase, notably in association with global warming, continued urbanisation and internationalisation of industrial production.

The deadly London smog

In 1952, London was brought to a standstill by a dense toxic smog that reduced visibility to a few feet and killed thousands of people, in what remains the worst air pollution crisis in European history.

A wintry cold snap had gripped the British capital in November and December 1952, with heavy snowfalls around the region, and temperatures far below seasonal averages. The people of London, trying to keep warm, burned large quantities of coal in their homes, causing smoke to pour from the chimneys of their houses. Under normal conditions, the smoke would rise into the atmosphere and be dispersed by the wind, but an anticyclone pushing the air downwards trapped the smoke at ground level. The anticyclone had the same effect on particles and gases emitted from factory chimneys and motor vehicles in the London area, along with pollution which the wind carried from industrial areas of the continent. Overall, it is estimated that during the period of the smog, 1,000 tonnes of particles, 2,000 tonnes of carbon dioxide and in particular 370 tonnes of sulphur dioxide were emitted[34].

The smog lasted from the 5th to the 9th of December. It was so dense that in some sections of the city residents were unable to see their feet as they walked. Except for the underground train system, all transportation was crippled: drivers had to turn on their headlights and walk in front of buses with flashlights in broad daylight to inch ahead in the gloom, while boat traffic on the Thames, flights and trains were cancelled.

This smog proved to be so deadly that undertakers ran out of coffins, and florists out of bouquets as the death rate in London's East End increased ninefold in the aftermath of the smog. It killed 4,000 people directly, but mortality did not return to normal levels for several months after the episode (total mortality rates were 50% and 40% higher for January and February, respectively, compared to the previous year). Overall, the most recent studies estimate that this event was directly responsible of the death of 12,000 people, and that more than 100,000 were made ill[35]. It led to several regulations such as the City of London Act of 1954, the Clean Air Acts of 1956 and 1968, as reducing the level of air pollution had become a priority.

[34] *Scott JA, 1963 [28]*
[35] *Stone R., 2002 [29]*

b. *Long-term exposure:*

It is reasonable to expect that impact estimates could be different for different time scales of exposure, that long-term repeated exposure could have greater, more persistent effects than transient short-term exposure, and that long-term average exposure could be different from the cumulative effect of transient short-term exposure. The multicentre European Study of Cohorts for Air Pollution Effects (ESCAPE) aimed to investigate the association between natural-cause mortality and long-term exposure to several air pollutants. The study used data from 22 European cohort studies from general population samples. The total study population consisted of over 350,000 participants who contributed over 5 million person-years to the at-risk database (with an average follow-up period of 13.9 years). Long-term exposure to fine particulate air pollution was associated with natural-cause mortality, even within concentration ranges well below the present European annual mean limit value[36,37].

In addition, a prospective longitudinal cohort study investigated very long-term (38 years) mortality risks of air pollution in England. This study modelled air pollution concentrations in 1971, 1981, 1991 and 2001 in relation to mortality up to 2009 in 367,658 members of the longitudinal survey, a 1% sample of the English Census. Pollutants such as SO2 remained associated with increased mortality decades after exposure. While PM_{10} exposure in 2001 was significantly associated with belated respiratory and cardiovascular mortality, adjusting it for past pollutant exposures in 1971, 1981 and 1991 made cardiovascular and respiratory associations lose significance. This large national study suggests that air pollution exposure has long-term effects on mortality that persist decades after exposure, and that historic air pollution exposures influence current estimates of associations between air pollution and mortality[38]. Furthermore, there is emerging evidence suggesting possible links between long-term $PM_{2.5}$ exposure and neurodevelopment, cognitive function[39], cellular aging[40] and neurodegenerative diseases[41] (Parkinson, Alzheimer's) as well as other chronic disease conditions, such as diabetes.

2. Respiratory diseases

As described in the introduction to this chapter, it is estimated that ambient air pollution is responsible for 27.5% of deaths due to lower respiratory tract infections.

In addition to respiratory infections, multiple sources have demonstrated that exposure to air pollution early in life might contribute to the development of asthma throughout childhood and adolescence. Ulrike Gehring, et al.[42], based on a cohort study of 14,126 participants from Germany, Sweden, and the Netherlands with 14-16 years of follow-up, showed an association between $PM_{2.5}$ and incidence of asthma. Reductions in levels of air pollution could therefore help prevent the development of asthma in children.

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[36] *A significantly increased hazard ratio (HR) for PM2.5 of 1.07 (95% CI 1.02-1.13) per 5 µg/m3 was recorded.*
[37] *Beelen et al., 2014 [2]*
[38] *WHO, 2003 [14], Anna Hansell et al., 2016 [30]*
[39] *Lett et al., 2017 [31]*
[40] *Ward-Caviness et al., 2016 [32]*
[41] *Liu et al, 2016 [33]*
[42] *Ulrike Gehring et al., 2015 [34]*

3. Cardiovascular diseases

Alongside respiratory disorders, cardiovascular diseases have also been widely documented as being associated with air pollution.

In particular, the progression of coronary artery calcium and common carotid artery intima-media thickness has been associated with long-term exposure to ambient air pollution[43]. In a prospective 10-year cohort study, researchers repeatedly measured coronary artery calcium in 6,795 participants aged 45-84 years in the USA. For each 5 $\mu g.m^{-3}$ increase in $PM_{2.5}$, coronary calcium progressed by 4.1 Agatston units per year (95% CI 1.4–6.8).

Particulate matter has also been associated with the short-term risk of mortality from ischaemic heart disease, haemorrhagic stroke and ischaemic stroke[44]. A cohort study in China concluded that an increase in $PM_{2.5}$ of 10 $\mu g.m^{-3}$ led to a 9.7% increase in the risk of mortality from ischaemic heart disease, a 4.4% increase in the risk of mortality from haemorrhagic stroke, and a 13.5% increase in the risk of mortality from ischaemic stroke[45]. Other studies have assessed the association between air pollution and acute decompensated heart failure including hospitalisation and heart failure mortality, and concluded that increases in PM concentration were associated with heart failure, hospitalisation or death[46].The strongest correlations were seen on the day of exposure, with more persistent effects for $PM_{2.5}$[47].

In conclusion, both short- and long-term exposure to air pollution have been shown to be linked to significant increases in both the development of and mortality from a number of cardiovascular diseases.

4. Stroke

Air pollution has also emerged as a significant contributor to the global stroke burden, especially in low-income and middle-income countries[48]. A study based on data from the Global Burden of Disease Study 2013, estimated the population-attributable fraction of stroke-related disability-adjusted life-years (DALYs) associated with potentially modifiable environmental, occupational, behavioural, physiological, and metabolic risk factors in different age and sex groups, in high, middle and low-income countries. The environmental factor of air pollution was the second-largest contributors to DALYs. Globally, 29.2%[49] of the burden of stroke was attributed to air pollution.

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[43] *Joel Schwartz et al., 1996 [20], Peng Yin et al., 2015 [35]*
[44] *Anoop S V Shah et al., 2013 [36]*
[45] *Resp. HR 1.097, 95% CI 1.079–1.116, HR 1.044, 95% CI 1.031–1.057, HR 1.135, 95% CI 1.113–1.158.*
[46] *PM2.5, 2.12% per 10 µg/m3, 95% CI 1.42-2.82; PM10, 1.63% per 10µg.m-3, 95% CI 1.20-2.07*
[47] *IARC of WHO, 2013 [37]*
[48] *Anoop S V Shah et al., 2015 [38]*
[49] *95% UI 28.2–29.6*

This association was confirmed notably by a systematic review and meta-analysis of 2,748 articles, accounting for a total of 6.2 million events across 28 countries and concluding that increases in $PM_{2.5}$ and PM_{10} concentration were associated with admission and mortality for stroke relative risk 1.011 per 10 $\mu g.m^{-3}$ and 1.003 per 10 $\mu g.m^{-3}$, respectively[50].

5. Cancer

The IARC, the World Health Organisation agency specialized in cancer, has classified outdoor air pollution as carcinogenic to humans since 2013. Particulate matter, a major component of outdoor air pollution, was evaluated separately and was also classified as carcinogenic to humans. The IARC report is based on an independent review of more than 1,000 scientific papers including large epidemiological studies of millions of people living in Europe, North and South America, and Asia[51]. It demonstrated an increased risk of lung cancer with increased levels of exposure to PM and air pollution. It also noted a positive correlation with the risk of bladder cancer. Although the composition of air pollution and levels of exposure can vary dramatically between locations, the conclusions of the working group apply to all regions of the world.

The IARC also specifically reviewed the risk of cancer due to outdoor air pollution in China. Epidemiologic data from China, although limited, associated several air pollutants with an increased risk of lung cancer. This association has also been replicated via in vivo experimentation on animals exposed to polluted outdoor air or extracted PM. Numerous studies from China, especially genetic biomarker studies in exposed populations, concurred that the polluted air in China is genotoxic and carcinogenic to humans[52].

Lung cancers take time to develop and could be associated with chronic long-term exposure to air pollution rather than with short-term peaks. Indeed, the incidence of lung cancer has been positively associated with increments in 2-year mean $PM_{2.5}$[53]. It has also been found that this risk is more pronounced for women than for men[54]. The association is less significant in rural areas than in urban areas[55] and is stronger in elderly people (>75 years of age) compared to younger people (30–65 years old)[56],which could be explained notably by differences in the duration of exposure to carcinogenic pollutants.

In Europe, a prospective analysis by the European Study of Cohorts for Air Pollution Effects, based on data from 17 European cohorts focused on the incidence of lung cancer cases during a 12.8-year follow-up period[57]. The meta-analyses demonstrated a statistically significant association between the risk of lung cancer and both PM_{10} and $PM_{2.5}$[58].

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[50] *Yuming Guo et al., 2016 [39]*
[51] *Dana Loomis et al., 2014 [40]*
[52] *Wang et al., 2016 [42]*
[53] *Ole Raaschou-Nielsen et al., 2013 [43]*
[54] *A significantly increased hazard ratio (HR) for PM2.5 of 1.07 (95% CI 1.02-1.13) per 5 µg/m3 was recorded.*
[55] *RR 1·037, 95% CI 0·998–1·078 and RR 1.060, 95%CI 1.044–1.075, respectively; P=0·03.*
[56] *RR 1.111, 95% CI 1.077-1.146, RR 1.074, 95% CI 1.052-1.096, respectively; P=0.07.*
[57] *Valery L Feigin et al., 2013 [44]*
[58] *Hazard ratio [HR] of 1.22, 95% CI 1.03–1.45 per 10 µg.m-3, 1.18, 95% CI 0.96-1.46 per 5.µm-3.*

From an individual standpoint, smoking has a bigger effect on the risk of developing lung cancer than air pollution. However, while only a portion of the population is exposed to smoking, almost everyone breathes polluted air at some point. This explains why air pollution is considered a significant driver for lung cancer in the overall population. In the UK, the Institute for Cancer Research estimates that 7.8% of lung cancers are linked to air pollution in the UK.

6. Other health effects

a. *Infant health*

The epidemiologic impact of pollution on the population also apply to infants. Recent studies have underlined specific risks that air pollution could represent for infants, and even on the intrauterine growth.

The relationship between intrauterine growth retardation (IUGR) and exposure to PM_{10} has been documented[59]. An analysis of the highly polluted district of Teplice (Northern Bohemia) led to an investigation on the possible role of the carcinogenic fraction of polycyclic aromatic hydrocarbons (c-PAHs), which are usually bound to fine particles. The results indicated that exposure to c-PAHs in early gestation may influence foetal growth. The particulate matter–IUGR association observed earlier may be at least partly explained by the presence of c-PAHs on particle surfaces. In 2011, a study was performed on 74,178 women who had had single baby deliveries between February 1994 and June 2011. For these births, information was available on infant birthweight, gestational age, and sex. The study concluded that a 5 $\mu g.m^{-3}$ increase in the concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term[60],[61]. An increased risk during pregnancy[62] was also recorded for concentrations below the present European Union annual $PM_{2.5}$ limit of 25 $\mu g.m^{-3}$.

PM_{10} was also associated with increased risk of low birthweight at term. The population attributable risk estimated for a reduction in $PM_{2.5}$ concentration to 10 $\mu g.m^{-3}$ during pregnancy corresponded to a decrease of 22% (95% CI 8–33%) in cases of low birthweight at term.

In addition, post-neonatal infant mortality was compared with levels of PM_{10} concentration during the two months after birth[63]. After controlling for maternal race, maternal education, marital status, month of birth, maternal smoking during pregnancy, and ambient temperatures, the study results demonstrated an association between PM pollution and post-neonatal infant mortality for respiratory causes and sudden infant death syndrome.

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[59] *Marie Pedersen et al., 2013 [46]*
[60] *Adjusted odds ratio [OR] 1.18, with 95% CI 1.06–1.33.*
[61] *Ulrike Gehring et al., 2015 [34]*
[62] *OR for 5 µg.m-3 increase in participants exposed to concentrations of less than 20 µg.m-3 1.41, 95% CI 1.20–1.65.*
[63] *C. Arden Pope III et al., 2006 [47]*

b. *Neurodegenerative and neurodevelopmental consequences of air pollution*

Recent studies have demonstrated that long-term exposure to air pollution is associated with age-related diseases. Firstly, all the aforementioned diseases (cardiovascular, cancer, respiratory diseases) are themselves associated with ageing[64]. Secondly, exposure to air pollution has been associated with DNA damage[65], epigenetic alterations[66] and oxidative stress. One possible explanation for this correlation is that air pollution, in particular particulate matter and nitric oxides, could be associated with the reduction of telomere length, a core parameter of the biological process of ageing[67]. Studies have therefore investigated the relationship between air pollution and cognitive ageing and have demonstrated that the former could accelerate the latter and with it the risk of Alzheimer's disease and other forms of dementia[68].

c. *Other human physiological functions*

In addition to the impact on the diseases mentioned in the previous paragraphs, several studies have provided evidence to suggest further consequences of air pollution on human health. A recent Chinese study demonstrated that increased concentrations of PM_{10}, SO_2, and NO_2 were significantly associated with deteriorations in many categories of physiological function, such as increase in heart rate and blood pressure (heart function), increase in urine creatinine and urea (renal function), decrease in haemoglobin and white blood cell (haematopoietic function), increase in cholesterol, low-density lipoprotein, high-density lipoprotein, and the ratio of low-density lipoprotein to high-density lipoprotein (metabolic endocrine function), increase in serum albumin, glutamic-pyruvic transaminase, and total bilirubin (liver function), and increase in C-reactive protein (inflammatory response function)[69]. Generally, elderly people, women, and overweight people appear to be more susceptible to air pollution than young people, men, and people of normal weight. This study illustrates the fact that air pollution could systemically damage a number of human physiological functions.

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[64] *Fries et al., 1980 [48]*
[65] *Risom et al., 2005 [49]*
[66] *Bind et al., 2014 [50]*
[67] *Ward-Caviness et al., 2016 [51]*
[68] *Underwood, 2017 [52]*[w]
[69] *Jan Dejmek et al., 2000 [45]*

7. Reversibility of air pollution effects

There is consistent evidence to suggest that lower air pollution levels following a sustained, long-term exposure results in health benefits for the population, with rapid improvement in population health (a few years after the reduction in pollution). A few examples are summarized below. These examples of successful intervention show that decreased levels of particulate air pollution can substantially diminish total respiratory and cardiovascular death rates[70].

A cohort of adults living in six cities in the United States was followed from 1974 to 2009 in order to estimate the effects of air pollution on mortality. Overall, $PM_{2.5}$ concentration had decreased to below 15 $\mu g.m^{-3}$. The main finding was that a 2.5 $\mu g.m^{-3}$ decrease in the annual average level of $PM_{2.5}$ was associated with a 3.5% reduction in all-cause mortality[71].

A copper smelter strike in 1967-68 in four US states, and the closure and reopening of a steel mill in Utah Valley in 1986-87, are two examples of unplanned events that had a positive impact on health by decreasing air pollution concentration in specific areas. Furthermore, the reported 3.2% drop in daily numbers of deaths was associated with a simultaneous fall in PM_{10} levels of approximately 15 $\mu g.m^{-3}$ while the steel mill was closed, the strongest association being with respiratory deaths[72].

A Swiss Study on Air Pollution and Lung Diseases in Adults assessed lung diseases in adults from eight Swiss communities in 1991 and again in 2002. Falling levels of regional PM_{10} were associated with a declining prevalence of various respiratory symptoms, including chronic cough, bronchitis, common cold, nocturnal dry cough and conjunctivitis symptoms[73].

[70] *WHO, 2013 [10]*
[71] *WHO, 2013 [10]*
[72] *WHO, 2013 [10]*
[73] *WHO, 2013 [10]*

III. Air pollution and the (re)insurance industry

1. The cost of air pollution: consequences on the economy

In addition to having an impact on the liabilities of (re)insurance companies, air pollution could have an impact on their assets, through a more global impact on the economy. Indeed, economic activity generates air pollution, which through its biophysical effects (morbidity and mortality) imposes economic costs. The World Bank stated in a recent report that *"premature deaths due to air pollution in 2013 cost the global economy about $225 billion in lost labour income, or about $5.11 trillion in welfare losses worldwide. This is about the size of the gross domestic product of India, Canada, and Mexico combined – and a sobering wake-up call"*[74].
These costs can be market-based, and materialise through concrete economic transactions that have a monetary cost, such as lost labour productivity, admissions to hospitals and disease treatment, diminished agricultural yields or decreased tourism revenues. They can also be non-market based, i.e. not give rise to concrete monetary transactions, in which case they can be inferred from the sum of money that economic agents would be ready to pay to avoid their detrimental consequences (death and suffering).

Computing the aggregate market-based cost of pollution requires the combination of at least three different models: one that translates economic activity into air pollution, another that quantifies the biophysical effects of air pollution (number and geographical extent of deaths and diseases) and one that models the effect of death and disease on economic activity.
Needless to say, such a chain of models gives a necessarily simplified representation of reality, because it requires numerous assumptions that (i) reflect our ignorance and (ii) make the mathematical system manageable.
The OECD has recently proposed a model of the market-based costs of outdoor air pollution that takes into account lost labour productivity, increased expenditure on health and diminished agricultural yields. Estimated at 0.3% of world GDP, these costs are expected to grow much faster than GDP and reach 1% of GDP in 2060. The bulk of the cost would be accounted for by lost labour productivity and health expenditures. These market costs tend to be concentrated in Eastern Europe and Asia. China, for example, would incur an economic loss equivalent to 2.5% of GDP in 2060. More developed OECD countries would suffer relatively less, with the impact on the USA for example, being less than 0.5% of GDP.

[74] *World Bank, 2016 [53]*

Non-market costs do not stem from concrete economic transactions, so their cost is purely virtual; it is evaluated on a willingness-to-pay basis. If people could pay to avoid the suffering and risk of death from air pollution, how much would they be ready to pay for it? This amount is estimated through direct valuation studies that aggregate individual willingness-to-pay to arrive at the aggregated value of a statistical life or life in good health.
Based on such measures, the OECD estimates that the non-market based cost of premature deaths would rise sevenfold by 2060, from USD 3 trillion in 2015 to EUD 18-25 trillion in 2060. The cost of morbidity, estimated at USD 2 trillion in 2060, should also be added to this figure . Taken together, this adds up to USD 20-27 trillion in 2060, which is several orders of magnitude higher than the amount of market-based costs (USD 3.3 trillion in 2060). This should not come as a surprise given that the bulk of the cost is accounted for by the value of lost lives, which is necessarily higher than that of lost agricultural crops or industrial output in people's own subjective valuation used to derive willingness-to-pay measures.

Microeconomic studies have also successfully established that local non-market based costs of air pollution can be very high. For example, Jha and Muller (2017)[75] demonstrated that non-market costs of higher mortality induced by US powerplant coal stockpiles in a 25-mile radius are four times higher than the market cost of coal paid by powerplants. Barreca et al. (2017) show that the non-market based costs of higher mortality induced by coal powerplants are high and increase markedly with the length of exposure to the pollutants[76].

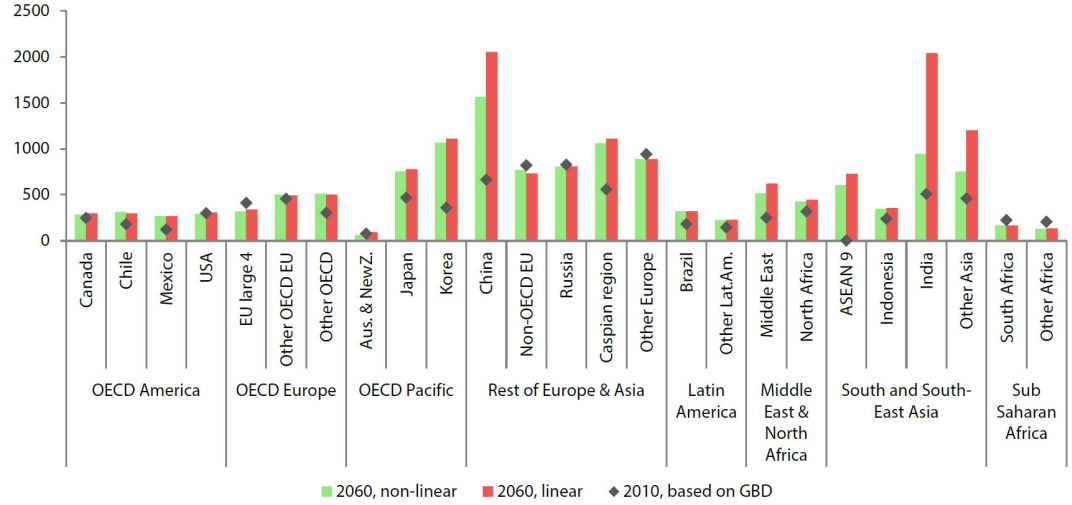
2. A growing challenge for life (re)insurers

As described in the numerous studies reviewed in this publication, air pollution has already had significant adverse effects on mortality and morbidity. The current forecasts for the development of air pollution in the coming decades indicate that its global impact will be even more severe in the future. This has significant consequences for human life and will therefore affect the insurance and reinsurance industries, which will have to adapt their policies and services to meet a growing threat.

[75] *Akshaya Jha, Nicholas Z. Muller 2017 [54]*
[76] *Barreca et al., 2017 [55]*

The first consequence is that air pollution increases mortality and morbidity of critical conditions on both the short and long term. Claims and premiums already reflect the impact of pollution in areas where air quality has been steady in recent years. Due to the projected increase in air pollution, however, the long-term trend will most likely lead to an increase in claims. On the other hand, the consequences of short-term smog events, which could occur more frequently as global levels of pollution rise, will have to be understood and might pose a particular threat to local (re)insurers. Actuaries, underwriters and risk management need to form a view on how much the levels of air pollution will affect future claims, and how these losses will be distributed between trend and short-term risk. Insurers and reinsurers will have to anticipate the possible lag between cause and effect in particular cases associated with long-term exposures in order to produce appropriate pricing. Last, discrepancies among local levels of air pollution could lead to significant variations in pricing assumptions. The OECD projected the number of premature deaths from exposure to ozone and particulate matter by 2060 in several areas of the world, taking into account the increase in pollutant concentrations, in urbanisation and aging population. The below figure highlight the geographical disparities mentioned in the first chapter of this report. The highest number of deaths is expected to take place in non OECD economies, but small increases can also be expected in OECD countries, as Japan or Korea.

Figure 5: Premature deaths from exposure to particulate mater and ozone by 2060[77]



[77] *OECD , 2016 [16]*

To manage a cause of aggravated risk, insurance companies often have recourse to medical selection, to propose a bespoke premium for a given policyholder risk. In the case of air pollution, as everyone must breathe outside air, such a process would be challenging to implement. One could even argue that behaviours normally associated with improved risk profile, such as the regular practice of sports, could see its benefits lowered if the activity is taking place in an area where there are high levels of air pollution. The latest findings indicate that the benefits of sports outweigh the consequences of air pollution, even in polluted areas[78]. On the other hand, other studies have demonstrated that outside air pollution discourages physical activity, possibly leading to other negative side effects[79].

3. Possible consequences for (re)insurance demand

In addition to the consequences on health, air pollution could directly impact insurers' businesses, because it could lead to an increase in demand for coverage as the threat of these potential illnesses becomes better known by people and governments. This demand could lead (re)insurers to develop appropriate products and services.

Some local initiatives demonstrate this trend, as in the case in China, where the Ministry of Environmental Protection and the China Insurance Regulatory Commission (CIRC) released a draft regulation in 2017 for mandatory pollution cover on specific industries. Indeed, companies in eight categories of businesses (including oil and gas, chemicals, pharmaceutical engineering) will now have to buy environmental insurance cover for the costs of pollution in a liability policy. This will lead to the development of new types of coverage for (re)insurers.

As described in the first chapter, air pollution is expected to increase more dramatically in less developed countries. In spite of substantial increases in premium growth rates in recent years, insurance penetration is still lower in these countries, leading to the so-called insurance protection gap[80]. The fact that air pollution could have more severe consequences in these regions of the world increases both the need to close the protection gap and the challenge it could create, in terms of the magnitude of potential losses for the (re)insurance industry.

[78] *Silva-Renno et al., 2017 [56]*
[79] *An et al., 2017 [57]*
[80] *Kai-Uwe Schanz and Shaun Wang (ed.), 2014 [58]*

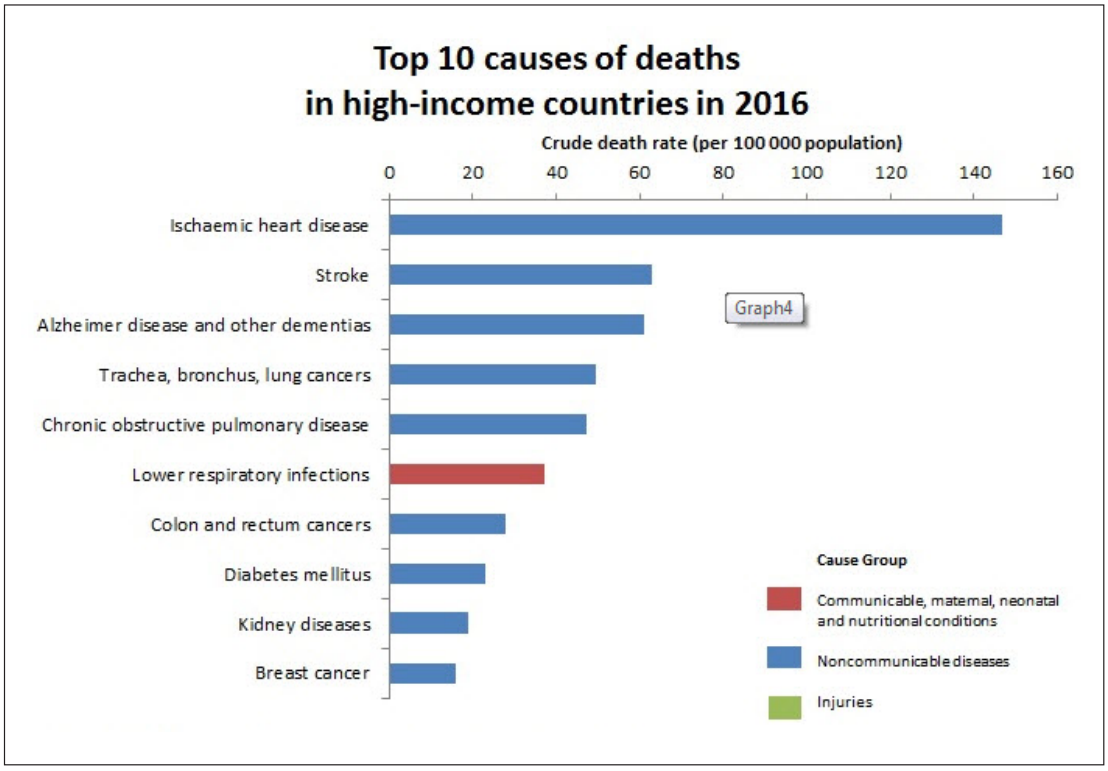
Conclusion

Air pollution will be a rising concern in the coming years, with significant consequences on the (re)insurance industry. Beyond its future impact, this emerging risk already significantly affects mortality and morbidity of several critical conditions including cardiopulmonary illnesses, cancer, stroke, etc. This report could incentivise (re)insurers to follow several recommendations, summarized below:

- Add air pollution to the list of emerging risks to be monitored by risk management;
- Identify the current exposure of mortality and morbidity portfolios to air pollution, and develop extreme scenarios to assess the potential impact of a severe pollution event;
- Draw the attention of public authorities to the need to provide high standards database and forecasts of air pollution levels;
- Promote awareness on the impact of air pollution and the best prevention measures that can be taken;
- Acknowledge that some sectors or lines of business such as thermal coal pose greater environmental risks and support endeavours to disengage from these industries on both the asset management and the underwriting sides.

Appendix

I. Top 10 causes of death globally (WHO)[81]



[81] *WHO [59]*

II. Methodology

Epidemiological, toxicological, and clinical studies have shown PM mass comprises fractions with varying types and degrees of health effects, suggesting a role for both the chemical composition and physical properties.

- **Epidemiological studies:** For coarse particles, there was only limited epidemiological data. The availability of epidemiological data has significantly increased since 2005. Taking into account the newest evidence on the effects of coarse PM on cardiorespiratory health, the EPA (United States Environmental Protection Agency) has integrated scientific assessment for PM and concluded that, in general, short-term epidemiological studies reported positive associations between mortality and cardiovascular and respiratory hospital admissions. Black carbon (BC) is the most strongly light-absorbing component of PM and it is emitted directly into the atmosphere in the form of fine and ultrafine particles. Two-pollutant models in time-series studies suggested that the effect of BC particles was more robust than the effect of PM mass[82].

- Evidence was also judged sufficient for an association between long-term BC concentration and all-cause and cardiopulmonary mortality[83]. Fine PM and sulphur oxide-related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10 $\mu g.m^{-3}$ elevation in fine PM air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively[84]. A link between ultrafine particles or total number concentrations and cardiovascular disease hospital admission was observed in European multicentre studies. The link between ultrafine particles or total number concentrations and natural cause mortality appeared to be more robust in time-series analyses[85].

- **Toxicological studies: For coarse particles, as** their inhalability and, therefore, their deposition efficiency in the respiratory tract is substantially lower, the interpretation of the risk of coarse versus fine PM has to be considered in that context. Intratracheal exposure in rats and mice as well as in vitro studies suggest that similar effects can be observed for coarse and fine PM in the bioassays of lung cells, and that coarse PM can be even more hazardous than fine PM. Again, given that the deposition efficiency and pattern of coarse and fine PM differ greatly, the health outcomes in a population can differ at equal mass exposure[86].

[82] *C Arden Pope III et al., 2002 [60]*
[83] *C Arden Pope III et al., 2002 [60]*
[84] *Mills NL et al., 2011, [61]*
[85] *C Arden Pope III et al., 2002 [60]*
[86] *C Arden Pope III et al., 2002 [60]*

- Another study found inhalation of ultrafine carbon particles (38 nm, 180 $\mu g.m^{-3}$ for 24 hours) caused increased heart rate and decreased heart-rate variability in rats, but there was no inflammatory response and no change in the expression of genes having thrombogenic relevance[87]. In spontaneously hypertensive rats exposed to similar ultrafine carbon particles (172 $\mu g.m^{-3}$ for 24 hours), blood pressure and heart rate increased with a lag of 1-3 days. Inflammatory markers in lavage fluid, lung tissue, and blood were unaffected, but mRNA expression of hemeoxygenase-1, endothelin-1, endothelin receptors, tissue factor, and plasminogen activator inhibitor in the lung showed a significant induction[88].

- There have been advances in the understanding of the action of ultrafine particles, which have the ability to translocate from the alveolar space into tissues and to spread systemically, reaching many organs, including the heart, liver, kidneys and brain. Specific toxicological action includes impairment of phagocytosis and breakdown of defence mechanisms, crossing tissues and cell membranes, injury to cells, generation of reactive oxygen species, oxidative stress, inflammation, production of cytokines, depletion of glutathione, mitochondrial exhaustion, and damage to protein and DNA, most of which also occur with larger size PM[89].

- **Clinical studies:** Graff et al. (2009) arrived at the conclusion that, in humans (2 hours, 90 $\mu g.m^{-3}$), exposure to coarse PM produces a mild, measurable physiological response in healthy young volunteers that is similar in scope and magnitude to that of volunteers exposed to fine PM, suggesting that both size fractions are comparable in inducing cardiopulmonary changes in acute exposure settings[90]. The effects of ultrafine carbon particles were observed in both heart-rate variability and cardiac repolarisation, but there were no changes in soluble markers of either systemic inflammation or coagulation. In a more recent study, no vascular impairment or effect on blood clotting was observed in volunteers exposed for 2 hours to 70 $\mu g.m^{-3}$ of ultrafine carbon particles[91]. In another clinical study, the removal of very high particle numbers by filters prevented the otherwise occurring arterial stiffness and increases of blood clotting[92]. Similar observations were made in health subjects and patients with coronary heart disease who were wearing a very simple, yet highly efficient face mask while walking in highly polluted areas in Beijing, China[93].

[87] *Swapna Upadhyay et al., 2008 [62]*
[88] *Brauner EV et al., 2008 [63]*
[89] *C Arden Pope III et al., 2002 [60]*
[90] *C Arden Pope III et al., 2002 [60]*
[91] *Volker Harder et al., 2005 [64]*
[92] *Jeremy P Langrish et al., 2009 [65]*
[93] *Joel D Kaufman et al., 2016 [66]*

III. WHO Air quality guidelines

The 2005 "WHO Air quality guidelines" offer global guidance on thresholds and limits for key air pollutants that pose health risks. Small PM pollution have health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed. Therefore, the WHO 2005 guideline limits aimed to achieve the lowest concentrations of PM possible[94].

Guideline values:

- $PM_{2.5}$, 10 µg.m^{-3} annual mean, 25 µg.m^{-3} 24-hour mean;
- PM_{10}, 20 µg.m^{-3} annual mean, 50 µg.m^{-3} 24-hour mean.

Long-term exposure: An annual average concentration of 10 µg.m^{-3} was chosen as the long-term guideline value for $PM_{2.5}$. This represents the lower end of the range over which significant effects on survival were observed in the American Cancer Society's (ACS) study. Besides the guideline value, three interim targets (IT) are defined for $PM_{2.5}$ (see Table 2). These have been shown to be achievable with successive and sustained abatement measures. Countries may find these interim targets particularly helpful in gaug¬ing progress over time in the difficult process of steadily reducing population exposure to PM.

[94] *Anoop S V Shah et al., 2015 [38]*

Table 1. WHO Air quality guidelines and interim targets for PM: annual mean concentrations[95]

	$PM_{10}(\mu g.m^{-3})$	$PM_{2.5}(\mu g.m^{-3})$	Basis for the selected level
Interim target-1(IT-1)	70	35	These levels are associated with about a 15% higher long-term mortality risk relative to the AQG level
IT-2	50	25	In addition to other health benefits, these levels lower the risk of premature mortality by approximately 6% (2-11%) relative to the IT-1 level
IT-3	30	15	In addition to other health benefits, these levels lower the risk of premature mortality by approximately 6% (2-11%) relative to the IT-2 level
Air quality guideline (AQG)	20	10	These are the lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to long-term exposure to $PM_{2.5}$

- **Short-term exposure:** Meeting the guideline values for the 24-hour mean will how¬ever protect against peaks of pollution that would otherwise lead to substantial excess morbidity or mortality. It is recommended that countries with areas not meeting the 24-hour guideline values undertake immediate action to achieve these levels in the shortest possible time.

[95] *Who , 2005 [67]*

Table 2. WHO Air quality guidelines and interim targets for PM: 24-hour concentrations[96]

	PM_{10} ($\mu g.m^{-3}$)	$PM_{2.5}$ ($\mu g.m^{-3}$)	Basis for the selected level
Interim target-1(IT-1)	150	75	Based on published risk coefficients from multi-centre studies and meta-analyses (about 5% increase in short-term mortality over the AQG value)
IT-2	100	50	Based on published risk coefficients from multi-centre studies and meta-analyses (about 2.5% increase in short-term mortality over the AQG value)
IT-3	75	37,5	Based on published risk coefficients from multi-centre studies and meta-analyses (about 1.2% increase in short-term mortality over the AQG value)
Air quality guideline (AQG)	50	20	Based on relationship between 24-hour and annual PM levels

[96] *Who , 2005 [67]*

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