TRAINING FOR THE HEALTH SECTOR

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OUTDOOR AIR POLLUTION

Children's Health and the Environment

WHO Training Package for the Health Sector

World Health Organization

www.who.int/ceh

July 2008 version

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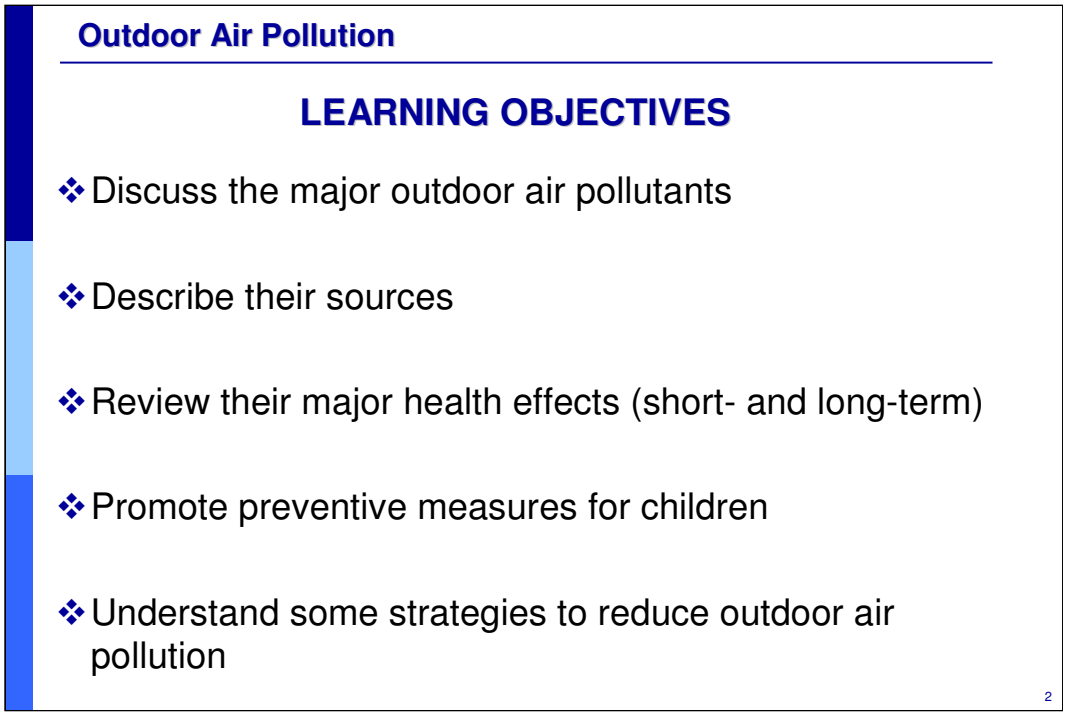
<<NOTE TO USER: Please add details of the date, time, place and sponsorship of the meeting for which you are using this presentation in the space indicated.>>

<<NOTE TO USER: This is a large set of slides from which the presenter should select the most relevant ones to use in a specific presentation. These slides cover many facets of the problem. A number of slides refer to the specific issues related to indoor air pollution in developing countries, as it represents a major determinant of the burden of disease in children. Present only those slides that apply most directly to the local situation in the region.>>



There are four objectives for this training module. At the end of the presentation, the individual will be able to:

1. List the major outdoor air pollutants
2. Describe their sources
3. Describe their major health effects
4. Promote preventive measures for children.
5. List some strategies to reduce outdoor air pollution.

It is very important to recognize that air pollution is variable and that each community has unique problems based on its geography, climate, industries, traffic, and a variety of other factors.

Outdoor Air Pollution

"As soon as I had escaped the heavy air of Rome and the stench of its smoky chimneys, which when stirred poured forth whatever pestilent vapours and soot they held enclosed, I felt a change in my disposition."

Roman philosopher Seneca, AD 61

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Introduce the topic of air pollution by describing its history.

<<READ SLIDE.>>

Air pollution first received recognition as an urban problem in England in the ninth century, when coal was discovered and complaints about foul air began to be heard. The possible menace to human health was recognized in the 17th century by John Evelyn, who dared to ascribe chronic respiratory ailments to the inhalation of coal smoke.

Ref:

Miller GT, Living in the Environment: An Introduction to Environmental Science. Wadsworth Publishers, 1998.

Outdoor Air Pollution

ACUTE HEALTH EFFECTS OF AIR POLLUTION

NOAA 4

Begin by giving a little of the history of outdoor air pollution.

These classic episodes of severe air pollution are important because they caused so many deaths that they served as a "wake up call" to the public and to policy-makers which made them realize that air pollution was more than just a nuisance. They led to the first major legislation designed to reduce air pollution. After these incidents, there was little doubt that high levels of air pollution were associated with an increase in premature deaths.

During the London Smog of 1952, the smog was so thick that road, rail and air transport were brought almost to a standstill and a performance at the Sadler's Wells Theatre had to be suspended when fog in the auditorium made conditions intolerable for the audience and the performers. There was a cattle show going on at the time in Smithfield, and the press reported that the cattle were asphyxiated. The fog was so thick that in many parts of London it was impossible for pedestrians to find their way at night, even in familiar districts. It is said that people could not even see their own feet. This kind of dense fog in London came to be known as a "pea souper". It was very different from the clean white fog of the countryside because it contained noxious emissions from factory chimneys which had an unpleasant odour and was a dirty yellow or brown colour.

It is not known how many people died as a direct result of the fog. Many who died already suffered from chronic respiratory or cardiovascular diseases. Without the fog, they might not have died when they did.

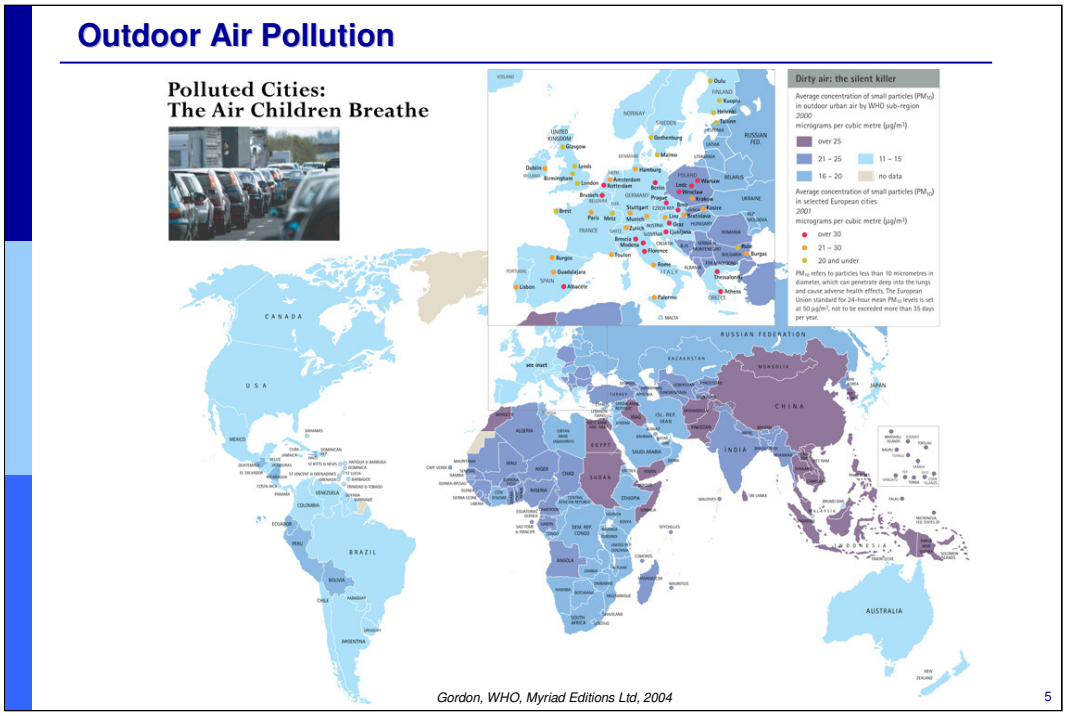
In England, the Clean Air Act of 1956 banned emissions of black smoke and decreed that the residents of urban areas and operators of factories must convert to smokeless fuels.

Ref:

•Ware JH et al. Assessment of the health effects of atmospheric sulfur oxides and particulate matter: evidence from observational studies. *Environ Health Perspect, 1981, 41:255.*

Steadily rising energy costs have increased the need for reliable information on the health effects of atmospheric sulfur oxides and particulate matter. Because ethical and practical considerations limit studies of this question under controlled conditions, observational studies provide an important part of the relevant information. This paper examines the currently available epidemiological evidence from population studies of the health effects of these pollutants. Nonexperimental studies also have important limitations, including the inability to measure accurately the exposure burden of free-living individuals, and the potential for serious confounding by other factors affecting health. We begin with a discussion of some of these methodological issues. The evidence is then reviewed, first in association with fluctuations in 24-hr mean concentration of sulfur oxides and particulate matter, and then in association with differences in mean annual concentration. In the last section, this evidence is summarized and used to approximate the exposure–response relationship linking pollutant concentrations with mortality and morbidity levels.

Picture: NOAA, Valley fog and pollution, Pennsylvania, at: www.photolib.noaa.gov/historic/nws/wea02160.htm

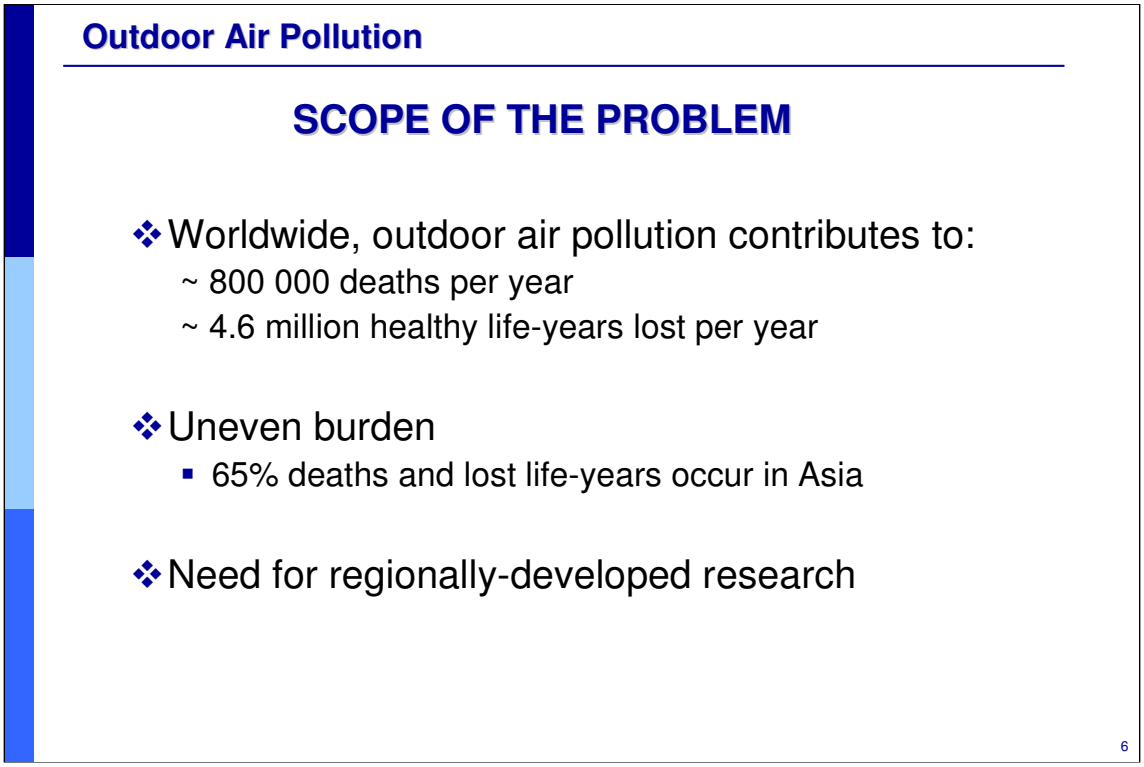


Power plants, factories and vehicles spew out harmful gases and small particles that can penetrate deep into children's lungs. In strong sunlight, oxides of nitrogen from vehicle exhaust fumes form ozone at ground level, which can trigger asthma attacks. Air pollution does not respect national borders. Heavy metals and persistent organic pollutants are carried by winds, contaminating water and soil far from their origin. In the late 1990s, forest fires, mainly in Indonesia, caused a haze of smoke to hang for months over neighbouring south-east Asian countries. Schools and kindergartens were forced to close, while local hospitals reported large numbers of haze-related illnesses in young children. The Great London Smog of 1952 focused the world's attention on the problem of air pollution, and since then there has been a marked improvement in air quality in developed countries. Nevertheless, every year outdoor air pollution is responsible for the death of hundreds of children in Europe, and of more than 24 000 globally.

Industrial growth and rapid urbanization aggravate the problem, with the pressure felt most acutely in the megacities of the developing world. Use of cleaner fuels and technologies, refined motor engines, and public transport are crucial in ensuring that children breathe clean air.

Ref:

•Gordon B et al. *Inheriting the world, the Atlas on Children's Health and the Environment.* World Health Organization, Myriad Editions Ltd, 2004.



Briefly describe the scope of the problem.

The burden of air pollution is not equally distributed: approximately 65% of the deaths and lost life-years occur in the developing countries of Asia.

One problem is that these estimates of the health impact of outdoor air pollution are based largely on the results of research conducted in Europe and North America that have been extrapolated to developing countries. Such extrapolation raises considerable uncertainties because developing countries differ from Europe and North American in the nature of their air pollution, the conditions and magnitude of exposures to that pollution, and the health status of the population. Thus, conducting and evaluating epidemiological studies in developing countries is a priority.

Refs:

•Suk W et al. Environmental threats to children's health in Southeast Asia and the Western Pacific. *Environmental Health Perspectives,* 2003, 111:1340.

•Briggs D. Environmental pollution and the global burden of disease. *Br Med Bull,* 2003, 68:1.

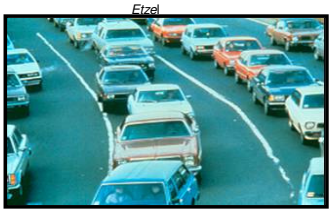
•Molina MJ et al. Megacities and atmospheric pollution. *J Air Waste Manag Assoc,* 2004, 54:644.

Outdoor Air Pollution

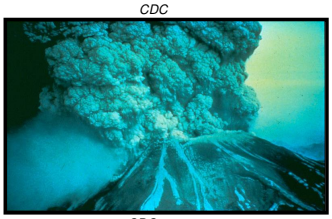
MAIN SOURCES

- Combustion of fossil fuels
  - Domestic heating
  - Power generation
  - Motor vehicles
- Industrial processes
- Agricultural processes
- Waste incineration
- Natural processes
  - Thunderstorms
  - Volcanoes

Etzel



CDC



CDC

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<<NOTE TO USER: This is an opportunity to mention that the source of outdoor air pollution depends on where you are. This slide should be customized to meet the needs of the community in which the talk is being presented.>>

Sources of outdoor air pollution may be quite different in rural and urban settings.

Pictures:

TOP: This is a picture of one source of air pollution. In many parts of the world, coal is still used for heating homes. This photograph is from Poland, where soft (brown) coal is used for heating homes. This man is shovelling coal, which has been delivered to the pavement next to his home, into his basement. Burning of soft coal results in more sulfur dioxide pollution in the air than burning of hard coal. *Photo: Dr. Ruth Etzel.*

MIDDLE: The major source of air pollution in industrialized countries is car exhaust. *Photo: CDC.*

BOTTOM: Another source of outdoor air pollution is illustrated in the bottom photo. This slide shows a volcanic eruption of Mount St. Helens in Washington. Disasters like this can result in air pollution emergencies. For example, the eruption of Mt. Pinatubo in the Philippines also resulted in serious air pollution. Along with the massive quantities of particulate matter, sulfur dioxide and nitrogen dioxide, other air pollutants are produced during volcanic eruptions. Photo: CDC.

Outdoor Air Pollution

AIR POLLUTION SOURCES

Outdoor air quality is affected by:

- Industrial or agricultural activities
- Treatment of industrial effluents and domestic residues
- Traffic
- Solid waste management
- Cottage industries
- Chemical incidents and spills

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Outdoor pollution primarily results from the combustion of fossil fuels by industrial plants and vehicles. This releases carbon monoxide, sulfur dioxide, particulate matter, nitrogen oxides, hydrocarbons and other pollutants. The characteristics of emissions and solid waste disposal may vary for each specific industry (e.g. smelting, paper production, refining and others).

<<NOTE TO USER: This is an opportunity to mention that the source of outdoor air pollution depends on where you are. This slide and the image should be customized to meet the needs of the community and region in which the talk is being presented.>>

Picture: WHO, J. Vizcarra. Environmental Air Pollution

Outdoor Air Pollution

INDOOR AIR POLLUTION ALSO AFFECTS OUTDOOR AIR

Nigel Bruce/ITDG

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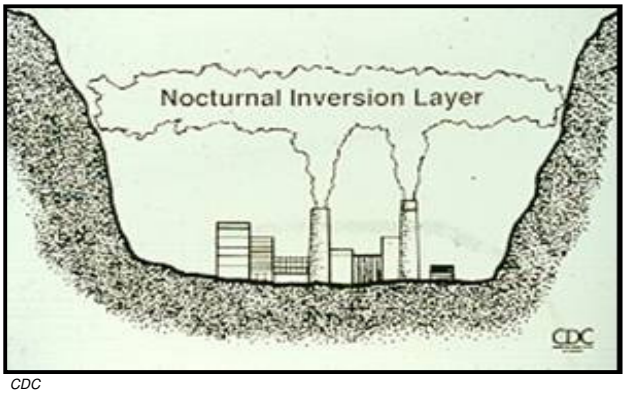
Indoor smoke polluting the ambient air in a small village in Nepal.

Picture: Courtesy of Nigel Bruce/ITDG. Used with permission.

OUTDOOR AIR POLLUTION CONSIDERATIONS



CDC

- Climate
  - Temperature
  - Prevailing winds
  - Seasonal changes
- Topography
  - Prevailing winds
  - Hills and valleys
  - Dominant vegetation
- Cities and surfaces

Air pollution levels are tightly linked to climate and topography. Air pollution episodes can be particularly troublesome if the affected city is located in a valley surrounded by mountains (this was the case in the Meuse Valley in Belgium and is the case in Mexico City, Mexico).

Surfaces such as roads (gravel, dirt, asphalt) can generate air pollution when cars drive on them.

"Nocturnal inversion layer: in meteorology, the atmospheric layer in which the usual temperature gradient — warm air below cold air — is reversed, preventing the mixing of warm and cold air as the warmer air rises. This traps dangerous concentrations of pollutants in the cool air below, sometimes causing dense smog over urban areas" (Encyclopedia Britannica Online, 2004).

Ref:

•www.britannica.com/ebi/article?tocId=9327200

Outdoor Air Pollution

HOW CHILDREN ARE DIFFERENT

Short stature
Breathe closer to the ground

Ongoing lung development

Increased air intake

WHO

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Children may be more vulnerable to the effects of air pollution than adults. Children's lung development is not complete at birth. Lung development proceeds through proliferation of pulmonary alveoli and capillaries until the age of 2 years. Thereafter, the lungs grow through alveolar expansion until 5–8 years of age. Lungs do not complete their growth until full adult stature is reached in adolescence.

Refs:

•*American Academy of Pediatrics Committee on Environmental Health. Developmental toxicity: Special considerations based on age and developmental stage. In: Etzel RA, ed. Pediatric Environmental Health. 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003.*

•*Selevan SG et al. Identifying critical windows of exposure for children's health. Environmental Health Perspectives, 2000, 108:451.*

•*Children's Health and the Environment – A global perspective. A resource guide for the health sector, WHO, 2005.*

Picture: WHO, A. Waak. Haiti.

CHILDREN'S UNIQUE VULNERABILITY

- Greater exposures because they spend more time outside
- Inhale more pollutants per kilogram of body weight than do adults
- Because airways are narrower, irritation can result in proportionately greater airway obstruction

Infants and young children have a higher resting metabolic rate and rate of oxygen consumption per unit body weight than adults because they have a larger surface area per unit body weight and because they are growing rapidly. Therefore, their exposure to any air pollutant may be greater.

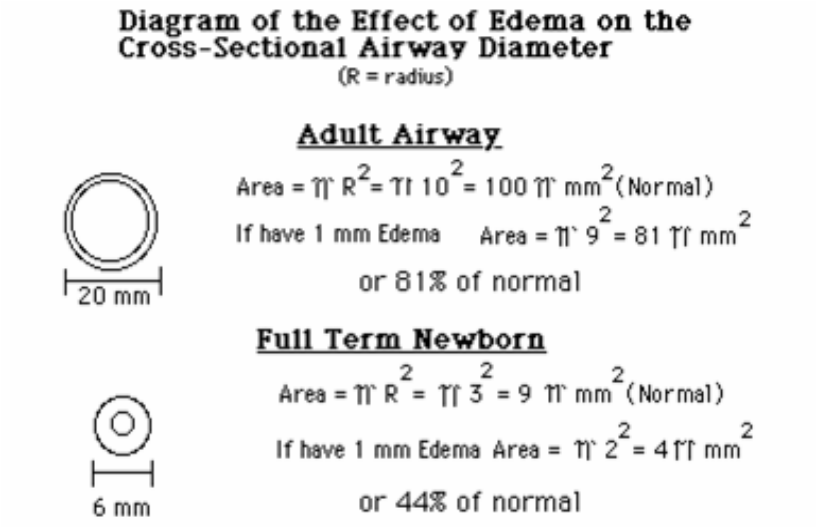
In addition to an increased need for oxygen relative to their size, children have narrower airways than do adults. Thus, irritation caused by air pollution that would produce only a slight response in an adult can result in potentially significant obstruction in the airways of a young child.

Ref:

•Moya J et al. Children's behavior and physiology and how it affects exposure to environmental contaminants. *Pediatrics,* 2004, 113:996.

Outdoor Air Pollution

SMALLER AIRWAYS MORE VULNERABLE



www.vh.org/pediatric/provider/pediatrics/ElectricAirway/Diagrams/AirwayDIaneterEdema.jpg

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The effect of oedema on the adult airway is much less dramatic that it is on the newborn's airway. One millimetre of oedema reduces the diameter of the adult airway by about 19% while it reduces the diameter of the infant airway by 56%.

Compared to that of adults, the peripheral airway (bronchioles) is both relatively and absolutely smaller in infancy allowing intralumenal debris to cause proportionately greater obstruction. In addition, infants have relatively larger mucous glands, with a concomitant increase in secretions. They also have the potential for increased oedema because their airway mucosa is less tightly adherent. Lastly, there are fewer interalveolar pores (Kohn's pores) in the infant, producing a negative effect on collateral ventilation and increasing the likelihood of hyperinflation or atelectasis.

The resting minute ventilation normalized for body weight in a newborn infant (400 cc/min/kg) is more than double that of an adult (150 cc/min/kg).

Ref:

•Bar-on ME et al. Bronchiolitis. *Prim Care,* 1996, 23:805.

Picture:
www.vh.org/pediatric/provider/pediatrics/ElectricAirway/Diagrams/AirwayDIaneterEdema.jpg - Copyright protected material used with permission of the authors: Drs. Michael and Donna D'Alessandro - and the University of Iowa's Virtual Hospital, www.vh.org

CHILDREN'S OUTDOOR EXPOSURE

Time spent outdoors is influenced by:

- Geographical region:
  - Seasons and temperature
  - Urban or rural area
- Economic development of the region
- Social and cultural aspects

<<NOTE TO USER: If you have local information on time spent outdoors, please insert here.>>

Outdoor Air Pollution

MAJOR OUTDOOR AIR POLLUTANTS

- Particulate matter
- Ozone
- Nitrogen oxides
- Carbon monoxide
- Sulfur dioxide

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These are some major outdoor air pollutants that can have an effect on child health. Listed here are five that will be briefly discussed: particulate matter, ozone, nitrogen oxides, carbon monoxide and sulfur dioxide. In some countries, these five pollutants are routinely measured (together with lead), and governments sometimes set standards for them. For example, in the USA there are National Ambient Air Quality Standards (NAAQS) for these pollutants.

Current levels of air pollution have chronic, adverse effects on lung development in children between the ages of 10 and 18 years, leading to clinically significant deficits in attained FEV_1 as children reach adulthood.

FEV_1 = forced expiratory volume in 1 second

Refs:

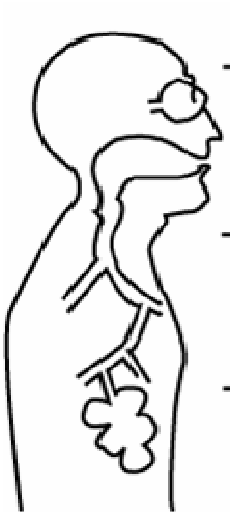
•Gauderman WJ et al. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med,* 2004, 351:1057-67.

Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown. Methods: *In this prospective study, we recruited 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The rate of attrition was approximately 10 per cent per year. The communities represented a wide range of ambient exposures to ozone, acid vapour, nitrogen dioxide and particulate matter. Linear regression was used to examine the relationship of air pollution to the forced expiratory volume in one second (FEV(1)) and other spirometric measures.* Results: *Over the eight-year period, deficits in the growth of FEV(1) were associated with exposure to nitrogen dioxide (P = 0.005), acid vapor (P = 0.004), particulate matter with an aerodynamic diameter of less than 2.5 micron (PM(2.5)) (P = 0.04), and elemental carbon (P = 0.007), even after adjustment for several potential confounders and effect modifiers. Associations were also observed for other spirometric measures. Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV(1) attained at the age of 18 years. For example, the estimated proportion of 18-year-old subjects with a low FEV(1) (defined as a ratio of observed to expected FEV(1) of less than 80 per cent) was 4.9 times as great at the highest level of exposure to PM(2.5) as at the lowest level of exposure (7.9 per cent vs. 1.6 per cent, P = 0.002).* Conclusions: *The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV(1) as children reach adulthood.*

Pictures: WHO, P. Virot. Ethiopia, 2002 (above) / C. Gaggero. Environmental air pollution, Americas (below)

Outdoor Air Pollution

DEPOSITION OF POLLUTANTS IN RESPIRATORY TRACT



CDC

Water Solubility	Initial Level of Impact	Compounds
High	Eyes Nose Pharynx Larynx	Aldehydes Ammonia Chlorine Sulfur dioxide
Medium	Trachea Bronchi	Ozone
Low	Bronchioles Alveoli	Nitrogen dioxide Phosgene

Respirable particles and gases affect different parts of the respiratory tree depending upon their inherent characteristics. For gases, relative solubility is important. For particles, size is important.

This slide shows the upper, middle and lower respiratory tract. Note that sulfur dioxide, because it is highly water soluble, initially affects the upper airway, whereas ozone, which has medium solubility, initially affects the middle airways, and nitrogen dioxide, which has low solubility, initially affects the lower airways.

Outdoor Air Pollution

PARTICULATE MATTER

- Complex mixture of particles that can be solid, liquid or both
- Vary in size, composition, and origin

- Sources:
  - Power plants and industry
  - Motor vehicles, domestic coal burning
  - Natural sources (volcanoes, dust storms)
  - Small particles form surface for acid aerosol formation

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Primary particles originating from combustion sources usually consist of a carbonaceous core with chemicals (such as sulfates, metals and polycyclic aromatic hydrocarbons) adsorbed to their surfaces. In addition, secondary particles are formed by chemical reactions in the atmosphere of primary particles with gases (such as nitric oxides, ozone and sulfur oxides, which are strong oxidants), leading to formation of nitrates and ammonia.

The specific composition and size distribution of particulate matter (PM) varies by region, time of year, time of day, weather conditions and other factors. For example, sulfates dominate the $PM_{2.5}$ mixture in the eastern United States, whereas nitrates are more abundant in the western United States.

Particulate matter goes by many different names. It may be referred to as total suspended particulates, black smoke, breathable particulates or thoracic particulates. Recently, there has been an effort to use more objective features such as the particulate diameter: particles with a diameter less than 10 micrometres are named PM_{10}; particles with a diameter less than 2.5 micometres are called $PM_{2.5}$ (or fine particulates) and particles with a diameter less than 0.1 micrometre are called $PM_{0.1}$ (or ultrafine particulates).

PM_{10} particles with a diameter less than 10 micrometres

$PM_{2.5}$ particles with a diameter less than 2.5 micometres (fine particulates)

$PM_{0.1}$ particles with a diameter less than 0.1 micrometre (ultrafine particulates)

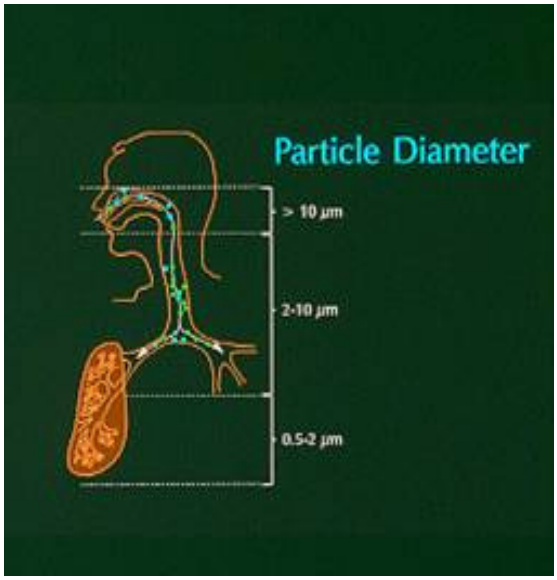
Ref:

WHO Fact Sheet: www.euro.who.int/document/mediacentre/fs0405e.pdf

Outdoor Air Pollution

SIZE MATTERS

- Coarse particles (2.5–10 micrometres) deposited in the upper respiratory tract and large airways
- Fine particles (< 2.5 micrometres) may reach terminal bronchioles and alveoli



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Particle size is the most important factor in determining where particles are deposited in the lung.

Compared with large particles, fine particles can remain suspended in the atmosphere for longer periods and be transported over longer distances.

Some studies suggest that fine particles have stronger respiratory effects in children than large particles.

This diagram shows that particles greater than 10 micrometres rarely make it past the upper airways, whereas fine particles smaller than 2 micrometres can make it as far as the alveoli.

PM_{10} particles with a diameter less than 10 micrometres (course particles)

$PM_{2.5}$ particles with a diameter less than 2.5 micometres (fine particulates)

$PM_{0.1}$ particles with a diameter less than 0.1 micrometre (ultrafine particulates)

Ref:

•World Health Organization. *Air quality guidelines.* Geneva, World Health Organization, Department of Protection of the Human Environment, 1999.

PARTICULATE MATTER: HEALTH EFFECTS

Fine particles deposit in distal airways:

- Increased acute respiratory morbidity (pneumonia, asthma)
- Increased mortality (from all causes)
- Decreased lung growth and function

Many studies have noted an association between particulate air pollution and mortality among people of all ages.

Meta-analysis of particulate health effects (for 10 micrograms/m^3):

Total mortality rate: 1%

Cardiovascular mortality rate: 1.4%

Respiratory mortality rate: 3.4%

Respiratory-related hospitalization: 0.8%

Asthma-related hospitalizations: 1.9%

Asthma-related emergency visits: 3.4%

Asthma exacerbations: 3%

These increases are for a 10 microgram/m^3 increase in PM10, which is a relatively small difference in exposure (*Dickey, 2000).*

Some data suggest that exposure to particulate matter may be associated with decreased birth weights. There are data from Brazil, Central and Eastern Europe and China to support this association.

Refs:

•Bobak M et al. Air pollution and infant mortality in the Czech Republic, 1986-88. Lancet, 1992, 340:1010.

An ecological study of infant mortality and air pollution was conducted in the Czech Republic. Routinely collected data on infant mortality and air pollution in the period 1986–1988 were analysed for the 46 of the 85 districts in the republic for which both were available. The independent effects of total suspended particulates (TSP-10), sulfur dioxide (SO2), and oxides of nitrogen (NOx) adjusted for district socioeconomic characteristics, such as income, car ownership, and abortion rate, were estimated by logistic regression. We found weak positive associations between neonatal mortality and quintile of TSP-10 and SO2. Stronger adjusted effects were seen for postneonatal mortality, with a consistent increase in risk from the lowest to the highest TSP-10 quintile ($p < 0.001$). Weaker and less consistent evidence of a positive association with NOx ($p = 0.061$) was observed. The strongest effects were seen for postneonatal respiratory mortality, which increased consistently from lowest to highest TSP-10 quintile ($p = 0.013$). There was also a suggestion of a positive association with SO2 ($p = 0.062$). The highest to lowest quintile risk ratios for postneonatal respiratory mortality were 2.41 (95% CI, 1.10–5.28) for TSP-10, 3.91 (0.90–16.9) for SO2, and 1.20 (0.37–3.91) NOx. The specificity of the association between air pollution quintile (especially TSP-10) and postneonatal respiratory mortality is consistent with the known effects of air pollution on respiratory disease morbidity in children. These ecological associations require confirmation in an individually based study.

•Dickey JH. Part VII. Air pollution: overview of sources and health effects. *Dis Mon,* 2000, 46:566.

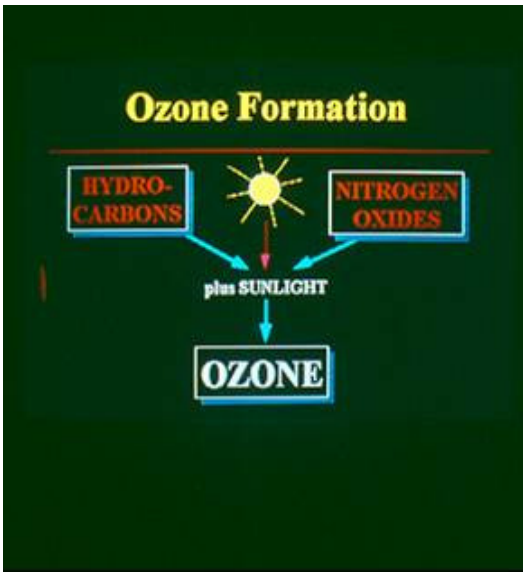
•Ha EH et al. Infant susceptibility of mortality to air pollution in Seoul, South Korea. *Pediatrics,* 2003, 111:284.

•Kaiser R et al. Air pollution attributable postneonatal infant mortality in U.S. metropolitan areas: A risk assessment study. *Environmental Health,* 2004, 3:4.

Outdoor Air Pollution

OZONE (O_3): SOURCES

- **"Secondary pollutant"**
- **Formed by photochemical reaction of VOCs, NO_2 + O_2**
  - Peaks late afternoon
  - Maximum in hot, stagnant air



Ozone is an important pollutant in many parts of the industrialized world. It is rarely measured in developing countries, so there is less information about its role in those countries.

The key distinction to be understood is that:

"Good" ozone occurs in the upper atmosphere. Ozone is a naturally occurring form of oxygen that provides a protective layer shielding the Earth from harmful ultraviolet radiation.

"Bad" ozone occurs in the lower atmosphere. Ozone is the major component of urban smog and a potent respiratory irritant that can also synergistically enhance a child's reaction to other air pollutants and pollen. Ozone is a secondary air pollutant formed in the atmosphere from a chemical reaction between hydrocarbons and nitrogen oxides in the presence of heat and sunlight. One important fact about ozone is that it requires sunlight for its formation, so it tends to peak on hot summer afternoons from 3 to 5 pm. This may be useful to guide scheduling of vigorous outside activities in the early morning or after dark. It is useful to know the "air quality index" if available — many countries provide this in newspapers and on the radio and television.

The primary sources of these precursor compounds include motor vehicle exhaust and power plants, although natural sources (trees) can also contribute.

VOCs: volatile organic compounds

NO_2: nitrogen dioxide

O_2: oxygen

OZONE HEALTH EFFECTS:
"LIKE A SUNBURN OF THE LUNGS"

- **Powerful oxidant**
- **Lung irritation and inflammation**
- **Impaired pulmonary function**
- **Eye, nose and throat irritation**
- **Mechanisms of toxicity:**
  - **Oxidant damage**
  - **Increased neutrophils and inflammatory cytokines (in bronchoalveolar lavage)**

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Patients may better understand the effects of ozone if clinicians describe it in terms with which they are familiar, e.g. it is "like a sunburn of the lungs".

After exposure to ozone, people with asthma have increased bronchial reactivity to subsequent allergens.

Most of the acute respiratory effects such as cough and shortness of breath are thought to be reversible.

Recent studies show that long-term exposure to ozone is associated with decrements in lung function that persist into the second decade.

Ref:

•Tager I et al. Air pollution and lung function growth: is it ozone? *Am J Respir Crit Care Med,* 1999, 160:387.

Outdoor Air Pollution

OZONE (O_3)

- Chronic exposure associated with:
  - **chronic lung disease**
  - **mild pulmonary fibrosis**
  - **small airway obstruction**
  - **Asthma exacerbation and clinic visits**
- Lifetime ambient ozone exposure associated with small-airway physiology
- Association between living (for 4 or more years) in areas with high levels of ozone, decreased FEV_1 and $FEF_{25-75\%}$ (USA) *(Gauderman, 2000)*

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Chronic exposure to ozone pollution has been associated with:

- *de novo* development of chronic lung disease;
- mild pulmonary fibrosis; and
- modest increases in small airway obstruction.

Lifetime exposure to ambient ozone is negatively associated with lung function measures that reflect small-airway physiology.

There is also an association between living for four or more years in areas of the USA with high levels of ozone and decreased FEV_1 (forced expiratory volume) and $FEF_{25-75\%}$ (forced expiratory flow).

Do these early changes in children and adolescents result in a greater risk of chronic obstructive pulmonary disease in later life?

Factors that may influence long-term health effects include age at exposure, sex, genetic factors, exercise and nutrition.

There is an association of exposure to ozone with chronic phlegm, wheeze (apart from colds), and a higher composite respiratory index. These are considered early indicators for pathological changes that might progress to chronic obstructive pulmonary disease.

Some evidence has linked ozone to chronic lung scarring, especially at the bronchoalveolar junction.

For basic training for ground-level ozone and health effects, see:
www.epa.gov/air/oaqps/eog/ozonehealth/index.html

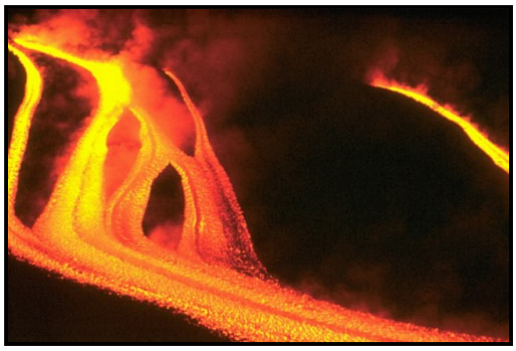
Ref:

•Gauderman WJ et al, Association between Air Pollution and Lung Function Growth in Southern California Children. *Am J Respir Crit Care Med,* 2000, 162:1383-1390.

•eea.europa.eu/maps/ozone

NITROGEN OXIDES (NO_X): SOURCES

- Power plants, industry
- Motor vehicles
- Natural sources (volcanoes, lightning, bacteria)



WHO

Most combustion processes produce nitrogen monoxide (NO) which through oxidation processes results in nitrogen dioxide (NO_2). Nitrogen dioxide combines with oxygen in the presence of sunlight to form ozone.

Pictures: WHO. Papua, New Guinea (left) / JP Revel, Disaster, Cred, Volcano and lava flow (right)

Outdoor Air Pollution

NITROGEN OXIDES (NO_X): HEALTH EFFECTS

- Emphysema in animals
- Decrements in lung function
- Increased airway reactivity
- Increased susceptibility to infection?

24

Research is being conducted to explore the hypothesis that exposure to nitrogen oxides and ozone may increase susceptibility to viral infections.

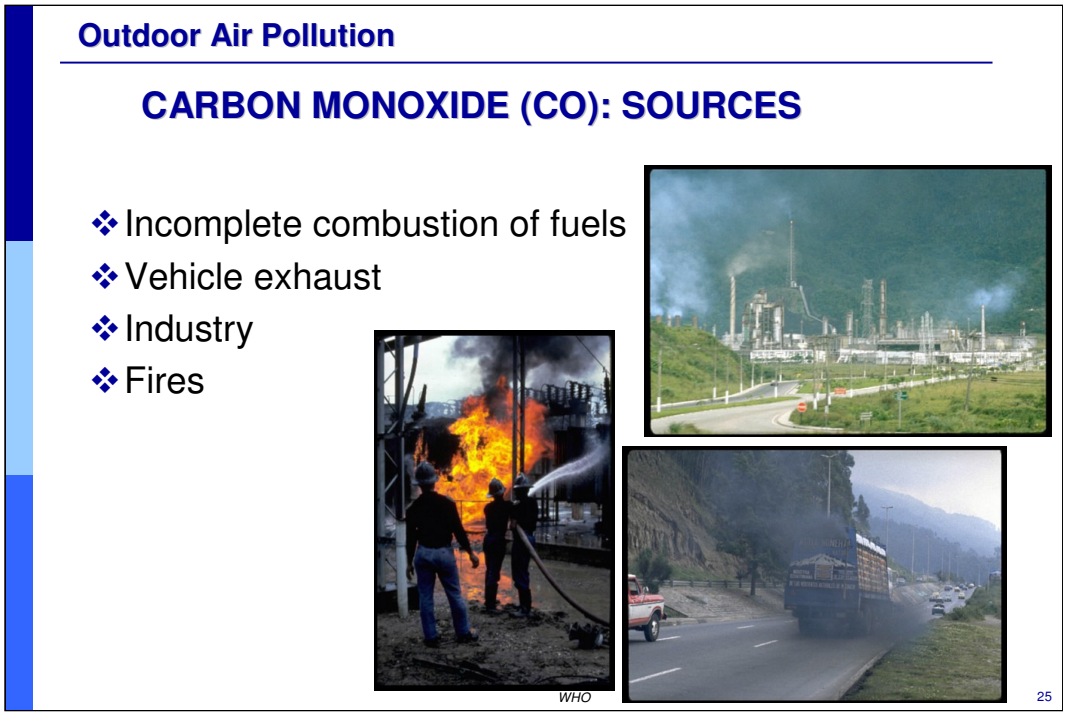
Refs:

•O'Connor GT, Acute respiratory health effects of air pollution on children with asthma in US inner cities. *J Allergy Clin Immunol.* 2008; 121(5):1133-1139.e1

BACKGROUND: Children with asthma in inner-city communities may be particularly vulnerable to adverse effects of air pollution because of their airways disease and exposure to relatively high levels of motor vehicle emissions. OBJECTIVE: To investigate the association between fluctuations in outdoor air pollution and asthma morbidity among inner-city children with asthma. METHODS: We analyzed data from 861 children with persistent asthma in 7 US urban communities who performed 2-week periods of twice-daily pulmonary function testing every 6 months for 2 years. Asthma symptom data were collected every 2 months. Daily pollution measurements were obtained from the Aerometric Information Retrieval System. The relationship of lung function and symptoms to fluctuations in pollutant concentrations was examined by using mixed models. RESULTS: Almost all pollutant concentrations measured were below the National Ambient Air Quality Standards. In single-pollutant models, higher 5-day average concentrations of NO2, sulfur dioxide, and particles smaller than 2.5 microm were associated with significantly lower pulmonary function. Higher pollutant levels were independently associated with reduced lung function in a 3-pollutant model. Higher concentrations of NO2 and particles smaller than 2.5 microm were associated with asthma-related missed school days, and higher NO2 concentrations were associated with asthma symptoms. CONCLUSION: Among inner-city children with asthma, short-term increases in air pollutant concentrations below the National Ambient Air Quality Standards were associated with adverse respiratory health effects. The associations with NO2 suggest that motor vehicle emissions may be causing excess morbidity in this population.

•Chen G et al. Short-term effects of ambient gaseous pollutants and particulate matter on daily mortality in Shanghai, China. *J Occup Health. 2008; 50(1):41-7.*

Identification of the specific pollutants contributing most to the health hazard of the air pollution mixture may have important implications for environmental and social policies. In the current study, we conducted a time-series analysis to examine the specific effects of major air pollutants [particulate matter less than 10 microns in diameter (PM(10)), sulfur dioxide (SO(2)), and nitrogen dioxides (NO(2))] on daily mortality in Shanghai, China, using both single-pollutant and multiple-pollutant models. In the single-pollutant models, PM(10), SO(2), and NO(2) were found to be associated with mortality from both all non-accidental causes and from cardiopulmonary diseases. Unlike some prior studies in North America, we found a significant effect of gaseous pollutants (SO(2) and NO(2)) on daily mortality even after adjustment for PM(10) in the multiple-pollutant models. Our findings, combined with previous Chinese studies showing a consistent, significant effect of gaseous pollutants on mortality, suggest that the role of outdoor exposure to SO(2) and NO(2) should be investigated further in China.



There are many sources of carbon monoxide: motor vehicle exhaust is the most important in urban communities. The amount of carbon monoxide released from a vehicle depends on the vehicle as well as the kind of fuel used. In some countries (including USA and Brazil) fuel additives have been used in areas with elevated carbon monoxide concentrations in the outdoor air. Two of the major fuel additives ("oxygenates") are ethanol and methyl tertiary butyl ether (MTBE).

Pictures: WHO : A Waak, Environmental air pollution, Ecuador above)/ Environmental Air Pollution Americas (below) and Environmental fires, Americas (left)

Ref:

•Miraglia SG. Health, environmental, and economic costs from the use of a stabilized diesel/ethanol mixture in the city of São Paulo, Brazil. *Cad Saude Publica. 2007;23 Suppl 4:S559-69.*

In Greater Metropolitan São Paulo, Brazil, fossil fuel combustion in the transportation system is a major cause of outdoor air pollution. Air quality improvement requires additional policies and technological upgrades in fuels and vehicle engines. The current study thus simulated the environmental and social impacts resulting from the use of a stabilized diesel/ethanol mixture in the bus and truck fleet in Greater Metropolitan São Paulo. The evaluation showed reductions in air pollutants, mainly PM10, which would help avert a number of disease events and deaths, as estimated through dose-response functions of epidemiological studies on respiratory and cardiovascular diseases. Valuation of the impacts using an environmental cost-benefit analysis considered operational installation, job generation, potential carbon credits, and health costs, with an overall positive balance of US$ 2.851 million. Adding the estimated qualitative benefits to the quantitative ones, the project's benefits far outweigh the measured costs. Greater Metropolitan São Paulo would benefit from any form of biodiesel use, producing environmental, health and socioeconomic gains, the three pillars of sustainability.

CARBON MONOXIDE (CO): HEALTH EFFECTS

- Combines with haemoglobin in blood
- Reduces oxygen-carrying capacity
- At higher risk: children with anaemia, heart disease or chronic lung disease, and foetuses

Infants and children have an increased susceptibility to CO toxicity because of their higher metabolic rates. Children with existing pulmonary or haematological illness (such as anaemia) or other conditions that compromise oxygen delivery are also more susceptible to adverse effects of exposure to CO at lower levels than are healthy children. Chronic exposure to low levels of CO causes headaches.

Ref:

•Kind T. Carbon monoxide. Pediatr Rev. 2005; 26(4):150-1.

SULFUR DIOXIDE (SO_2): SOURCES

- Combustion of sulfur-containing coal or oil
- Industrial processes, smelting of sulfur-containing ores
- Natural sources (volcanoes)
- Combines with water to form H_2SO_4 (acid aerosol, acid rain)

Sulfuric acid (H_2SO_4) aerosol is formed in the atmosphere from the oxidation of sulfur dioxide (SO_2) in the presence of moisture. Facilities that either manufacture or use acids can also emit H_2SO_4.

SO_2 contributes to the formation of acid rain.

Ref:

•Acid aerosols explained www.windows.ucar.edu/tour/link=/earth/Atmosphere/aerosol_cloud_nucleation_dimming.html

•Pikhart H et al. Outdoor sulfur dioxide and respiratory symptoms in Czech and Polish school children: a small-area study (SAVIAH). Small-Area Variation in Air Pollution and Health. *Int Arch Occup Environ Health,* 2001, 74:574

•O'Connor GT, Acute respiratory health effects of air pollution on children with asthma in US inner cities. *J Allergy Clin Immunol.* 2008; 121(5):1133-113

SULFUR DIOXIDE (SO_2): HEALTH EFFECTS

- Gas is upper airway irritant
  - Primarily affects nasopharynx and proximal airways
- Acid aerosol particles reach distal airways
  - bronchoconstriction
  - slowing of bronchial mucociliary clearance
  - chronic bronchitis

<<READ SLIDE.>>

Ref:

•Acid aerosols explained
www.windows.ucar.edu/tour/link=/earth/Atmosphere/aerosol_cloud_nucleation_dimming.html

•Pikhart H et al. Outdoor sulfur dioxide and respiratory symptoms in Czech and Polish school children: a small-area study (SAVIAH). Small-Area Variation in Air Pollution and Health. *Int Arch Occup Environ Health,* 2001, 74:574

Air pollution has been linked to respiratory outcomes but controversy persists about its long-term effects. We used a novel technique to estimate the outdoor concentrations of sulfur dioxide (SO_2) at small-area level to study the long-term effects on respiratory symptoms and disease in children. As part of the international SAVIAH study, parents of 8013 children aged 7–10 studied in Prague (Czech Republic) and Poznan (Poland) completed a questionnaire covering respiratory health, demographic and socioeconomic factors and health behaviours (response rate 91%). This report is based on 6959 children with complete data. Outdoor SO_2 was measured by passive samplers at 80 sites in Poznan and 50 sites in Prague during 2-week campaigns. Concentrations of SO_2 at each point (location) in the study areas were estimated from these data by modelling in a geographical information system. The mean of the estimated SO_2 concentrations at children's homes and schools was used as an indicator of exposure to outdoor SO2. The prevalence of respiratory outcomes was similar in both cities. In the pooled data, 12% of children had experienced wheezing/whistling in the past 12 months; 28% had a lifetime prevalence of wheezing/whistling; 14% had a dry cough at night; and 3% had had asthma diagnosed by a doctor. The estimated mean exposure to outdoor SO_2 was 80 (range 44–140) microg/m^3 in Poznan and 84 (66–97) microg/m^3 in Prague. After socioeconomic characteristics and other covariates were controlled for, SO_2 was associated with wheezing/whistling in the past 12 months (adjusted OR per 50 microg/m^3 1.32, 95% CI 1.10–1.57), lifetime prevalence of wheezing/whistling (OR 1.13, 95% CI 0.99–1.30), and lifetime prevalence of asthma diagnosed by a doctor (OR 1.39, 95% CI 1.01–1.92). The association with dry cough at night did not reach statistical significance. In these two Central European cities with relatively high levels of air pollution, small-area based indicators of long-term outdoor winter concentrations of SO_2 were associated with wheezing/whistling and with asthma diagnosed by a doctor.

Outdoor Air Pollution

LONG-TERM EFFECTS OF ACID AEROSOLS

Long-term intermittent exposure to acid aerosols (sulfate and bisulfate) associated with:

-Higher likelihood of reported bronchitis in the past year among 8 to12 year old children

-Decreased lung function

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Bronchitis has been associated with higher levels of exposures to acid aerosols among 8 to 12-year-old children. However, asthma, persistent wheeze, chronic cough and chronic phlegm were not significantly associated with higher levels of acid aerosols among 8 to 12-year-old children.

Long-term exposure to acid aerosols was associated with statistically significant decrements in FVC and FEV_1 among 8 to 12-year-old children.

Refs:

•Dockery DW et al. Health effects of acid aerosols on North American children: respiratory symptoms. *Environ Health Perspect,* 1996, 104:500.

We examined the respiratory health effects of exposure to acidic air pollution among 13 369 white children 8 to 12 years old from 24 communities in the United States and Canada between 1988 and 1991. Each child's parent or guardian completed a questionnaire. Air quality and meteorology were measured in each community for a 1-year period. We used a two-stage logistic regression model to analyse the data, adjusting for the potential confounding effects of sex, history of allergies, parental asthma, parental education, and current smoking in the home. Children living in the community with the highest levels of particle strong acidity were significantly more likely [odds ratio (OR) = 1.66; 95% confidence interval (CI) 1.11–2.48] to report at least one episode of bronchitis in the past year compared to children living in the least-polluted community. Fine particulate sulfate was also associated with higher reporting of bronchitis (OR = 1.65; 95% CI 1.12–2.42). No other respiratory symptoms were significantly higher in association with any of the air pollutants of interest. No sensitive subgroups were identified. Reported bronchitis, but neither asthma, wheeze, cough, nor phlegm, were associated with levels of particle strong acidity for these children living in a nonurban environment.

•Raizenne M et al. Health effects of acid aerosols on North American children: pulmonary function. *Environ Health Perspect,* 1996, 104:506.

We examined the health effects of exposure to acidic air pollution among children living in 24 communities in the United States and Canada. Parents of children between the ages of 8 and 12 completed a self-administered questionnaire and provided consent for their child to perform a standardized forced expiratory maneuver at school in 22 of these communities. Air quality and meteorology were measured in each community for the year preceding the pulmonary function tests. Forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV1.0) measurements of 10,251 white children were examined in a two-stage regression analysis that adjusted for age, sex, height, weight, and sex-height interaction. In this study, a 52 nmol/m3 difference in annual mean particle strong acidity was associated with a 3.5% (95% CI, 2.0-4.9) decrement in adjusted FVC and a 3.1% (95% CI, 1.6-4.6) decrement in adjusted FEV1.0. The FVC decrement was larger, although not significantly different, for children who were lifelong residents of their communities (4.1%, 95% CI, 2.5-5.8). The relative odds for low lung function (that is, measured FVC less than or equal to 85% of predicted), was 2.5 (95% CI, 1.8-3.6) across the range of particle strong acidity exposures. These data suggest that long-term exposure to ambient particle strong acidity may have a deleterious effect on lung growth, development, and function.

Outdoor Air Pollution

SUMMARY MAJOR OUTDOOR POLLUTANTS

Pollutant	Sources	Health Effects
Particulate Matter	Automobile, bus and truck exhaust, fuel burning (wood stoves, fireplaces), industry, construction.	↑ infant respiratory mortality ↓ lung function ↓ lung growth ↑ symptoms in asthmatics
Ozone	Produced when nitrogen oxides (vehicle emissions) and volatile organic compounds (VOC) chemically react under sunlight.	↓ lung growth ↑ asthma exacerbations ↑ all respiratory hospitalization ↑ asthma hospitalization ↑ asthma ED visit ↑ school absence for respiratory illness
Nitrogen dioxide	Results from high temperature fuel combustion and atmospheric reactions.	↑ symptoms in asthmatics ↓ lung growth
Carbon monoxide	Formed when carbon-containing fuel is not burned completely, emitted by motor vehicles more than any other source.	↑ asthma hospitalization ↑ clinic visits for lower respiratory tract disease headache
Sulfur dioxide	Industrial sites such as smelters, paper mills, power plants and steel manufacturing plants are the main sources.	↑ asthma hospitalization ↑ clinic visits for lower respiratory tract disease

30

<<NOTE TO USER: Here is an opportunity to summarize the important pollutants found in the local area.>>

Levels of air pollution are determined by sources, weather, climate and topography. Local conditions will determine the spectrum of acute and chronic health effects found in a given population of children.

ED: emergency department

Ref:

•American Academy of Pediatrics Committee on Environmental Health. *Pediatric Environmental Health. 2nd edition*. Etzel RA, Ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003.

•American Lung Association bibliography: http://www.cleanairstandards.org/article/articlereview/454/1/15

Outdoor Air Pollution

LEAD (Pb) IN AIR

SOURCES

- Tetraethyl lead in petrol
- Mining and smelting of lead ores
- Industry
- Waste incineration
- Dust (e.g. homes with old lead-based paint, battery recycling, smelters)

Ceppi, Corra

31

Emissions from motor vehicles are the main source of lead in air. Lead is principally present in the form of particles less than 1 micrometre. With the introduction of unleaded fuels in many countries, the amount of lead in the air is decreasing and population lead levels have fallen in those areas.

Note that all but 16 countries in the world are using unleaded petrol (as of July 2008). Updated regional petrol quality information can be found at www.unep.org/pcfv/regions/global.asp

Picture: R. Ceppi. L. Corra, Argentina. Used with permission.

Ref:

•*UNEP.* www.unep.org/pcfv/regions/global.asp

LEAD (Pb): HEALTH EFFECTS

- Interferes with many enzyme systems
  - Haem synthesis
  - Neurotoxic effects
  - Blood pressure
- Most severe effects in infants and young children

<<NOTE TO USER: This will be summarized quickly, and not emphasized here because full details are provided in the module on lead.>>

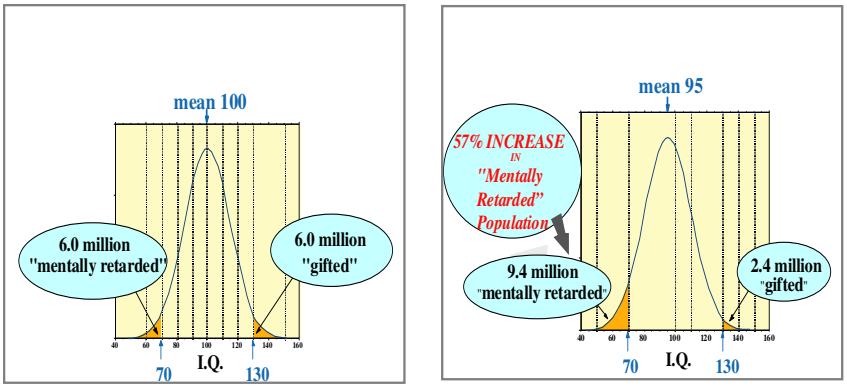
Ref:

•Diouf A et al. Environmental lead exposure and its relationship to traffic density among Senegalese children: a cross-sectional study. Hum Exp Toxicol. 2006 Nov;25(11):637-44.

Leaded-gasoline is probably the primary source of lead (Pb) exposure in Dakar (Senegal). The present cross-sectional study was undertaken to investigate the levels of Pb in Senegalese children and to present helpful data on the relationship between Pb levels and changes in biological markers of heme biosynthesis and oxidative stress. A total of 330 children, living since birth either in rural or urban areas (ie, Khombole (n = 162) and Dakar (n = 168), respectively) were included. During this cross-sectional study, the mean blood (B)-Pb level in all children was 7.32 +/- 5.33 microg/dL, and was influenced by the area of residence and gender. In rural children, 27 subjects (16.7%), 18 boys (19.6%) and nine girls (12.9%), had a B-Pb level > 10 microg Pb/dL, whereas 99 urban children (58.9%), respectively, 66 boys (71.8%) and 33 girls (43.4%), had alarmingly high B-Pb levels. Accordingly, urine delta-aminolevulinic acid levels were higher in children living in the urban area than in the rural areas (P < 0.001), and closely correlated with the B-Pb levels (P < 0.01). Moreover, glutathione peroxidase (GPx) activity, selenium (Se) level, glutathione reductase (GR) activity, and glutathione status were significantly influenced by area of residence and/or by gender. GPx activity and Se level were not only negatively correlated with B-Pb levels, but also positively correlated together (P < 0.01). Taken together, the present results allow us to conclude that urban children have higher B-Pb levels than rural children, and that of these children, boys have higher B-Pb levels than girls, leading thereby to alterations of heme biosynthesis and pro-oxidant/antioxidant balance. We also suggest that exposure to Pb and the Pb-induced adverse effects merits attention and that the development of preventive actions are of increasing importance in Senegal.

Outdoor Air Pollution

EFFECT OF LEAD EXPOSURE ON POPULATION IQ



www.prwventingharm.org/execsum.html
Schettler,,GBPSR, 2000

33

A 5-point loss in intelligence quotient (IQ) might not affect the ability of an individual to live a productive life. But if that loss is experienced by an entire population, the implications for that society could be profound.
Professor Bernard Weiss, a behavioural toxicologist at the University of Rochester, examined the societal impact of seemingly small decreases in intelligence. Imagine an unaffected population of 260 million people (such as in the USA) with an average IQ of 100 and a standard deviation of 15 (left-hand graph). In that population there would be 6 million people with IQs above 130 and 6 million below 70.

A decrease in average IQ of 5 points would shift the distribution to the left (right-hand graph). The number of people scoring above 130 would decline by 3.6 million whereas the number scoring below 70 would increase by 3.4 million.

IQ: intelligence quotient

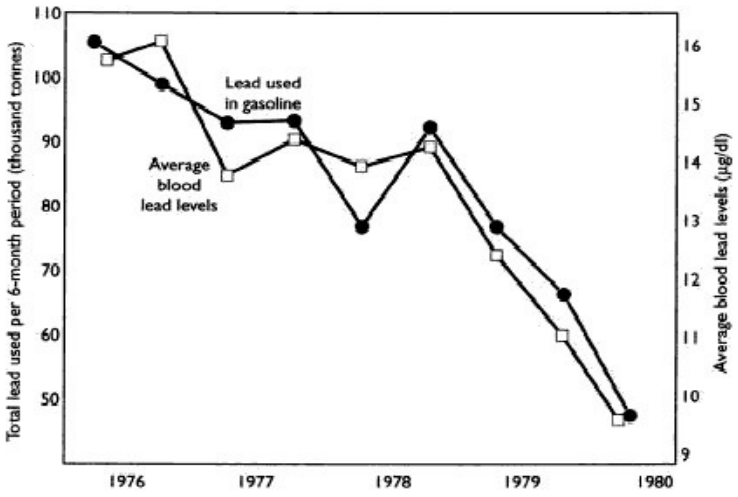
Graphic adapted from Schettler T. *In Harm' s Way.* Greater Boston Physicians for Social Responsibility, 2000. *Used with permission.*

Ref:

•Schettler T et al. *In Harm' s Way.* Greater Boston Physicians for Social Responsibility (GBPSR), 2000.

Outdoor Air Pollution

LEAD IN PETROL & BLOOD LEAD



Institute of Medicine, EPA, 1996

34

Effect of primary prevention

Removing lead from gasoline (petrol) in the USA closely parallelled the reduction of average blood lead levels in the American population. This strong correlation, illustrated by the graph of falling lead levels, is dramatic proof of how an intervention can profoundly improve population health.

Notes and picture from:

•Institute of Medicine. *Lead in the Americas: A Call for Action.* EPA, 1996.

Outdoor Air Pollution

CARCINOGENS IN AIR

Pollutant	*System affected*
❖ Arsenic	lung
❖ Benzene	leukaemia
❖ Chromium VI	lung
❖ Nickel	lung
❖ PAHs	lung
❖ Vinyl chloride	liver
❖ Radon	lung, gastrointestinal?

35

Whereas the previously mentioned pollutants (known as “criteria pollutants” in the USA) have long been recognized as ubiquitous hazards, there is increasing concern about a group of chemicals called “hazardous air pollutants” which are associated with adverse health effects. This is a list of some of the other chemical pollutants that are hazardous to health. PAHs stands for polycyclic aromatic hydrocarbons. Note that several different kinds of cancer may result from exposure (lung cancer, leukaemia and liver cancer). These cancers usually present during adulthood.

Refs:

•Liu J et al. Chronic arsenic poisoning from burning high-arsenic containing coal in Guizhou, China. *Environ Health Perspect,* 2002, 110:119.

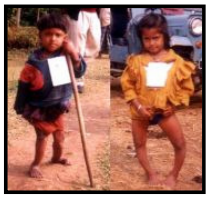
Arsenic is an environmental hazard and the reduction of drinking-water arsenic levels is under consideration. People are exposed to arsenic not only through drinking-water, but also through arsenic-contaminated air and food. Here we report the health effects of arsenic exposure from burning high arsenic-containing coal in Guizhou, China. Coal in this region has undergone mineralization and thus produces high concentrations of arsenic. Coal is burned inside the home in open pits for daily cooking and crop drying, producing a high concentration of arsenic in indoor air. Arsenic in the air coats and permeates food being dried producing high concentrations in food; however, arsenic concentrations in the drinking water are in the normal range. The estimated sources of total arsenic exposure in this area are from arsenic-contaminated food (50-80%), air (10-20%), water (1-5%), and direct contact in coal-mining workers (1%). At least 3000 patients with arsenic poisoning were found in the south-west Prefecture of Guizhou, and approximately 200 000 people are at risk for such overexposures. Skin lesions are common, including keratosis of the hands and feet, pigmentation on the trunk, skin ulceration and skin cancers. Toxicities to internal organs, including lung dysfunction, neuropathy, and nephrotoxicity, are clinically evident. The prevalence of hepatomegaly was 20%, and cirrhosis, ascites and liver cancer are the most serious outcomes of arsenic poisoning. The Chinese government and international organizations are attempting to improve the house conditions and the coal source, and thereby protect human health in this area.

Ref:

•*WHO air quality guidelines, 2nd ed.* Regional Office for Europe, European Series No. 91, 2000.

Outdoor Air Pollution

FLUOROSIS



- Endemic in at least 25 countries
  - Sources
    - Water (primary)
    - Air
    - Iatrogenic
- In China
  - From burning high-fluoride coal
  - Affects over 10 million people: dental and skeletal fluorosis

A. K. Susheela of Fluorosis Research & Rural Development Foundation of India 36

Fluorosis is a good example of how different regions may have to control the same pollutant by very different means to prevent the same disease. Dental and skeletal fluorosis are devastating problems in many countries, most commonly from fluoride-contaminated water, followed by overuse of dental preparations to prevent caries. In China, however, the major source of excess fluoride is air pollution from the burning of high-fluoride coal. In China, over 10 million people are suffering from this devastating condition.

Pictures: A. K. Susheela of Fluorosis Research & Rural Development Foundation of India Fluorosis Research And Rural Development (used with permission): A group of children who are fluoride-poisoned and reveal deformities or abnormalities including short stature (cretinism), bow-leg and knock-knee; they also suffer from deaf mutism, low IQ and mental retardation.

Ref:

•Ando M et al. Health effects of indoor fluoride pollution from coal burning in China. *Environ Health Perspect.* 1998;106(5):239-44.

The combustion of high fluoride-content coal as an energy resource for heating, cooking, and food drying is a major exhaust emission source of suspended particulate matter and fluoride. High concentrations of these pollutants have been observed in indoor air of coal-burning families in some rural areas in China. Because airborne fluoride has serious toxicological properties, fluoride pollution in indoor air and the prevalence of fluorosis have been analyzed in a fluorosis area and a healthy nonfluorosis area in China and in a rural area in Japan. For human health, fluoride in indoor air has not only been directly inhaled by residents but also has been absorbed in stored food such as corn, chilies, and potatoes. In the fluorosis area in China, concentrations of urinary fluoride in the residents have been much higher than in the nonfluorosis area in China and in the rural area in Japan. In the fluorosis area, almost all elementary and junior high school students 10-15 years of age had dental fluorosis. Osteosclerosis in the skeletal fluorosis patients was very serious. Urinary deoxypyridinoline in rural residents in China was much higher than in rural residents in Japan. Data suggest that bone resorption was extremely stimulated in the residents in China and that fluoride may stimulate both bone resorption and bone formation. Because indoor fluoride from combustion of coal is easily absorbed in stored food and because food consumption is a main source of fluoride exposure, it is necessary to reduce airborne fluoride and food contamination to prevent serious fluorosis in China.

•Chen Y et al. Air pollution-type fluorosis in the region of Pingxiang, Jiangxi, Peoples' Republic of China. *Arch Environ Health.* 1993 Jul-Aug;48(4):246-9.

This paper reports on the epidemiology, environmental factors, geological features, and total amount of fluoride intake by residents of the Pingxiang region in the Peoples' Republic of China where there is a high incidence of endemic fluorosis. The results demonstrate that the type of endemic fluorosis is related to air pollution, the major source of which comes from coal that is burned by the residents in their homes. Air pollution also originates from

Outdoor Air Pollution

BIOLOGICAL POLLUTANTS

- Moulds
- Pollens
- Castor bean dust
- Soya bean dust

Courtesy of Ruth A. Etzel, MD,

37

The chemical pollutants in the outdoor air are not the only pollutants of importance to child health. Several early epidemics of asthma were found to be linked to biological pollutants.

One example of a biological pollutant is soya bean dust. This is a picture of a ship in the harbour of Barcelona, Spain. It is unloading soya beans. In the 1980s, epidemics of asthma were occurring in Barcelona, and epidemiologic investigations linked the asthma epidemics to the loading and unloading of soya beans from ships in the harbour. The epidemics stopped when filters were put in place, preventing soya dust from being released into the outdoor air.

Ref:

•Figley KD et al. Endemic asthma due to castor bean dust. *JAMA,* 1928, 90:79.

•Strauss A. Collective asthma due to castor bean allergy in Ourinhos, S.P.: follow-up study after industrial processing of castor bean was stopped. Rev Inst Med Trop Sao Paulo. 1975 Mar-Apr;17(2):79-82. Links

•Anto JM et al. Community outbreaks of asthma associated with inhalation of soybean dust. *N Engl J Med,* 1989, 320:1097.

Since 1981, 26 outbreaks of asthma have been detected in the city of Barcelona. The geographical clustering of cases close to the harbour led us to consider the harbour as the probable source of the outbreaks. We therefore studied the association between the unloading of 26 products from ships in the harbour and outbreaks of asthma in 1985 and 1986. All 13 asthma-epidemic days in these two years coincided with the unloading of soya beans (lower 95 per cent confidence limit of the risk ratio, 7.2). Of the remaining 25 products, only the unloading of wheat was related to the epidemics of asthma, although when adjusted for the unloading of soya beans the relation was not statistically significant. High-pressure areas and mild southeasterly to south-westerly winds, which favored the movement of air from the harbour to the city, were registered on all epidemic days. Particles of starch and episperm cells that were recovered from air samplers placed in the city had morphological characteristics identical to those of soya bean particles. Furthermore, the lack of bag filters at the top of one of the harbour silos into which soya beans were unloaded allowed the release of soya bean dust into the air. We conclude that these outbreaks of asthma in Barcelona were caused by the inhalation of soya bean dust released during the unloading of soya beans at the city harbour.

•Anto JM et al. Preventing asthma epidemics due to soybeans by dust-control measures. *N Engl J Med,* 1993, 329:1760.

•Sunyer J et al. Case-control study of serum immunoglobulin-E antibodies reactive with soybean in epidemic asthma. *Lancet ,* 1989, 1:179.

Since 1981, twenty-six asthma outbreaks have been identified in Barcelona, all coinciding with the unloading of soya bean in the harbour. Serum from patients with epidemic asthma and individually matched controls with non-epidemic asthma was assayed for immunoglobulin-E (IgE) antibodies against soya bean antigens by means of a radioallergosorbent test. In 64 of 86 cases (74.4%) there was a reaction with commercial soya bean antigen extracts, compared with only 4 of the 86 controls (4.6%) (odds ratio = 61; lower 95% confidence limit = 8.1). The statistical significance was greater for reactions with extracts of soya bean dust taken from Barcelona harbour (odds ratio, unquantifiably high; lower 95% confidence limit = 11.7). No other serological covariate (total serum IgE levels or specific IgE levels against the commonest airborne allergens or legumes) confounded the association between serum anti-soya bean IgE antibodies and epidemic asthma. These results support a causal relation between the release of dust during unloading of soya bean at the harbour and the occurrence of asthma outbreaks, suggesting an underlying allergic mechanism.

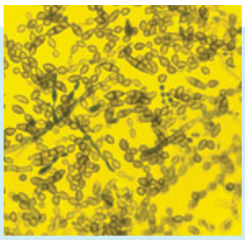
•White MC et al. Reexamination of epidemic asthma in New Orleans, Louisiana, in relation to the presence of soy at the harbor. *Am J Epidemiol,* 1997, 145:432.

Photo: Courtesy of Ruth A. Etzel, MD (Unloading of soya beans from a ship in the harbour in Barcelona, Spain)

Outdoor Air Pollution

MOULDS

EPA Mold growing on fallen leaves.



Magnified mold spores.

Molds can gradually destroy the things they grow on. You can prevent damage to your home and furnishings, save money, and avoid potential health problems by controlling moisture and eliminating mold growth.

EPA

- 60 species of moulds have spores that are allergenic
- 30% of patients with respiratory allergies are particularly sensitive to moulds
- Odds of death from asthma twice as high on days with outdoor mould spore counts $\geq$1000 spores/m^3

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Moulds are an important pollutant of the outdoor air. Exposure to moulds can cause severe asthma morbidity and mortality. Daily increases in mould spore counts are associated with daily increases in hospital admissions for asthma.

There may also be a synergistic effect between ozone and some mould spores. That is, the combined effects of exposure to ozone and mould spores are greater than the effects of either exposure alone.

Refs:

•Dales RE et al. Influence of outdoor aeroallergens on hospitalization for asthma in Canada. *J Allergy Clin Immunol ,* 2004, 113:303.

The risk of hospitalization for asthma caused by outdoor aeroallergens is largely unknown. The objective of this study was to determine the association between changes in outdoor aeroallergens and hospitalizations for asthma from the Pacific coast to the Atlantic coast of Canada. A daily time series analysis was done to test the association between daily changes in aeroallergens and daily changes in hospitalizations for asthma during a 7-year period between 1993 and 2000 in 10 of the largest cities in Canada. Results were adjusted for long-term trends, day of the week, climate, and air pollution. A daily increase, equivalent to the mean value of each allergen, was associated with the following percentage increase in asthma hospitalizations: 3.3% (95% CI, 2.3 to 4.1) for basidiomycetes, 3.1% (95% CI, 2.8 to 5.7) for ascomycetes, 3.2% (95% CI, 1.6 to 4.8) for deuteromycetes, 3.0% (95% CI, 1.1 to 4.9) for weeds, 2.9% (95% CI, 0.9 to 5.0) for trees, and 2.0% (95% CI, 1.1 to 2.8) for grasses. After accounting for the independent effects of trees and ozone, the combination of the two was associated with an additional 0.22% increase in admissions averaged across cities (P < 0.05). These findings provide evidence for the hypothesis that aeroallergens are an important cause of severe asthma morbidity across Canada, and in some situations there might be a modest synergistic adverse effect of ozone and aeroallergens combined.

•Jenkins HS et al. The effect of exposure to ozone and nitrogen dioxide on the airway response ot atopic asthmatics to inhaled allergen: dose-and time-dependent effects. *Am J Respir Crit Care Med,* 1999, 160:33.

•Molfino NA et al. Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet ,* 1991, 338:199.

•O'Hollaren MT et al. Exposure to aeroallergen as a possible precipitating factor in respiratory arrest in young patients with asthma. *N Engl J Med,* 1991, 324:359.

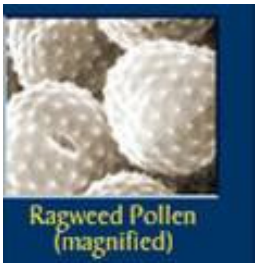
•Vagaggini B et al. Ozone exposure increases eosinophilic airway response induced by previous allergen challenge. *Am J Respir Crit Care Med,* 2002, 166:1073.

•www.epa.gov/iaq/molds/moldguide.html

Outdoor Air Pollution

POLLENS

- Penetrate into home
  - Air-conditioning
  - Air-filtration systems: HEPA filter
- High grass pollen levels linked to asthma admissions, epidemics
- Avoid outside play on high pollen days
- Antihistamine use



NIAID/NIH

WHO

Pollen is the male reproductive structure of flowering plants. Pollen exposure has long been recognized as a stimulant for symptoms of allergic disease, especially for allergic rhinitis (hay fever).

Pollen grains range in size from about 10 to 100 micrometres; the most common types are in the range of 15-30 micrometres. However, pollen allergens have been documented in air on much smaller particles.

Pollen is produced seasonally. In general, tree pollens are released early in the year, grasses during late spring and early summer, and weed pollens in the late summer and autumn. Major exceptions occur. For example, some grass pollen is produced throughout the year in some areas.

There is an association between grass pollen counts and admissions of patients with asthma in Mexico City in both dry and wet seasons.

In England, thunderstorms following periods of high pollen counts are more likely to lead to asthma epidemics.

Refs:

•Newson R et al. Acute asthma epidemics, weather and pollen in England, 1987-1994. *European Respiratory Journal*, 1998, 11:694.

Recent epidemics of acute asthma have caused speculation that, if their causes were known, early warnings might be feasible. In particular, some epidemics seemed to be associated with thunderstorms. We wondered what risk factors predicting epidemics could be identified. Daily asthma admissions counts during 1987–1994, for two age groups (0–14 yrs and > or = 15 yrs), were measured using the Hospital Episodes System (HES). Epidemics were defined as combinations of date, age group and English Regional Health Authority (RHA) with exceptionally high asthma admission counts compared to the predictions of a log-linear autoregression model. They were compared with control days 1 week before and afterwards, regarding seven meteorological variables and 5-day average pollen counts for four species. Fifty-six asthma epidemics were identified. The mean density of sferics (lightning flashes), temperature and rainfall on epidemic days were greater than those on control days. High sferics densities were overrepresented in epidemics. Simultaneously high sferics and grass pollen further increased the probability of an epidemic, but only to 15% (95% confidence interval 2–45%). Two thirds of epidemics were not preceded by thunderstorms. Thunderstorms and high grass pollen levels precede asthma epidemics more often than expected by chance. However, most epidemics are not associated with thunderstorms or unusual weather conditions, and most thunderstorms, even following high grass pollen levels, do not precede epidemics. An early warning system based on the indicators examined here would, therefore, detect few epidemics and generate an unacceptably high rate of false alarms.

•Rosas I et al. Analysis of the relationships between environmental factors (aeroallergens, air pollution, and weather) and asthma emergency admissions to a hospital in Mexico City. *Allergy,* 1998, 53:394.

Picture above: US National Institute of Allergy and Infectious Diseases/National Institutes of Health
Picture below: WHO

Outdoor Air Pollution

POPULATION-LEVEL
REDUCING EXPOSURE TO OUTDOOR AIR POLLUTION

- What interventions have been studied?
  - Industry closures (decreased respiratory admissions)
  - Replacement of "brown" coal as fuel
  - Changes in transportation patterns

<<READ SLIDE.>>

There is much health care providers can do to influence air pollution policies at the regional and national level. This slide gives a few examples of some interventions. These illustrative examples demonstrate how changes in industry, fuel and transport can improve respiratory health. The first example comes from the USA. When a steel mill in the Utah Valley, USA, closed, doctors observed a fall in hospital admissions for respiratory diseases. Children's admissions to the hospital for pneumonia, pleurisy, bronchitis and asthma were two to three times higher when the mill was open than when it was closed.

Ref:

•Pope CA. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. *Am J Public Health,* 1989, 79:623.

This study assessed the association between hospital admissions and fine particulate pollution (PM10) in Utah Valley during the period April 1985–February 1988. This time period included the closure and reopening of the local steel mill, the primary source of PM10. An association between elevated PM10 levels and hospital admissions for pneumonia, pleurisy, bronchitis and asthma was observed. During months when 24-hour PM10 levels exceeded 150 micrograms/m³, average admissions for children nearly tripled; in adults, the increase in admissions was 44 per cent. During months with mean PM10 levels greater than or equal to 50 micrograms/m3 average admissions for children and adults increased by 89 and 47 per cent, respectively. During the winter months when the steel mill was open, PM10 levels were nearly double the levels experienced during the winter months when the mill was closed. This occurred even though relatively stagnant air was experienced during the winter the mill was closed. Children's admissions were two to three times higher during the winters when the mill was open than when it was closed. Regression analysis also revealed that PM10 levels were strongly correlated with hospital admissions. They were more strongly correlated with children's admissions than with adult admissions and were more strongly correlated with admissions for bronchitis and asthma than with admissions for pneumonia and pleurisy.

•Ebelt S et al. Air quality in postunification Erfurt, East Germany: associating changes in pollutant concentrations with changes in emissions. *Environ Health Perspect,* 2001, 109:325.

The unification of East and West Germany in 1990 resulted in sharp decreases in emissions of major air pollutants. This change in air quality has provided an opportunity for a natural experiment to evaluate the health impacts of air pollution. We evaluated airborne particle size distribution and gaseous co-pollutant data collected in Erfurt, Germany, throughout the 1990s and assessed the extent to which the observed changes are associated with changes in the two major emission sources: coal burning for power production and residential heating, and motor vehicles. Continuous data for sulfur dioxide, total suspended particulates (TSP), nitric oxide, carbon monoxide, and meteorological parameters were available for 1990–1999, and size-selective particle number and mass concentration measurements were made during the winters of 1991 and 1998. We used hourly profiles of pollutants and linear regression analyses, stratified by year, weekday/weekend, and hour, using NO and SO_2 as markers of traffic- and heating-related combustion sources, respectively, to study the patterns of various particle size fractions. Supplementary data on traffic and heating-related sources were gathered to support hypotheses linking these sources with observed changes in ambient air pollution levels. Substantially decreased (19–91%)

Outdoor Air Pollution

POPULATION-LEVEL
DECREASED EMISSIONS→ IMPROVED HEALTH

- During the 1996 Olympic Games, public transport was encouraged, areas of Atlanta were closed to private vehicles, telecommuting was encouraged

  →Decreased levels of ozone pollution occurred

Acute asthma events decreased by 42%

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This slide summarizes the changes associated with changes in transportation patterns that were implemented by the city of Atlanta during the 1996 Summer Olympic Games. The strategy for decreasing emissions during the Games included the following measures:

•integrated 24-hour-a-day public transport system

•1000 additional buses

•local business use of alternative working hours and telecommuting

•closure of city centre to private cars

•public warnings of potential traffic and air quality problems

The following results were reported:

•28% drop in ozone concentrations during the Olympic Games

•217% increase in overall public transportation use

•11–44% reduction in the number of acute asthma events

Ref:

•Friedman MS et al. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA,* 2001, 285:897.

Outdoor Air Pollution

WHO AIR QUALITY GUIDELINES

Substance	Lowest concentration at which adverse effects are observed	Duration of exposure
Ozone	120 μg/m^3	8 hours
NO_2	200 μg/m^3	1 hour
CO *(a)*	30 mg/m^3	1 hour
SO_2	500 μg/m^3	10 minutes
PM *(b)*	Dose–response	
Lead	0.5 μg/m^3	1 year

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In countries with strong air pollution laws and good enforcement, air quality improved significantly in the latter half of the 20th century. WHO has proposed air quality guidelines for the major "criteria" air pollutants. Reductions to these levels offer significant health benefits.

The guideline values for individual substances based on effects other than cancer and annoyance from odour are given in the table above. The emphasis in the guidelines is placed on exposure (lowest concentration at which adverse effects are observed), because this is the element that can be controlled to lessen dose and hence lessen the consequent health effect. When general ambient air levels are orders of magnitude lower than the guideline values, present exposures are unlikely to cause concern. Guideline values in those cases are directed only to specific release episodes or specific indoor pollution problems.

For example: with carbon monoxide, the lowest concentrations at which adverse effects are observed are:

- 100 000 μg/m^3 (100 mg/m^3) with averaging time of exposure: 15 minutes
- 60 000 μg/m^3 (60 mg/m^3) with averaging time of exposure: 30 minutes
- 30 000 μg/m^3 (30 mg/m^3) with averaging time of exposure: 1 hour
- 10 000 μg/m^3 (10 mg/m^3) with averaging time of exposure: 8 hours

(a) Exposure at these concentrations should be for no longer than the indicated times and should not be repeated within 8 hours.

(b) The available information for short- and long-term exposure to PM_{10} and $PM_{2.5}$ does not allow a judgement to be made regarding concentrations below which no effects would be expected. For this reason no guideline values have been recommended, but instead risk estimates have been provided (for more information, see chapter 7, Part 3 of WHO Air Quality Guidelines).

PM: particulate matter

PM_{10} particles with a diameter less than 10 micrometres

$PM_{2.5}$ particles with a diameter less than 2.5 micometres (fine particulates)

Ref:

- *Air Quality Guidelines. Global update 2005. Particulate matter, ozone, nitrogen dioxide and sulfur dioxide, WHO, 2006.*
- *WHO Air Quality Guidelines, 2nd ed.* Regional Office for Europe, European Series No. 91, 2000.

Regulation is an important step, and prevention should include regulation of emissions. This slide is to be used in the USA; in other countries it will be important to show country- specific legislation.

<<NOTE TO USER: Please insert information on the current and/or needed laws in your region or country.>>

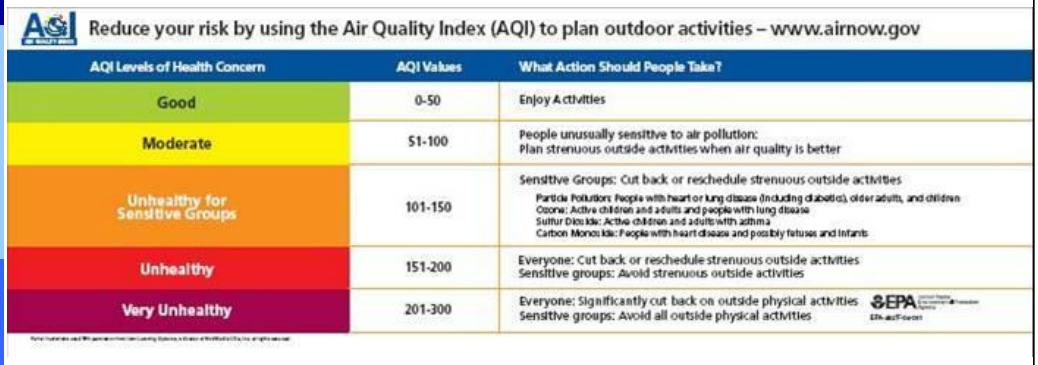
The American Academy of Pediatrics has formulated advice to paediatricians on how to integrate issues regarding air quality into patient education and health advocacy.

Ref:

•Committee on Environmental Health, American Academy of Pediatrics. Ambient air pollution: health hazards to children. *Pediatrics,* 2004, 114:1699.

Outdoor Air Pollution

PREVENTION AT INDIVIDUAL LEVEL
THE EXAMPLE OF EPA RECOMMENDATIONS (USA)



Reduce your risk by using the Air Quality Index (AQI) to plan outdoor activities – www.airnow.gov

AQI Levels of Health Concern	AQI Values	What Action Should People Take?
Good	0-50	Enjoy Activities
Moderate	51-100	People unusually sensitive to air pollution: Plan strenuous outside activities when air quality is better
Unhealthy for Sensitive Groups	101-150	Sensitive Groups: Cut back or reschedule strenuous outside activities Particle Pollution: People with heart or lung disease (including diabetics), older adults, and children Ozone: Active children and adults and people with lung disease Sulfur Dioxide: Active children and adults with asthma Carbon Monoxide: People with heart disease and possibly fetuses and infants
Unhealthy	151-200	Everyone: Cut back or reschedule strenuous outside activities Sensitive groups: Avoid strenuous outside activities
Very Unhealthy	201-300	Everyone: Significantly cut back on outside physical activities Sensitive groups: Avoid all outside physical activities

AQI for: O_3, PM, CO, SO_2 and NO_2

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The Air Quality Index (AQI) is an index for reporting daily air quality. It tells you how clean or polluted your air is, and what associated health effects might be a concern for you. The AQI focuses on health effects you may experience within a few hours or days after breathing polluted air. USEPA calculates the AQI for five major air pollutants regulated by the Clean Air Act: ground-level ozone, particle pollution (also known as particulate matter), carbon monoxide, sulfur dioxide and nitrogen dioxide. For each of these pollutants, EPA has established national air quality standards to protect public health.

How does the AQI work?

Think of the AQI as a yardstick that runs from 0 to 500. The higher the AQI value, the greater the level of air pollution and the greater the health concern. For example, an AQI value of 50 represents good air quality with little potential to affect public health, whereas an AQI value over 300 represents hazardous air quality.

An AQI value of 100 generally corresponds to the national air quality standard for the pollutant, which is the level EPA has set to protect public health. AQI values below 100 are generally thought of as satisfactory. When AQI values are above 100, air quality is considered to be unhealthy – at first for certain sensitive groups of people, then for everyone as AQI values get higher.

Notes and table taken from: www.epa.gov/airnow/aqi.html

O_3: ozone

PM: Particulate matter

CO: Carbon monoxide

SO_2: sulfur dioxide

NO_2: nitrogen dioxide

Activities causing emissions of pollutants affecting health are often related with other impacts on the environment or health. For example, combustion of fossil fuels results in emission of carbon dioxide, one of the gases responsible for green house effect. Reduction of combustion, e.g. through implementation of fuel or energy efficiency measures, may lead to the reduction of CO_2 emissions. It is important to note that not all pollution reduction actions will contribute to the "climate change" policies and vice versa. However, there are examples showing that the selection of the options assuring synergies of the both policies can be the most economically viable and profitable of various options (M. Krzyzanowski, WHO/EURO).

The United Nations Framework Convention on Climate Change (1992) sets an overall framework for intergovernmental efforts to tackle the challenge posed by climate change. It recognizes that the climate system is a shared resource whose stability can be affected by industrial and other emissions of carbon dioxide and other heat-trapping gases.

Under the Convention, governments:

•Gather and share information on greenhouse gas emissions, national policies and best practices.

•Launch national strategies for addressing greenhouse emissions and adapting to expected impacts, including the provision of financial and technological support to developing countries to enable them to cooperate in preparing for adaptation to the impacts of climate change.

When they adopted the Convention, governments knew that its commitments would not be sufficient to seriously tackle climate change. At COP 1 (Convention of the Parties, Berlin, March/April 1995), in a decision known as the Berlin Mandate, Parties therefore launched a new round of talks to decide on stronger and more detailed commitments for industrialized countries. After two and a half years of intense negotiations, the Kyoto Protocol was adopted at COP 3 in Kyoto, Japan, on 11 December 1997. The 1997 Kyoto Protocol shares the Convention's objective, principles and institutions, but significantly strengthens the Convention by committing Annex I Parties to individual, legally-binding targets to limit or reduce their greenhouse gas emissions. These add up to a total cut in greenhouse-gas emissions of at least 5% from 1990 levels in the commitment period 2008–2012.

The complexity of the negotiations, however, meant that considerable "unfinished business" remained even after the Kyoto Protocol itself was adopted. The Protocol sketched out the basic features of its "mechanisms" and compliance system, for example, but did not flesh out the all-important rules of how they would operate. Although 84 countries signed the Protocol, indicating that they intended to ratify, many were reluctant to actually do so and bring the Protocol into force before having a clearer picture of the treaty's rulebook. A new round of negotiations was therefore launched to flesh out the Kyoto Protocol's rulebook, conducted in parallel with negotiations on ongoing issues under the Convention. This round finally culminated at COP 7 with the adoption of the Marrakesh Accords, setting out detailed rules for the implementation of the Kyoto Protocol. As discussed above, the Marrakesh Accords also took some important steps forwards regarding the implementation of the Convention.

These notes are taken from: unfccc.int

Outdoor Air Pollution

CRITICAL ROLE OF HEALTH & ENVIRONMENT PROFESSIONALS

- Diagnose and treat
- Do research and publish
  - Sentinel cases
  - Community-based interventions
- Educate
  - Patients and families
  - Colleagues and students
- Advocate
- Provide good role model

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Health and environment professionals have a critical role to play in maintaining and stimulating changes that will restore and protect children's environmental health. Although the human genome project is critically important and scientifically exciting, we all know that genes express themselves within an environment and understanding gene–environment interactions will help keep our children healthy. So, as we look to our political and personal lives to support sustainable development, we can look to our practices for ways to enhance the environmental health of our patients. All of us can do something. At the one-to-one patient level we can include environmental etiologies in our differential diagnoses and preventive advice. We can be dissatisfied with the diagnosis of "idiopathic" and look hard for environmental causes of disease and disability. We can publish sentinel cases and develop and write up community-based interventions. We can educate our patients, families, colleagues and students didactically. Finally, we can become vigorous advocates for the environmental health of our children and future generations. It's not enough to be an informed citizen, we need to write letters, testify at hearings, approach our elected officials with education and messages. And, we can recognize that as professionals with an understanding of both health and the environment, we are powerful role models. Our choices will be noticed and they should be thoughtful and sustainable.

Ref:

•*WHO/IPA leaflet: http://www.who.int/indoorair/publications/paediatrician/en/index/html*

Picture: WHO

Outdoor Air Pollution

To end this presentation, a beautiful reminder to us from a child who has drawn a clean environment, where a healthy atmosphere brings happiness and health.

Thank you.

POINTS FOR DISCUSSION

<<NOTE TO USER: Add points for discussion according to the needs of your audience.>>

ACKNOWLEDGEMENTS

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Outdoor Air Pollution

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