Health effects from air pollution

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Backgrounddocument to:

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contents

01 introduction

1.1 Mechanisms of health effects of air pollution

The inhalation of elevated concentrations of particulate matter, nitrogen dioxide, and ozone leads to inflammatory responses of the mucous membranes in the respiratory tract and lungs.[1-3] Depending on the duration of the exposure, these inflammatory responses can cause the following:

- reduced pulmonary function;
- increased airway reactivity;
- allergic reactions;
- respiratory symptoms, especially in asthma patients;
- an increased susceptibility to infections of the respiratory tract and lungs;
- the development of asthma (in case of long-term exposure).

Another possible mechanism operates via “oxidative stress”. This means that the inhalation of air pollutants leads to an increase in reactive oxygen radicals, which can not only cause damage to lung tissue but also to the cardiovascular system and other organs.

1.2 Burden of disease attributed to air pollution

The burden of disease attributed to air pollution is the sum of the years of life lost due to premature death and the healthy life years lost due to disabilities (i.e. disability-adjusted life years). Premature death is often expressed in two different ways, namely as the loss of years or months of life or as the number of premature deaths. The second formulation would seem to be easier to understand than the first, but upon further consideration, calculating the number of deaths that can be attributed to air-pollution is a complex affair.

The years of life lost can be deduced directly from studies in which the remaining life years as well as the remaining healthy life years are compared for groups of people exposed to higher or lower levels of air pollution. Such studies can also be used to calculate relative risks that indicate how much higher the risk of dying is as a result of exposure to a certain level of air pollution. The relative risk can then be used to calculate which fraction of the deaths can be attributed to air pollution (the population-attributive fraction: proportion of deaths attributable to air pollution). This then results in a calculation of the number of deaths per year that can be attributed to air pollution. However, the problem with this last calculation is that it is not really possible to determine how the loss in years of life is distributed over the population exposed to air pollution. In one extreme case, the loss would be distributed over a very small number of persons who all die at a very early age. At the other end of the scale,

the loss would be distributed over the entire population whereby each member suffers a much smaller decrease in life expectancy.
In practice, both methods for the calculation of premature death are often presented: the number of premature deaths as well as the number of years or months of life lost. We have also done so in the advice at hand.

More information
For a detailed overview of estimates of the health effects of exposure to air pollution for the Dutch population, the committee refers the reader to the website: www.volksgezondheidenzorg.info.[4]

1.3 Weight of evidence for causal relationship with air pollution

In this advice, the committee is limiting itself to a description of those health effects that are certainly or most likely caused by exposure to air pollution. The committee is thereby basing itself on the following distinction made by the *Environmental Protection Agency* (EPA) in the *Integrated Science Assessments* (ISA) regarding the weight of evidence for a causal relationship between air pollution and health effects: *demonstrated* ('proven') or *likely*.[5]

Demonstrated
There is *sufficient evidence to conclude that there is a causal relationship* if the relationship can be inferred on the basis of consistent findings from multiple high-quality studies in which chance, confounding, and other biases can be ruled out with reasonable confidence. Studies that comply with the above criteria are:

- controlled human exposure studies, or
- observational studies supported by other lines of evidence such as animal studies.

Likely
There is *sufficient evidence to conclude that there is likely to be a causal relationship* if the relationship can be inferred on the basis of multiple high-quality studies the results of which cannot be explained by chance, confounding, and other biases, but uncertainties remain in the evidence overall. Studies that comply with the above criteria are:

- observational studies that show an association with indicators but where the association can also be ascribed to exposure to other agents, or where other lines of evidence, such as animal studies, are limited or inconsistent;
- animal toxicological studies that are not supported by human data.

1.4 Concentration-effect relationships

In 2013, the WHO published the report *Health risks of air pollution in Europe (HRAPIE)*, which was summarised in 2015 in an article by Heroux et al.[6,7] Based on the information available until 2013, the WHO summarised the concentration-effect relationships between particulate matter, nitrogen dioxide, and ozone on the one hand and the various

short-term and long-term health effects and premature death on the other. In the concentration-effect relationship, concentration is compared to relative risk. In the case at hand, it informs us about the extra risk that adverse health effects will occur for each additional increment of 10 microgram/m^3.

Small risks, major consequences

The relative risks per substance are presented in the following paragraphs. These – often statistically significant – relative risks per 10 micrograms/m^3 appear to be small at first sight, but as practically the entire population is exposed to air pollution, including large variations in time and place, the health effects at the population level can be considerable.

02 health effects of particulate matter

Particulate matter is a collective name for all particles in the air that are small enough to be inhaled by human beings. The abbreviation PM (*particulate matter*), followed by a number, is used to specify the size of the particles. For example, PM2.5 means that 50% of the particles have a diameter of less than 2.5 micrometres.

2.1 Weight of evidence for health effects of particulate matter

The tables below present the weight of evidence for a relationship between short-term and long-term exposure to particulate matter (PM2.5), respectively, and health effects.[1] *Short-term exposure* is exposure lasting between one day and one week. *Long-term exposure* is exposure over a period of between 1 year and 5 to 10 years.

Table 1. Weight of evidence for causal relationships between short-term exposure to PM2.5 and health effects (EPA, 2009)[1]

Health effects	Weight of evidence for causal relationship
Adverse effects on cardiovascular system, in particular an increase in hospital admissions and emergency department visits for cardiovascular diseases.	Demonstrated
Adverse effects on cardiovascular system, in particular increased mortality from cardiovascular diseases.	Demonstrated
Adverse effects on the respiratory tract and lungs, in particular: • decrease in lung function growth in children • increase in respiratory symptoms in adults	Likely
All-cause mortality	Demonstrated

Table 2. Weight of evidence for causal relationships between long-term exposure to PM2.5 and health effects (EPA, 2009)[1]

Health effects	Weight of evidence for causal relationship
Adverse effects on cardiovascular system, in particular increased mortality from cardiovascular diseases	Demonstrated
Adverse effects on the respiratory tract and lungs, in particular: • reduced lung function growth in children • more respiratory symptoms in adults	Likely
All-cause mortality	Demonstrated

2.2 Short-term (peak) exposure to particulate matter

Adverse effects on the cardiovascular system

Epidemiological studies show associations between short-term exposure (24-hour average concentration) and an increase in effects on the cardiovascular system, including emergency admissions to hospital due to acute cardiac symptoms such as (threat of) a heart attack or heart failure.[1] The EPA concludes that the health effects found are certainly caused by exposure to PM2.5. In doing so, the EPA also bases itself on clinical and toxicological studies in which effects have been demonstrated on blood pressure, heart rhythm, and various markers of vasoconstriction. The resulting acute physiological changes can be fatal (heart attack or heart failure) for persons with heart disease.

Adverse effects on the respiratory tract and lungs

According to the EPA, there is a likely causal relationship between short-term exposure to particulate matter and effects on the respiratory tract and lungs. Epidemiological studies show that periods of smog are accompanied by an increase in effects on the respiratory tract and lungs, including an increase in infections of the respiratory tract and lungs and of emergency admissions to hospitals due to respiratory symptoms.[1] Clinical and toxicological studies provide indications of acute inflammatory responses of tissues in the respiratory tract and lungs and acute hypersensitivity reactions.

Mortality

According to the EPA, there is convincing evidence that exposure to PM2.5 results in an increase in all-cause mortality as well as of cause-specific mortality from cardiovascular diseases and diseases of the respiratory tract and lungs.[1]

2.3 Long-term exposure to particulate matter

Adverse effects on the cardiovascular system and mortality

Epidemiological studies involving large cohorts in Europe as well as North America show that long-term exposure to PM2.5 is associated with increased effects on the cardiovascular system as well as mortality from heart attack or heart failure.[1] The EPA concludes that these studies, together with increased insight into the plausibility of the mechanism involved obtained from toxicological studies, provides convincing evidence for a causal relationship.

Adverse effects on the respiratory tract and lungs

The EPA concludes that the effects on the respiratory tract and lungs found in epidemiological studies can, with a high degree of probability, be ascribed to long-term exposure to PM2.5.[1] Although the epidemiological studies do show reduced lung function and an increase in respiratory symptoms, they do not show increased mortality from diseases of the respiratory tract and lungs. The EPA concludes that there is sufficient

evidence to infer a likely causal relationship with adverse effects on the respiratory tract and lungs, in part on the basis of the increase in inflammatory responses of tissues in the respiratory tract and lungs found in clinical and toxicological studies. This reveals an underlying mechanism that leads to the adverse effects on the respiratory tract and lungs found in epidemiological studies.

Adverse health effects of black carbon

When it comes to the effects of large-scale air pollution, it does not make any difference whether black carbon or PM2.5 is used to estimate health effects due to the strong correlation between black carbon and PM2.5. Only at the local level, for example near busy traffic routes, are the estimated health effects higher if black carbon is used as an indicator.[8] The WHO concludes that not enough clinical and toxicological studies have been carried out to be able to fully explain the mechanism of how exposure to black carbon results in health effects.

2.4 Concentration-effect relationships for particulate matter

In 2013, the WHO published the report *Health risks of air pollution in Europe (HRAPIE)*, which was summarised in 2015 in an article by Heroux et al.[6,7] Based on the information available until 2013, the WHO summarised the concentration-effect relationships between particulate matter, nitrogen dioxide, and ozone on the one hand and the various short-term and long-term health effects and premature death on the other.

In the concentration-effect relationship, concentration is compared to relative risk. In the case at hand, it informs us about the extra risk that adverse health effects will occur for each additional increment of 10 micrograms/m^3. The tables below presents the relative risks for particulate matter.

Table 3. Concentration-effect relationships for particulate matter, *short-term effects*[6]

PM Indicator of exposure	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
PM2.5 daily average Concentrations	Mortality, all (natural) causes of death (traffic deaths, victims of crime et cetera not included), all ages	1.012 (1.005-1.020)
	Hospital admissions for cardiovascular diseases (including stroke), all ages	1.009 (1.002-1.017)
	Hospital admissions for diseases of the respiratory tract, all ages	1.019 (0.998-1.040)
PM10 daily average Concentrations	Incidence of respiratory symptoms in asthmatic children between the ages of 5 and 19	1.028 (1.006-1.051)

[a] Relative risk with 95% confidence interval.

Table 4. Concentration-effect relationships for particulate matter, *long-term effects*[6]

PM Indicator of exposure	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
PM2.5 annual average concentrations	Mortality, all (natural) causes of death, up to 30 years	1.062 (1.040-1.083)
	Mortality from cardiovascular diseases (including stroke), chronic obstructive pulmonary disorder (COPD), and cancer of the respiratory tract and lungs, up to 30 years	-
PM10 annual average concentrations	Post-neonatal mortality (1 to 12 months) from all (natural) causes of death	1.04 (1.02-1.07)
	Incidence of bronchitis in children between the ages of 6 and 12/18	1.08 (0.98-1.19)
	Incidence of chronic bronchitis in adults (>18 years)	1.117 (1.040-1.189)

[a] Relative risk with 95% confidence interval.

2.5 High-risk groups for exposure to particulate matter

Table 5. High-risk groups for health effects from (short-term) exposure to particulate matter[1,9]

Risk factors and risk groups studied	Rationale for conclusion that there is 'sufficient evidence'
Children (<18 years)	Children show increased sensitivity to respiratory symptoms
Older adults (>65 years)	Older adults show increased sensitivity to respiratory and cardiac symptoms and high blood pressure. A recent literature review presents strong indications of a high risk of hospital admission or death[9]
Cardiovascular patients	Cardiovascular patients show an increase in heart symptoms and high blood pressure
Asthma patients	Asthma patients show an increase in respiratory symptoms

03 health effects of nitrogen dioxide

3.1 Evidence for health effects of nitrogen dioxide

Tables 6 and 7 present the weight of evidence for a relationship between short-term exposure and long-term exposure to nitrogen dioxide, respectively, and health effects. *Short-term exposure* to nitrogen dioxide is defined as lasting more than a few minutes to 1 hour but less than 1 week to 1 month. *Long-term exposure* to nitrogen dioxide is exposure over a period between 1 year and 5 to 10 years.

Table 6. Weight of evidence for a causal relationship between short-term exposure to nitrogen dioxide (NO2) and health effects (EPA, 2016)[2]

Health effects	Weight of evidence for causal relationship
Adverse effects on the respiratory tract and lungs, in particular: • temporary decrease in pulmonary function • increased bronchial reactivity • increase in inflammatory responses of respiratory tract and lungs • increase in respiratory symptoms and use of medication in children with asthma • increased susceptibility to infections of the respiratory tract and lungs • increased allergic reactions	Demonstrated

Table 7. Weight of evidence for a causal relationship between long-term exposure to nitrogen dioxide (NO2) and health effects (EPA, 2016)[2]

Health effects	Weight of evidence for causal relationship
Adverse effects on the respiratory tract and lungs, in particular an increased incidence of asthma in children	Likely

3.2 Short-term (peak) exposure to nitrogen dioxide

Adverse effects on the respiratory tract and lungs

The EPA concluded at the beginning of 2016 that there is convincing evidence for a causal relationship between short-term exposure to nitrogen dioxide and effects on the respiratory tract and lungs.[2]

Adverse effects on the cardiovascular system

The EPA concludes that the weight of evidence for a causal relationship with adverse effects on the cardiovascular system is less strong but is still likely. The EPA based this conclusion on epidemiological studies under adults which revealed, among other things, a consistent relationship between short-term exposure to nitrogen dioxide and the occurrence of (the threat of) a heart attack as well as hospital admissions and mortality due to cardiovascular diseases.[10,11]

Mortality

After publication of the EPA report, a meta-analysis was released of some dozens of time-series studies on the effects of short-term exposure to nitrogen dioxide on hospital admissions for respiratory and cardiovascular diseases and mortality due to such diseases as well as on all-cause mortality.[12] This meta-analysis shows that the effects of nitrogen dioxide on deaths, expressed per 10 micrograms/m^3, are of the same order of magnitude as the effects of particulate matter. A great number of the underlying studies reported that the effects found for nitrogen dioxide do not (significantly) change after being adjusted for particulate matter or black carbon. It is interesting to note that the effects of particulate matter itself are reduced quite strongly by adjusting for nitrogen dioxide concentrations. Other recent studies have also shown that the associations between increased short-term exposure to nitrogen dioxide and the various health effects are independent of the volume of particulate matter.[13]

3.3 Long-term exposure to nitrogen dioxide

Adverse effects on the respiratory tract and lungs

The EPA concluded in 2016 that a causal relationship is likely to exist between long-term exposure to nitrogen dioxide and an increased incidence of asthma under children.[2] The EPA based this conclusion on the consistency of the findings from several studies, which indicates that nitrogen dioxide, in and of itself, has an effect on the development of asthma.[14-16]

Adverse effects on the cardiovascular system and diabetes

In 2016, based on large-scale prospective cohort studies, the EPA found some indications of relationships between long-term exposure to nitrogen dioxide and adverse effects on the cardiovascular system and diabetes.[17-21] However, due to inconsistencies between the results of the studies, the EPA concluded that the weight of evidence for a causal relationship with these health effects was still insufficient.[2]

Missing studies

The EPA report did not include two relevant studies of long-term exposure to nitrogen dioxide. In the first place, a 2014 meta-analysis was not included of more than ten cohort studies into the effects on cause-specific mortality from respiratory and cardiovascular diseases as well as on all-cause mortality.[22] This meta-analysis shows that the effects of nitrogen

dioxide on mortality, expressed per interquartile range, are of the same order of magnitude as the effects of particulate matter. A number of the underlying studies reported that the effects found for nitrogen dioxide do not (significantly) change after being adjusted for particulate matter or black carbon or traffic indicators. Secondly, a 2015 cohort study was not included that shows a strong association with mortality from cardiovascular diseases, non-malignant chronic lung diseases, and lung cancer as well as with all-cause mortality; this association was practically independent of particulate matter and ozone.[23]

3.4 Concentration-effect relationships for nitrogen dioxide

In 2013, the WHO published the report *Health risks of air pollution in Europe (HRAPIE)*, which was summarised in 2015 in an article by Heroux et al.[6,7] Based on the information available until 2013, the WHO summarised the concentration-effect relationships between particulate matter, nitrogen dioxide, and ozone on the one hand and the various short-term and long-term health effects and increased risk of premature death on the other. In the concentration-effect relationship, the dose is compared to the relative risk, which is the extra risk that adverse health effects will occur for each additional increment of 10 micrograms/m^3. Tables 8 and 9 present the relative risks associated with nitrogen dioxide only for causal relationships that are considered to be ‘likely’ or ‘demonstrated (proven)’.

Table 8. Concentration-effect relationships for nitrogen dioxide (NO2), *long-term effects*[6]

Indicator of exposure	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
Annual average	Prevalence of respiratory symptoms in children with asthma (5 to 14 years old)	1.021 (0.990-1.060) per 1 microgram/m^3 increase in annual average concentration of nitrogen dioxide
Annual average > 20 micrograms/m^3 (~ 10.6 ppb)	Mortality, over the age of 30	1.055 (1.031-1.080)

[a] Relative risk with 95% confidence interval.

Table 9. Concentration-effect relationships for nitrogen dioxide (NO2), *short-term effects*[6]

Indicator of exposure	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
1-hour maximum	Mortality, all ages	1.003 (1.002-1.004)
1-hour maximum	Hospital admissions for diseases of the respiratory tract, all ages	1.002 (0.999-1.004)
24-hour maximum	Hospital admissions for diseases of the respiratory tract, all ages	1.018 (1.011-1.025)

[a] Relative risk with 95% confidence interval.

3.5 High-risk groups for exposure to nitrogen dioxide

Table 10. High-risk groups for health effects from (short-term) exposure to nitrogen dioxide[2]

Risk factors and risk groups studied	Rationale for conclusion that there is ‘sufficient evidence’
Asthma patients	Consistent indications of an increased sensitivity to a worsening of respiratory symptoms, based in part on an increased airway reactivity in controlled human exposure studies.
Children	Consistent indications of an increased sensitivity to a worsening of respiratory symptoms, based in part on an increase in hospital admissions
Older adults	Consistent indications of an increased sensitivity to a worsening of respiratory symptoms, based in part on an increase in hospital admissions

04 health effects of ozone

4.1 Weight of evidence for health effects of ozone

Table 11. Weight of evidence for a causal relationship between *short-term* O_3 exposure and health effects (EPA, 2013)[3]

Health effects	Weight of evidence for causal relationship
Adverse effects on the respiratory tract and lungs, including: • temporary decrease in pulmonary function • increased bronchial reactivity • increase in inflammatory responses of respiratory tract and lungs • increase in respiratory symptoms and use of medication in children with asthma • increased susceptibility to infections of the respiratory tract and lungs • increased allergic reactions • increase in hospital admissions for respiratory and lung diseases • increased mortality from respiratory and lung diseases	Demonstrated
Adverse effects on cardiovascular system, in particular increased mortality from cardiovascular diseases	Likely
All-cause mortality, in particular also mortality from other diseases than those of the respiratory tract, lungs, and cardiovascular system	Likely

Table 12. Weight of evidence for a causal relationship between *long-term* O_3 exposure and health effects (EPA, 2013)[3]

Health effects	Weight of evidence for causal relationship
Adverse effects on the respiratory tract and lungs, including: • increased incidence of asthma under children • increase in hospital admissions for asthma under children	Likely

4.2 Short-term (peak) exposure to ozone

Adverse effects on the respiratory tract and lungs

Human and toxicological experiments show damage to tissues of the respiratory tract and lungs, thereby providing corroborating evidence that the relationships found in epidemiological studies between short-term exposure to ozone and effects on the respiratory tract and lungs are really of a causal nature.[3]

Mortality

In addition, the EPA concludes that a causal relationship is likely to exist between short-term exposure and mortality. This refers to peak concentrations during periods lasting from 1 to 8 hours.

4.3 Long-term exposure to ozone

Long-term exposure to ozone is defined as exposure during an average of one or more summer seasons or years, whereby the average values are often calculated on the basis of the 1-hour or 8-hour peak concentrations during the days of the year or season.

Adverse effects on the respiratory tract and lungs

The EPA concludes that long-term exposure to ozone is also very likely to cause adverse effects on the respiratory tract and lungs.[3] In particular, this

involves an increase in the number of new cases of asthma in children and of hospital admissions of children with asthma.

4.4 Concentration-effect relationships for ozone

In 2013, the WHO published the report *Health risks of air pollution in Europe (HRAPIE)*, which was summarised in 2015 in an article by Heroux et al.[6,7] Based on the information available until 2013, the WHO summarised the concentration-effect relationships between particulate matter, nitrogen dioxide, and ozone on the one hand and the various short-term and long-term health effects and increased mortality risk on the other. In the concentration-effect relationship, concentration is compared to relative risk. In the case at hand, it informs us about the extra risk that adverse health effects will occur for each additional increment of 10 microgram/m^3. Tables 13 and 14 present the relative risks associated with ozone only for causal relationships that are considered to be 'likely' or 'demonstrated'.

Table 13. Concentration-effect relationships for ozone (O_3): *long-term effects*.[6,7]

Indicator of exposure to ozone	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
Summer (April-September), average of 8-hour maximum values insofar as >35 ppb	Mortality from respiratory diseases, older than 30 years	1,014 (1,005-1,024)

[a] Relative risk with 95% confidence interval.

Table 14. Concentration-effect relationships for ozone (O_3): *short-term effects*.[6,7]

Indicator of exposure to ozone	Health indicator	RR (95% CI)[a] per 10 micrograms/m^3
8-hour maximum >35 ppb	All-cause mortality	1,003 (1,001-1,004)
8-hour maximum >10 ppb	All-cause mortality	1,003 (1,001-1,004)
8-hour maximum >35 ppb	Cardiovascular diseases and mortality from respiratory diseases	Cardiovascular system: 1.005 (1.001-1.009); Respiratory tract: 1,003 (0,999-1,007)
8-hour maximum >10 ppb	Cardiovascular diseases and mortality from respiratory diseases	
8-hour maximum >35 ppb	Cardiovascular diseases and hospital admissions for respiratory diseases >65 years	Cardiovascular system: 1.009 (1.005-1.013); Respiratory tract: 1,004 (1,001-1,008)
8-hour maximum >10 ppb	Cardiovascular diseases and hospital admissions for respiratory diseases >65 years	

[a] Relative risk% confidence interval.

4.5 High-risk groups for exposure to ozone

Table 15. High-risk groups for health effects from (short-term) exposure to ozone[3]

Risk factors and risk groups studied	Rationale for conclusion that there is 'sufficient evidence'
Genetic predisposition	Various gene variants, such as glutathione S-transferase polymorphisms, have been identified that increase the risk of respiratory symptoms
Asthma patients	Asthma patients show an increase in respiratory symptoms
Children	Children are exposed to higher concentrations and are admitted to hospital more frequently for respiratory symptoms
Older adults	Older adults have an increased mortality risk
Reduced nutritional status	Persons with a reduced intake of vitamins C and E suffer from respiratory symptoms more frequently
Outdoor activities	Persons who are active outdoors are exposed to higher concentrations and suffer from respiratory symptoms more frequently

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