TRAINING FOR THE HEALTH SECTOR

OUTDOOR AIR POLLUTION

Children's Health and the Environment

CHEST Training Package for the Health Sector

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

This module is based on the WHO moduel "Outdoor air pollution".

First draft prepared by Ruth A. Etzel, MD PhD, USA

LEARNING OBJECTIVES

- Introduce the major outdoor air pollutants
- Describe their sources
- Describe their major health effects (short- and long-term)
- List some strategies to reduce outdoor air pollution

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

- List the major outdoor air pollutants
- Describe their sources
- Describe their major health effects
- 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.

HISTORY

"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."

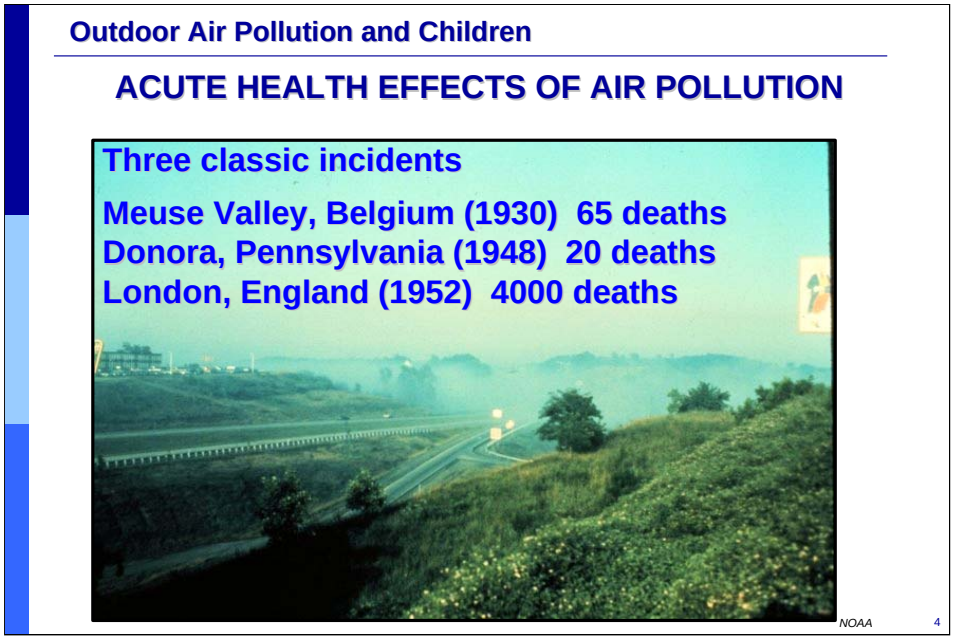
Roman philosopher Seneca, AD 61

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

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.



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.

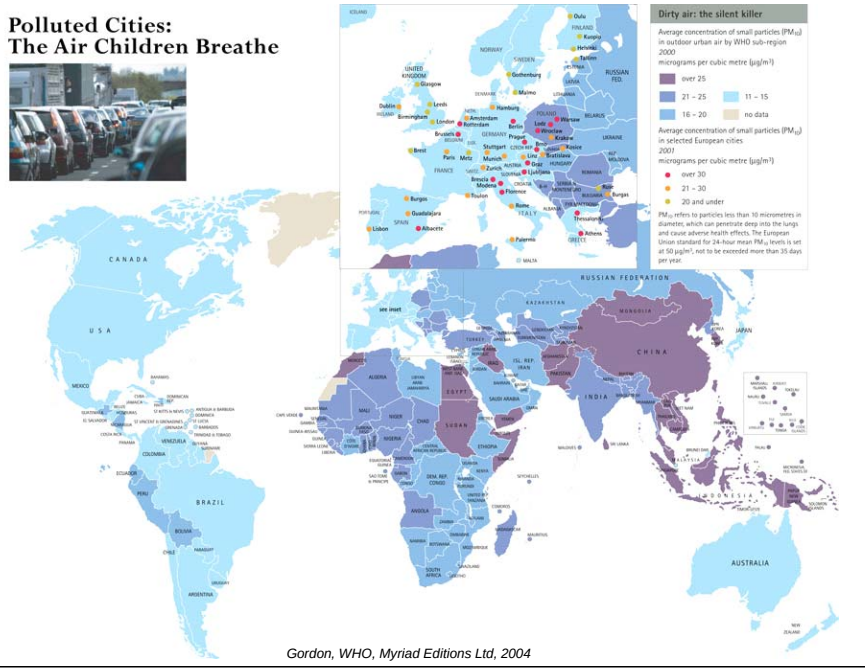
In England, the Clean Air Act of 1956 banned emissions of black smoke and decreed that the residents of urban areas and operators of factories must convert to smokeless fuels.

Ref: Ware. 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

Outdoor Air Pollution and Children



Gordon, WHO, Myriad Editions Ltd, 2004

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

SCOPE OF THE PROBLEM

- Worldwide, outdoor air pollution contributes to:
  - ~ 800 000 deaths per year
  - ~ 4.6 million healthy life-years lost per year
- Uneven burden
  - 65% deaths and lost life-years occur in Asia
- Need for regionally developed research

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

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

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

PRINCIPAL SOURCES

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

Etzel

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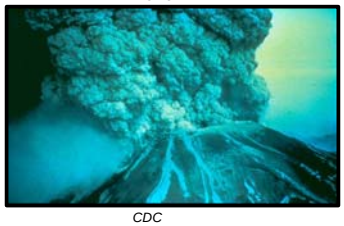


CDC

<<READ SLIDE.>>

<<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.

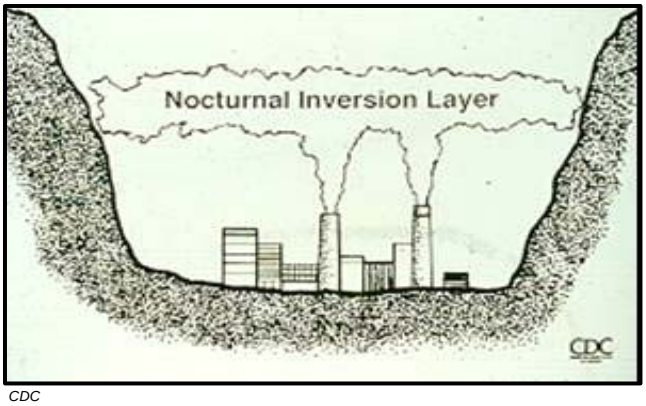
Pictures:

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, that 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: 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: OTHER SOURCES



CDC

- Climate
  - Temperature
  - Prevailing winds
  - Seasonal changes
- Topography
  - 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).

"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 and Children

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

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.

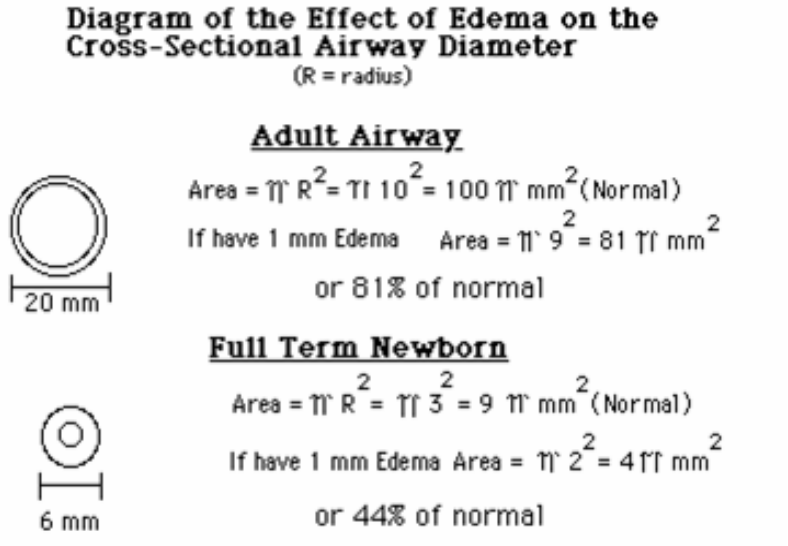
In addition to an increased need for oxygen relative to their size, children have narrower airways than do adults. Thus, irritation caused by air pollution that would produce only a slight response in an adult can result in potentially significant obstruction in the airways of a young child.

Ref:

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

Outdoor Air Pollution and Children

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. 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

Outdoor Air Pollution and Children

MAJOR OUTDOOR AIR POLLUTANTS

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

WHO

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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.

Ref: Gauderman. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med,* 2004, 351:1057.

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)

DEPOSITION OF POLLUTANTS IN RESPIRATORY TRACT

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

CDC

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.

PARTICULATE MATTER

- Complex heterogeneous mixture of solid and liquid components

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

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.

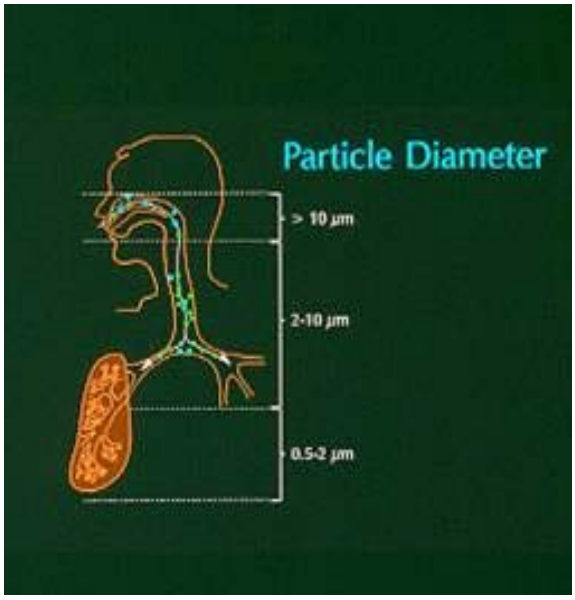
The specific composition and size distribution of PM varies by region, time of year, time of day, weather conditions and other factors. For example, sulfates dominate the $PM_{2.5}$ mixture in the eastern United States, whereas nitrates are more abundant in the western United States.

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).

Outdoor Air Pollution and Children

SIZE MATTERS

- Coarse particles (2.5–10 microns) deposited in the upper respiratory tract and large airways
- Fine particles (< 2.5 microns) 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.

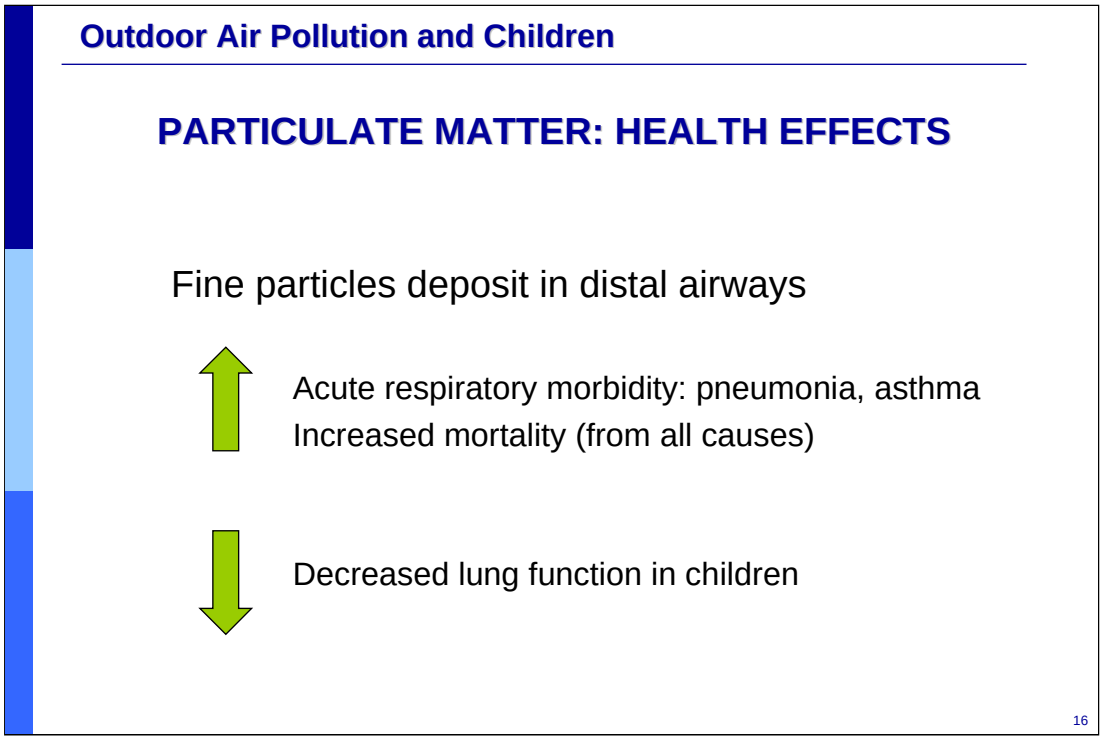
Compared with large particles, fine particles can remain suspended in the atmosphere for longer periods and be transported over longer distances.

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

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

Ref:

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



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. 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.

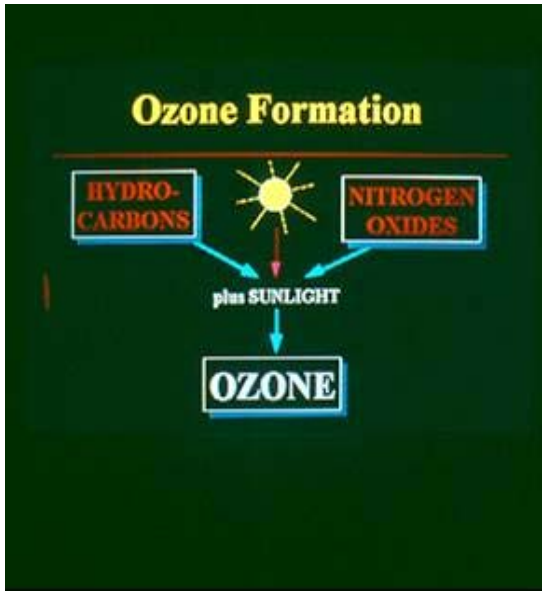
•Dickey. Part VII. Air pollution: overview of sources and health effects. *Dis Mon,* 2000, 46:566.

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

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

OZONE (O_3): "SECONDARY POLLUTANT"

- **Formed by photochemical reaction of VOCs, NO_2 + O_2**
  - Peaks late afternoon
  - Maximum in hot, stagnant air
- **High concentrations increase asthma attacks in children**



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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.

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)**

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 college years.

Ref:

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

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OZONE

- Chronic exposure to ozone is associated with:
  - chronic lung disease
  - mild pulmonary fibrosis
  - small airway obstruction
- 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 $FEF_{25-75\%}$ (in 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.

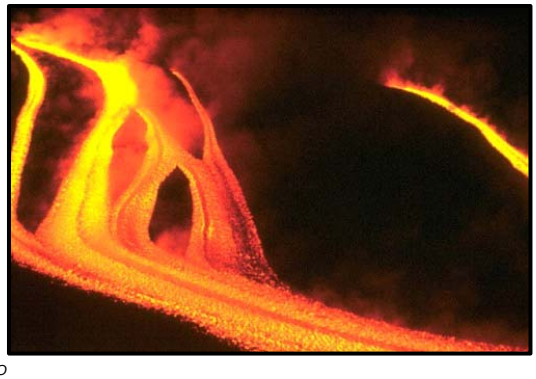
Some evidence has linked ozone to chronic lung scarring, especially at the bronchoalveolar junction.

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

Ref: Gauderman, Association between Air Pollution and Lung Function Growth in Southern California Children. *Am J Respir Crit Care Med,* 2000, 162:1383.

NITROGEN OXIDES: 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)

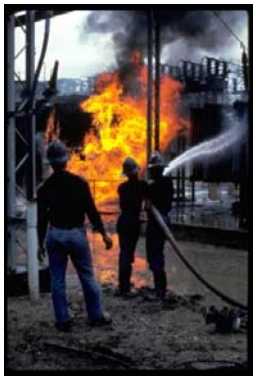
NITROGEN OXIDES: HEALTH EFFECTS

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

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

CARBON MONOXIDE (CO): SOURCES

- Incomplete combustion of fuels
- Vehicle exhaust
- Industry
- Fires



WHO

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 the USA, some new fuel additives are being suggested for use in areas with serious carbon monoxide problems. The two primary “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)

CARBON MONOXIDE: HEALTH EFFECTS

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

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) that compromises oxygen delivery are also more susceptible to adverse effects of exposure to CO at lower levels than are healthy individuals.

Chronic exposure to low levels of CO causes headaches.

SULFUR DIOXIDE: 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.

Outdoor Air Pollution and Children

SULFUR DIOXIDE: HEALTH EFFECTS

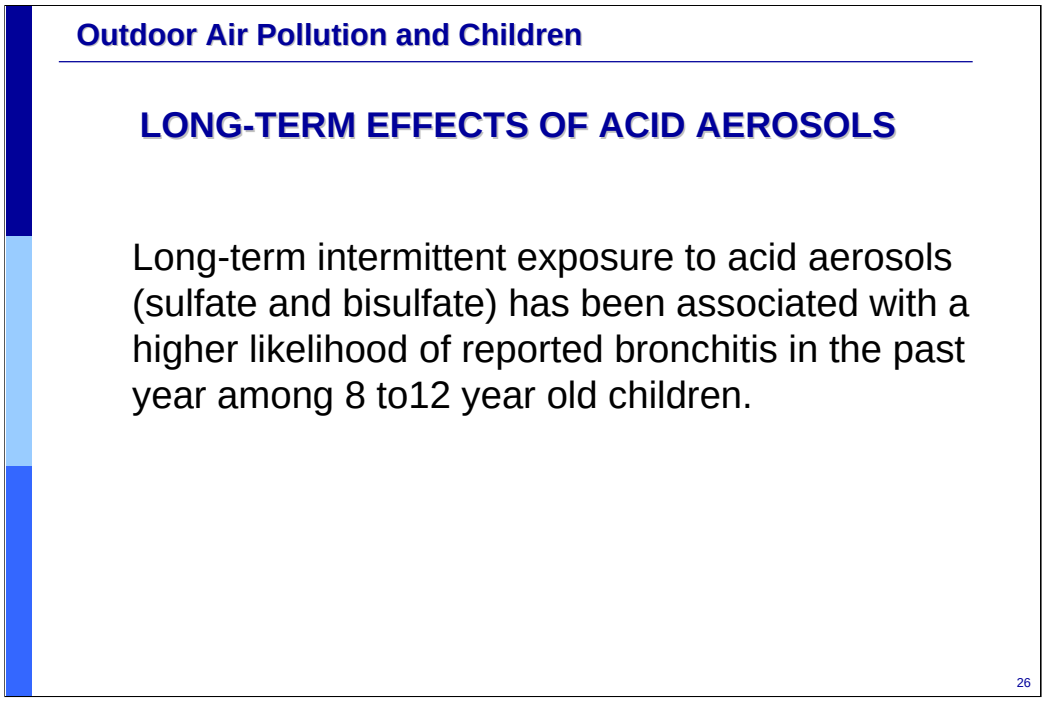
- Absorbed on upper airways (irritant)
- Primarily affects nasopharynx and proximal airways
- Acid aerosol reaches distal airways
  - bronchoconstriction
  - slowing of bronchial mucociliary clearance
  - chronic bronchitis

25

<<READ SLIDE.>>

Ref: Pikhart. 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.



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. 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. 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.

LONG-TERM EFFECTS OF ACID AEROSOLS

Long-term intermittent exposure to acid aerosols (sulfate and bisulfate) has been associated with a higher likelihood of reported bronchitis in the past year among 8-12-year-old children.

Asthma, persistent wheeze, chronic cough and chronic phlegm were not significantly associated with higher levels of acid aerosols among 8-12- year-old children.

Long-term exposure to acid aerosols was associated with statistically significant decrements in FVC and FEV1 among 8-12-year-old children.

Ref: Dockery, Health effects of acid aerosols on North American children: respiratory symptoms, Environ Health Perspect. (1996) 104(5):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 analyze 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.

Outdoor Air Pollution and Children

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

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ED, emergency department.

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.

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

LEAD: SOURCES IN AIR

- Tetraethyl lead in petrol
- Mining and smelting of lead ores
- Industry
- Waste incineration
- Dust around homes with old lead-based paint

Other media

- Water pipes, paint, food

Ceppi, Corra

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.

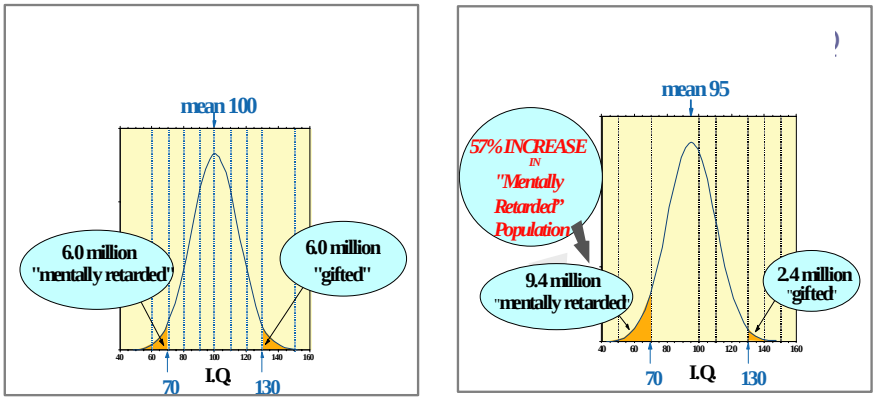
Picture: R. Ceppi. L. Corra, Argentina. Used with permission.

LEAD: 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 given in the module on lead.>>

EFFECT OF LEAD EXPOSURE ON POPULATION IQ



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

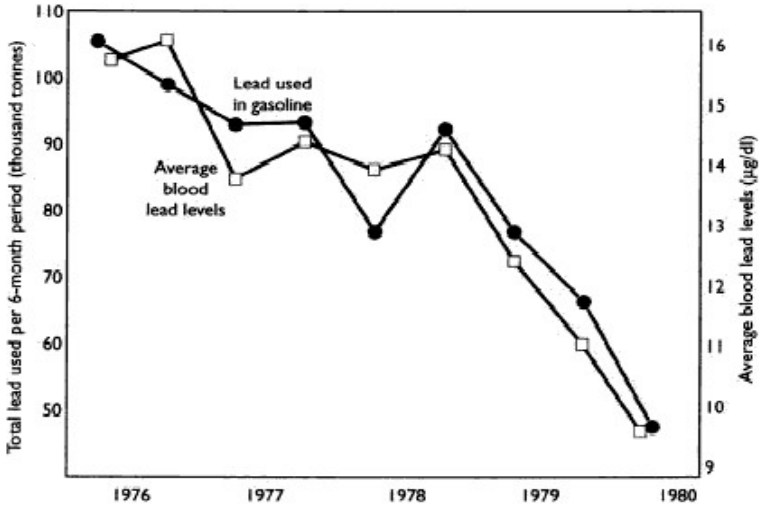
A 5-point loss in IQ might not affect the ability of an individual to live a productive life. But if that loss is experienced by an entire population, the implications for that society could be profound.
Bernard Weiss, a behavioural toxicologist at the University of Rochester, examined the societal impact 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.

Picture adapted from Schettler. *In harm' s way.* GBPSR, 2000. *Used with permission.*

Outdoor Air Pollution and Children

GET THE LEAD OUT



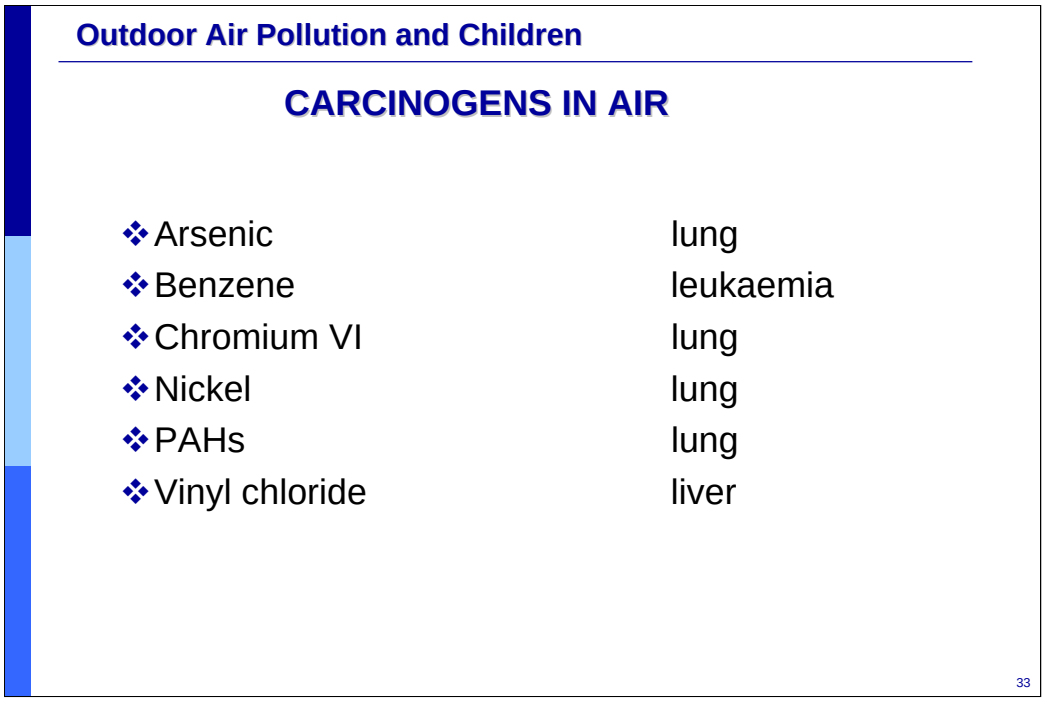
Institute of Medicine, EPA, 1996

Effect of primary prevention

Removing lead from gasoline (petrol) in the USA closely parallels 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 a single intervention can profoundly improve population health.

Ref:

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



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.

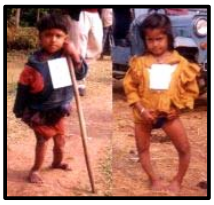
*Refs:*Liu. Chronic arsenic poisoning from burning high-arsenic containing coal in Guizhou, China. *Environ Health Perspect,* 2002, 110:119.

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.

•*WHO Air Quality Guidelines for Europe. 2nd ed.* European Series, No. 91, 2000.

Outdoor Air Pollution and Children

FLUOROSIS



- Endemic in at least 25 countries
  - Sources
    - Water
    - 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

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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.

Outdoor Air Pollution and Children

BIOLOGICAL POLLUTANTS

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

Etzel

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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. You may remember that epidemics of asthma were occurring in Barcelona, and epidemiological investigations linked the asthma epidemics to the loading and unloading of soya beans from ships in the harbour.

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

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

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

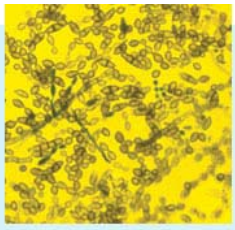
Since 1981, twenty-six asthma outbreaks have been identified in Barcelona, all coinciding with the unloading of soya bean in the harbour. Serum from patients with epidemic asthma and individually matched controls with non-epidemic asthma was assayed for immunoglobulin-E (IgE) antibodies against soya 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. Reexamination of epidemic asthma in New Orleans, Louisiana, in relation to the presence of soy at the harbor. *Am J Epidemiol,* 1997, 145:432.

Outdoor Air Pollution and Children

MOULDS

EPA Mold growing on fallen leaves.



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.

Magnified mold spores.

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

36

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

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

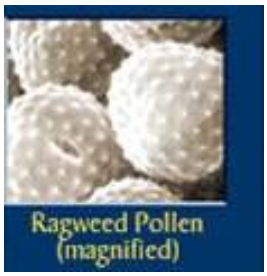
•Vagaggini. Ozone exposure increases eosinophilic airway response induced by previous allergen challenge. *Am J Respir Crit Care Med,* 2002, 166:1073.

•www.epa.gov/iaq/molds/moldguide.html

Outdoor Air Pollution and Children

POLLENS

- Avoid outside play on high pollen days
- Antihistamine use
- Penetrate into home
  - Air-conditioning
  - Air-filtration systems: HEPA filter



NIAID/NIH

WHO 37

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 microns; the most common types are in the range of 15-30 microns. 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. 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. 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.

Outdoor Air Pollution and Children

TOOLS FOR CLINICIANS AND CITIZENS

Good

Moderate

Unhealthy

Very Unhealthy

Air Quality Index Levels of Health Concern	Numerical Value	Cautionary Statements	Meaning
Good	0-50		Air quality is considered satisfactory, and air pollution poses little or no risk.
Moderate	51-100		Air quality is acceptable; however, for some pollutants there may be a moderate health concern for a very small number of people who are unusually sensitive to air pollution.
Unhealthy for Sensitive Groups	101-150	Active children and adults, and people with respiratory disease, such as asthma, should limit prolonged outdoor activity.	Members of sensitive groups may experience health effects. The general public is not likely to be affected.
Unhealthy	151-200	Active children and adults, and people with respiratory disease, such as asthma, should avoid heavy outdoor exertion. Everyone else, especially children, should limit heavy outdoor exertion.	Everyone may begin to experience health effects; members of sensitive groups may experience more serious health effects.
VeryUnhealthy	201-300	Active children and adults, and people with respiratory disease, such as asthma, should avoid all outdoor exertion. Everyone else, especially children, should limit outdoor exertion.	Health alert: everyone may experience more serious health effects.
Hazardous	> 300	Everyone should avoid all outdoor exertion.	Health warnings of emergency conditions. The entire population is more likely to be affected

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The air quality index is used in the United States to help provide information about pollution levels to the public. This index converts the concentrations of five specific pollutants (CO, ozone, NO_2, SO_2 and particulate matter) into one number, on a scale from 0 to 500. The air quality index of 100 corresponds to the short-term national ambient air quality standard. Thus a concentration above 100 indicates that the concentration of one or more pollutants exceeds its national standard. The descriptor terms associated with different air quality index values are as follows:

0 to 50 “good”

51 to 100 “moderate”

101 to 150 “unhealthy for sensitive groups”

151 to 200 “unhealthy”

201 to 300 “very unhealthy”

301 to 500 “hazardous”

Ref:

•US Environmental Protection Agency. *Guidelines for reporting of daily air quality index (AQI). Washington, DC, US Environmental Protection Agency, 1999.*

POPULATION-LEVEL
REDUCING OUTDOOR AIR POLLUTION

- What interventions have been studied?
  - Industry closures
  - Changes in transportation patterns
  - Replacement of “brown” coal as fuel

<<READ SLIDE.>>

There is much health care providers can do to influence air pollution policies at the regional and national level. Three illustrative examples will show how changes in industry, transport and fuel can improve respiratory health.

POPULATION- LEVEL
INDUSTRY CLOSURES

When a steel mill in the Utah Valley, USA, closed, doctors observed a fall in hospital admissions for respiratory diseases.

Ref:

•Pope. 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/m^3, 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.

Outdoor Air Pollution and Children

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%

41

The strategy for decreasing emissions during the Olympic Games in Atlanta 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; and

•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; and

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

Ref:

•Friedman. 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 and Children

POPULATION-LEVEL
STUDIES IN EAST AND WEST GERMANY

- Since German reunification in 1990, levels of air pollution have declined tremendously in eastern Germany
  - Closure of numerous industrial plants
  - Brown “dirty” coal replaced by gas (for heating)
- Eastern Germany, 1992–1999
  - 92% decrease in sulfur dioxide
  - 58% decrease in total suspended particulates
  - Notable declines in bronchitis, sinusitis and frequent colds
  - No decline in asthma

42

The speaker should acknowledge that there is still some scientific disagreement regarding the role that outdoor air pollution may play in the rates of respiratory diseases and asthma in eastern and western Germany.

<<READ SLIDE.>>

Refs: Ebelt. 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%) concentrations were observed for all pollutants, with the exception of particles in the 0.01–0.03 micrometre size range (representing the smallest ultrafine particles that were measured). The number concentration for these particles increased by 115% between 1991 and 1998. The ratio of these ultrafine particles to TSP also increased by more than 500%, indicating a dramatic change in the size distribution of airborne particles. Analysis of hourly concentration patterns indicated that in 1991, concentrations of SO(2) and larger particle sizes were related to residential heating with coal. These peaks were no longer evident in 1998 due to decreases in coal consumption and consequent decreased emissions of SO(2) and larger particles. These decreases in coal combustion and the decreased concentrations of SO(2) and particles of larger size classes may have led to decreased particle scavenging and may be partially responsible for the observed increases in ultrafine particles. Traffic-related changes, such as increased numbers of trucks and increased use of diesel vehicles in Erfurt, were also associated with increased number concentrations of ultrafine particles. Morning particle peaks of all sizes were associated with NO and CO (markers for traffic) in both the 1991 and 1998 periods. There were significant differences in the ultrafine particle levels for morning hours between 1991 and 1998, suggesting that traffic was the cause of this increase.

•Heinrich. Nonallergic respiratory morbidity improved along with a decline of traditional air pollution levels: a review. *Eur Respir J (Suppl),* 2003, 40:64s.

•Weiland. Prevalence of respiratory and atopic disorders among children in the East and West of Germany five years after reunification. *Eur Respir J,* 1999, 14:862.

US CLEAN AIR LEGISLATION

- 1955 Clean Air Legislation
- 1963 Clean Air Act
- The Clean Air Quality Act of 1967
- 1970 Clean Air Act Amendments
- 1977 Clean Air Act Amendments
- 1990 Clean Air Act Amendments

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 specific legislation.

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

Ref:

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

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

Outdoor Air Pollution and Children

WHO AIR QUALITY STANDARDS

Substance	Lowest concentration at which adverse effects are observed	Duration of exposure
Ozone	120 ìg/m^3	8 hours
NO_2	200 ìg/m^3	1 hour
CO *(a)*	30 mg/m^3	1 hour
SO_2	500 ìg/m^3	10 minutes
PM *(b)*	Dose–response	
Lead	0.5 ìg/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 standards 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 ìg/m^3 (100 m/m^3) with averaging time of exposure: 15 minutes
- 60 000 ìg/m^3 (60 m/m^3) with averaging time of exposure: 30 minutes
- 30 000 ìg/m^3 (30 m/m^3) with averaging time of exposure: 1 hour
- 10 000 ìg/m^3 (10 m/m^3) with averaging time of exposure: 8 hours

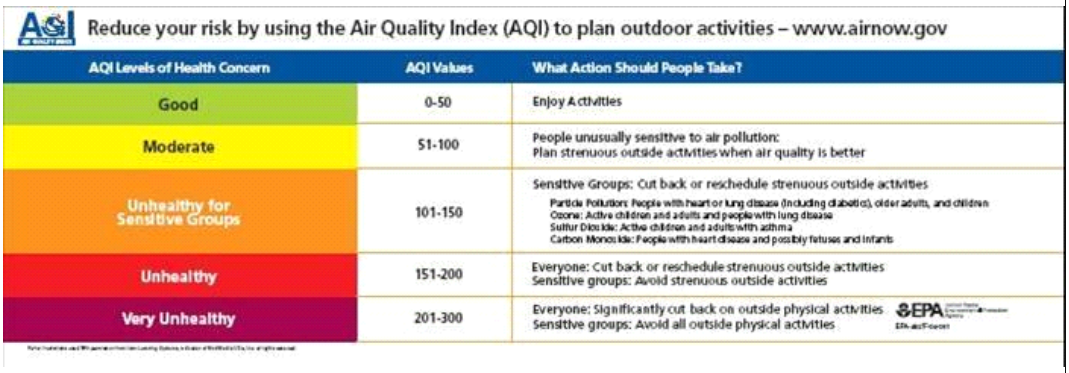
(a) Exposure at these concentrations should be for no longer than the indicated times and should not be repeated within 8 hours.

(b) The available information for short- and long-term exposure to PM10 and PM2.5 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).

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

Outdoor Air Pollution and Children

PREVENTION AT INDIVIDUAL LEVEL
THE EXAMPLE OF EPA RECOMMENDATIONS



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 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. EPA 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

PREVENTION AT INTERNATIONAL LEVEL

- 1992: UN Framework Convention on Climate Change
- 1997: Kyoto Protocol: entered into force, 16 February 2005
- 2001: Marrakesh Accords

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: http://unfccc.int

CRITICAL ROLE OF HEALTH & ENVIRONMENT PROFESSIONALS

WHO

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

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 must all 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 must all 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.