TRAINING FOR THE HEALTH SECTOR

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INDOOR 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: 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.>>

LEARNING OBJECTIVES TO UNDERSTAND, RECOGNIZE AND KNOW:

- ❖ Hazards of indoor air pollution to children's health
- Different toxicants in indoor air, according to sources, settings and activities
- Characteristics and issues relating to indoor air pollution in industrialized and developing countries
- ❖ How to recognize, assess and address health effects
- **❖** How to prevent exposure to indoor air contaminants

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<<READ SLIDE>>

Indoor Air Pollution				
OUTLINE				
□ Scope of the problem of indoor air pollution □ Particulate matter □ Carbon monoxide □ Secondhand tobacco smoke				
□ Pesticides □ Solvents □ Volatile organic compounds □ Biological pollutants - Mites				
- Allergens - Moulds - Built environment - Radon - Asbestos - Occupation-related contaminants				

<<READ SLIDE>>

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The indoor contaminants addressed in this module include:

- Particulate matter
- •Carbon monoxide
- ·Secondhand tobacco smoke
- Pesticides
- Solvents
- •Volatile organic compounds
- •Biological pollutants
 - Mites
 - Allergens
 - Moulds
- •Built environment
- •Radon
- Asbestos
- •Occupation-related contaminants

CHILDREN'S UNIQUE VULNERABILITY

- Inhale more pollutants per kilogram of body weight than do adults
- Because airways are narrower, irritation can result in proportionately greater airway obstruction



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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.
- •American Academy of Pediatrics Committee on Environmental Health. *Pediatric Environmental Health, 2nd ed.* Etzel RA, Ed. Elk Grove Village, IL: American Academy of Pediatrics, 2003.
- Children's Health and the Environment A global perspective. A resource guide for the health sector, WHO, 2005.

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SMALLER AIRWAYS MORE VULNERABLE

Diagram of the Effect of Edema on the Cross-Sectional Airway Diameter (R = radius)

Adult Airway



Area =
$$\text{Tr R}^2$$
 = Tr 10^2 = 100 Tr mm^2 (Normal)
If have 1 mm Edema Area = Tr 9^2 = 81 Tr mm^2
or 81% of normal

Full Term Newborn



Area =
$$\eta \hat{R} = \eta \hat{3} = 9 \eta \hat{m} \text{ mm}^2 \text{(Normal)}$$

If have 1 mm Edema Area = $\eta \hat{2} = 4 \eta \hat{m} \hat{m}^2$
or 44% of normal

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

5

The effect of oedema on the adult airway is much less dramatic than it is on the newborn's airway. One millimetre of oedema reduces the diameter of the adult airway by about 19% whereas 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).

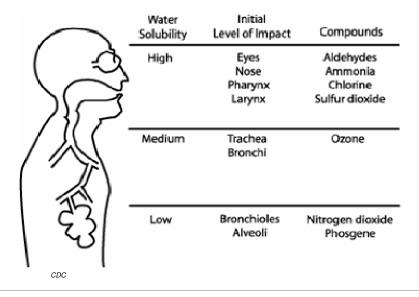
Ref:

•Bar-on ME et al. Bronchiolitis. Prim Care, 1996, 23:805-19.

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

DEPOSITION OF POLLUTANTS IN RESPIRATORY TRACT

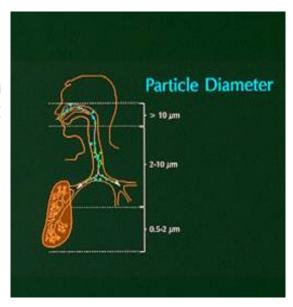


Respirable particles and gases affect different parts of the respiratory tree depending upon their inherent characteristics. For gases, relative solubility is important. For particles, size is important.

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.

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</p>



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

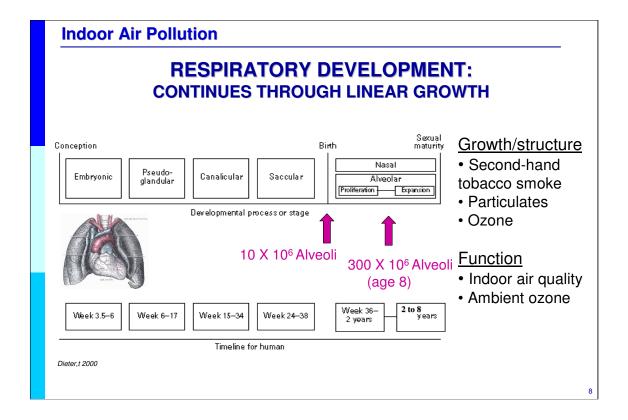
Compared with large particles, fine particles can remain suspended in the atmosphere for longer periods and be transported over longer distances.

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.

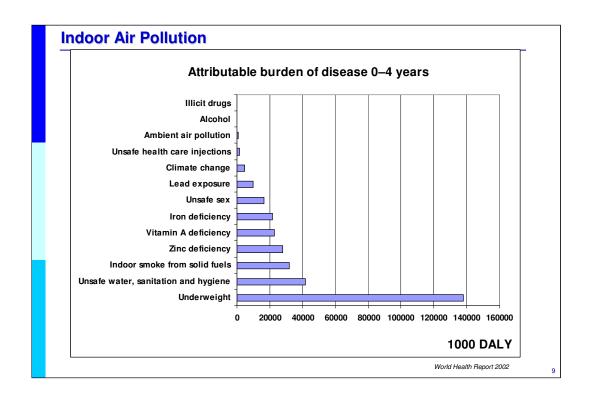
Ref:

•World Health Organization. *Air Quality Guidelines*. Geneva, World Health Organization: Department of Protection of the Human Environment, 2005.



Like the nervous system, the respiratory system continues to grow and develop through linear growth. The upper section of the diagram depicts the different developmental phases of the lungs corresponding to the age of the embryo/fetus. It may be seen that at birth, a baby has about 10 million alveoli, but at age 8 years, the lungs have grown and the number of alveoli has reached 300 million. Exposures during this growth period are known to have adverse consequences on both structure (growth of the lungs, as illustrated in the diagram) and function (which is affected by indoor air quality and ozone exposure).

Figure: Dietert RR et al. Workshop to identify critical windows of exposure for children's health: immune and respiratory systems – work group summary. Environmental Health Perspectives, 2000, 108:483-90. Reproduced with permission from Environmental Health Perspectives



In analyses by the World Health Organization (WHO) in 2002, the indoor smoke from solid fuels accounted for the third highest disability-adjusted life years (DALYs) for children 0 to 4 years of age.

The DALY is a health measure that incorporates loss of quality of life as well as loss of years of life. One DALY is the loss of one healthy life year.

Ref:

• World Health Report 2002 (www.who.int/whr/2002/en/).

Picture: World Health Report 2002

CHILDREN'S INDOOR EXPOSURE

Level of economic development is a key factor

- Developing and industrialized countries
- Rural and urban areas
- Local climate
- Architecture/ventilation

In urban areas, children may spend most of their time indoors.



Most exposure to air contaminants occurs inside homes and schools.

<<READ SLIDE>>

The level of economic development is a key factor in determining children's exposures and the potential for responding to or improving their environment.

The level of social and economic development is linked closely to determinants of indoor air pollution (IAP).

There are major differences between developing and industrialized countries: IAP results from solid fuel use in the former, and from "chemicals" and "new substances" (e.g. formaldehyde, insecticides and phthalates) in the latter. However, secondhand tobacco smoke is a pollutant common to both settings.

IAP also differs between rural and urban areas due to the different economies and lifestyles. For example, dust and organic particles are more common in agricultural areas and mites or fungal contaminants in closed, unventilated urban dwellings.

The local climate conditions should also be taken into consideration, as they have an impact on architecture (building materials used, structure, room distribution and characteristics) and - particularly - on the ventilation of the dwelling.

Children in urban areas spend most of their time indoors, which means that their primary exposure to air pollution may come from air inside homes and schools rather than outdoors.

There are numerous situations in homes and schools which may result in possible exposure to contaminants, such as second-hand tobacco smoke, spraying of insecticides, accumulation of pollutants in carpets, poor quality air and others. Children may also be exposed where they play or at workplaces. The quality of children's environments can cause or prevent illness, disability and injury.

Picture: WHO.

INDOOR AIR QUALITY

Indoor air quality is influenced by:

- Outdoor air pollution: vehicles and industrial plants
- Secondhand tobacco smoke
- Fuels used for heating and cooking
- Confined and poorly ventilated spaces
- Overcrowded homes and insufficient living space
- Customs, habits, traditions
- Level of economic development: Industrialized ≠ developing countries

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Indoor air quality is influenced by concentrations of outdoor air pollutants, indoor sources of pollution, characteristics of the building and the habits of the residents.

Indoor air pollution may arise from the use of open fires, unsafe fuels or combustion of biomass fuels, coal and kerosene. Gas stoves or badly installed wood-burning units with poor ventilation and maintenance can increase the indoor levels of carbon monoxide, nitrogen dioxide and particles.

Other pollutants not associated with fuel combustion include building materials such as asbestos and cement, wood preservatives and others.

Volatile organic compounds may be released by various sources including paints, glues, resins, polishing materials, perfumes, spray propellants and cleaning agents. Formaldehyde is a component of some household products and can irritate the eyes, nose and airways.

HOMES OF POOR CHILDREN MAY BE UNHEALTHY



- 2 000 000 deaths from ARI in
 5 yr olds (½ due to solid fuel use)
- Rising trends of "wheezing"

WH

- * Coal and biomass fuel: a major source of indoor air pollution
- Suspended particulate matter increases the risk of acute respiratory infections
- * CO and other toxic gases may impair development and health
- Secondhand tobacco smoke a major concern

-1

The home is the first indoor environment a child will know. It should be a safe and healthy place. But the homes of poor children may be unhealthy places.

ARI = acute respiratory illness

CO = carbon monoxide

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Picture: WHO, C. Gaggero. Child housework, Costa Rica.

ADVERSE HEALTH EFFECTS OF AIR POLLUTANTS

Acute:

- Irritation of the mucous membranes (eyes, nose, throat)
- Cough, wheeze, chest tightness
- Increased airway responsiveness to allergens
- Increased incidence of acute respiratory illness:
 - "cold", pneumonia, otitis media
- Tracheobronchitis
- Exacerbation of asthma

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ADVERSE HEALTH EFFECTS OF AIR POLLUTANTS

Chronic:

- Long-term exposure decreases lung growth
- Impairment of pulmonary function
- Increased susceptibility to chronic obstructive lung diseases, including asthma
- Other

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ENVIRONMENT AND POLLUTION

Indoor environments also reflect outdoor air quality

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



WHO

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The indoor environment also reflects outdoor air quality and pollution. 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).

Picture: WHO, J. Vizcarra. Environmental Air Pollution

INDOOR AIR POLLUTION ALSO AFFECTS OUTDOOR AIR QUALITY



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

Picture: Nigel Bruce/ITDG. Used with permission.

COMBUSTION PRODUCTS

Sources

Gas stoves and appliances

Wood and coal stoves

Gas and propane engines

Fireplaces

Tobacco smoke

Candles and incense

Mosquito coils

Combustion products

Carbon monoxide (CO)

Nitrogen dioxide (NO₂)

Sulfur dioxide (SO₂)

Nitrogenated compounds (NO_x)

Particulate matter (PM)

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A large number of combustion products originate from various different sources. The main ones are listed here.

<<READ SLIDE.>>

SMOKY COOKING FUELS

Open fire cooking stoves produce heavy smoke containing:

- Fine particles
- Carbon monoxide (CO)
- Polycyclic aromatic hydrocarbons (PAHs)
- Strongly linked to pneumonia
- Suggested link to low birth weight
- In adults: chronic obstructive pulmonary disease, lung cancer



MILIO

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Girls are at most risk as they are often requested to help their mothers with household chores. Infants are exposed to pollutants when carried on the backs of their mothers as they tend fires. Irritation that would not affect adults may result in severe obstruction or damage to children's lungs because they are more vulnerable.

Ref:

- •Environmental threats to children. In: *Children in the New Millennium, Environmental Impact on Health.* UNEP, UNICEF & WHO, 2002.
- •Smith KR et al. Chapter 18: Indoor smoke from household use of solid fuels. In: Ezzati Eds. Comparative quantification of health risks: The global burden of disease due to selected risk factors, Vol 2. Geneva: World Health Organization, 2004.

HOUSEHOLD ENERGY, IAP AND HEALTH

- ❖ 3 billion people rely on solid fuels
- ❖ Solid fuels comprise 10--15% of the total fuels used worldwide
- Cooking and heating levels indoor air pollution
- * Most concern: particulate matter and carbon monoxide
- Women and children heavily exposed

Dung Wood **Agricultural** residues Charcoal Coal



Nigel Bruce/ITDG

Some 3 billion people (half of the world's population!) rely on solid fuels (e.g. dung, wood, agricultural residues, charcoal, coal) for their basic energy needs. Cooking and heating with solid fuels leads to high levels of indoor air pollution (IAP), a complex mix of health-damaging pollutants (e.g. particulate matter and carbon monoxide). Women and young children, who spend most time at home, experience the largest exposures and health burdens.

IAP = indoor air pollution

Picture: Nigel Bruce/ITDG. Used with permission.

INDOOR AIR POLLUTION LEVELS ARE VERY HIGH



- Level of particulates in home using biofuel: >1000 µg/m³ (24 hr mean)
- Can reach 10 000 µg/m³ PM₁₀ (if using an open fire)
- ❖ EPA: 50 µg/m³ PM₁₀ annual mean
- Women and young children have greatest exposure

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Standards and guidelines

US EPA standards are illustrated here. 150 $\mu g/m^3$ PM₁₀ is the 24-hour 99% percentile value, thus it should be exceeded only on 1% of occasions. The recommended annual mean limit is 50 $\mu g/m^3$ PM₁₀ (PM₁₀ are respirable particles \leq 10 micrometre (μm) in diameter).

Levels of pollution in homes using biomass fuel

Numerous studies have shown that the levels of particulates are very high, with 24-hour means of around 1000 $\mu g/m^3$ PM₁₀, and even exceeding 10 000 $\mu g/m^3$ PM₁₀ when sampling is carried out during use of an open fire. It is reasonable to compare the EPA recommended annual mean limit of 50 $\mu g/m^3$ PM₁₀ with the typical 24-hour mean for a home in which biomass fuel is used, of 1000 $\mu g/m^3$ PM₁₀ quoted, as this latter value is typical of the level every day (thus, annual mean levels can be expected to be around 1000 $\mu g/m^3$ PM₁₀). This comparison shows that average pollution levels are around 20 times the EPA recommended limit.

Ambient pollution and personal exposure

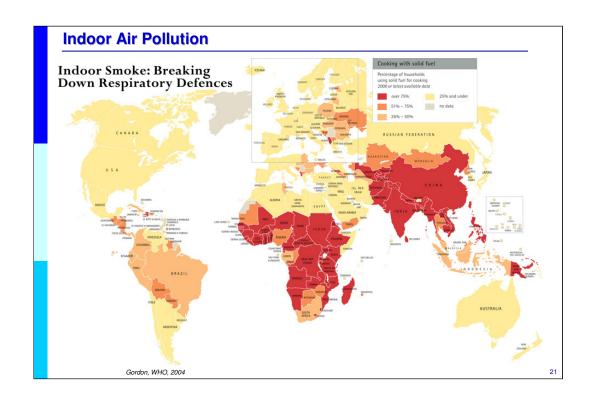
Two important components are (a) the level in the home, and (b) the length of time for which each person in the home is exposed to that level. We know that typically women and young children (until they can walk), and girls (as they learn kitchen skills) are often exposed for at least 3–5 hours a day, often more. In some communities, and where it is cold, exposure will be for a much longer period each day.

Ref:

•Addressing the links between indoor air pollution, household energy and human health. Based on the WHO-USAID Consultation on the Health Impact of Household Energy in Developing Countries (Meeting report). Geneva, World Health Organization, 2002.

Additional information can be found at: www.who.int/indoorair/publications/en/

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



Cooking is central to our lives, yet the very act of cooking is a threat to children's health and well-being. Half of the world's population relies on solid fuels, such as dung, wood, crop waste or coal to meet their most basic energy needs. In most developing countries, these fuels are burned in open fires or rudimentary stoves that give off black smoke. Children, often carried on their mother's back during cooking, are most exposed. The indoor smoke inhaled leads to pneumonia and other respiratory infections – the biggest killer of children under 5 years of age. Indoor air pollution is responsible for nearly half of the more than 2 million deaths each year that are caused by acute respiratory infections. Good ventilation and improved cooking stoves can dramatically reduce children's exposure to smoke. Ultimately, making the transition to gas and electricity will save lives and reduce the physical toll on women and children from gathering wood, freeing time for education and development. This problem has been largely ignored by policy-makers.

Ref:

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

AGE DISTRIBUTION OF GLOBAL BURDEN OF DISEASE ATTRIBUTABLE TO SOLID FUEL USE

Age group (yrs)	Attributable mortality	Attributable DALYs
0-4	56	4 83
5-14	0	0
15-59	5	8
60+	38	9

Young children at high risk due to:

Lung immaturity

High exposure to IAP: nearly 1 million children under 5 yrs die every year due to solid fuel use!



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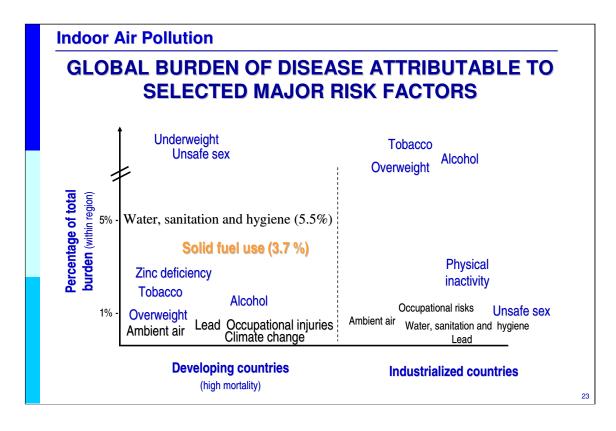
DALY = disability-adjusted life year. The DALY is a health measure that incorporates loss of quality of life as well as loss of years of life. One DALY is the loss of one healthy life year. IAP = indoor air pollution

Ref:

- The health effects of indoor air pollution exposure in developing countries. Concise summary of the evidence for health effects of exposure to indoor air pollution from solid fuel use in children and adults. Geneva, World Health Organization, 2002.
- •Indoor air pollution: national burden of disease estimates. Geneva, World Health Organization, 2007.
- •Fuel for life: household energy and health. Geneva, World Health Organization, 2006.
- •Indoor air pollution from solid fuels and risk of low birthweight and stillbirth. Geneva, World Health Organization, 2007.
- •Indoor air pollution and respiratory tract infections in children. Geneva, World Health Organization, 2007.

Additional information on these references can be found at: www.who.int/indoorair/publications/en/

Picture: WHO. Americas.



Household energy practices vary widely around the world, as does the resultant death toll due to indoor smoke. In high-mortality developing countries, indoor smoke is responsible for 3.7% of the overall disease burden, making it the most important risk factor after malnutrition (9%), unsafe sex (6%) and lack of safe water and adequate sanitation. In low-mortality developing countries, indoor smoke occupies the 8th rank and accounts for 1.9% of the disease burden. In contrast, in industrialized countries the impact of cooking and heating with solid fuels becomes negligible in relation to risk factors such as tobacco, high blood pressure and alcohol consumption. *Notes taken from www.who.int/indoorair/info/en/briefing2.pdf*

Refs:

- •Indoor air pollution in developing countries: a major environmental and public health challenge. Bulletin of the World Health Organization, 2000.
- World Health Report 2002. Indoor Air Thematic Briefing 2 (www.who.int/indoorair/info/en/briefing2.pdf).

Additional information can be found at: www.who.int/indoorair/publications/en/

WHAT INTERVENTIONS ARE AVAILABLE TO REDUCE INDOOR AIR POLLUTION FROM SOLID FUELS?

Source of pollution	Home environment	User behaviour
- Improved stoves - Cleaner fuels (kerosene, gas, electricity)	-Hoods and chimneys -Windows, ventilation holes, eaves spaces -Separate kitchen	 Fuel drying Use of pot lids Good maintenance Keeping children away from smoke

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<<READ SLIDE>>

- ❖Solid fuels comprise only 10–15% of fuel used.
- •Nearly one half of the world's population uses solid fuels for cooking and heating homes.
- 2 billion people are exposed to particulate matter (PM) and gases at levels up to 100 times higher than in ambient (outdoor) air.
- Women and children are most exposed: levels may be 10 to 100 times above safety standards for ambient air.
- ${\color{red} \bullet } \text{Combustion produces hundreds of toxic chemicals that concentrate inside homes}. \\$

Biomass (wood, agricultural produce, straw and dung) produces:

- a wide variety of liquids, suspended particles, gases and mixtures.

Coal produces:

- Polycyclic aromatic hydrocarbons (PAHs), benzene, formaldehyde, sulfur, heavy metals and fluoride.

These pollutants affect the most vulnerable populations, women of childbearing age, infants and children in the poorest circumstances.

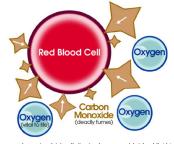
Ref:

Air pollution: what a paediatrician needs to know. Leaflet published by the World Health Organization in collaboration with the International Pediatric Association, 2003.

Additional information on these references can be found at: www.who.int/indoorair/publications/en/

CARBON MONOXIDE: THE "SILENT KILLER" A COMMON CAUSE OF ACUTE AND LETHAL POISONING

- Colourless, odourless gas formed by incomplete burning of carbon-based fuels
- CO's affinity for Hb is 240–270 times greater than oxygen
- Fetal Hb has higher affinity for CO
- CO causes a leftward shift of the oxyhaemoglobin (OHb) dissociation curve
- Intoxication results in tissue hypoxia
- Multiple organ systems are affected



t.htm

- CO is a colourless, odourless gas formed by incomplete burning of carbon-based fuels.
- CO's affinity for haemoglobin (Hb) is 240–270 times greater than that of oxygen:
 - it decreases the capacity of Hb for carrying oxygen.
- Fetal Hb has a higher affinity for CO.
- CO causes a leftward shift of the oxyhaemoglobin dissociation curve:
 - it decreases oxygen delivery to tissues.
- · Intoxication results in tissue hypoxia.
- · Multiple organ systems are affected:
 - Mainly systems with high metabolic rates;
 - CNS, cardiovascular system.

Exposure to carbon monoxide reduces the blood's ability to carry oxygen. The chemical is odourless and some of the symptoms of exposure are similar to those of common illnesses. This is particularly dangerous because carbon monoxide's deadly effects may not be recognized until it is too late to take action.

Exposure to carbon monoxide is particularly dangerous to unborn babies, infants and people with anaemia or a history of heart disease. Breathing low levels of the chemical can cause fatigue and increase chest pain in people with chronic heart disease. Breathing higher levels of carbon monoxide causes symptoms such as headaches, dizziness and weakness in healthy people. Carbon monoxide also causes sleepiness, nausea, vomiting, confusion and disorientation. At very high levels it causes loss of consciousness and death. Poisoning may have irreversible sequelae.

These notes are taken from the US EPA website www.epa.gov/iag/co.html

Hb = haemoglobin COHb = carboxyhaemoglobin

Refs:

1013.

CARBON MONOXIDE (CO): SOURCES

- Gas, kerosene, wood stoves and coal
- Fires, fireplaces, furnaces
- Leaking chimneys and vents
- Room and water heaters
- Vehicle exhaust in closed garage
- Tobacco smoke



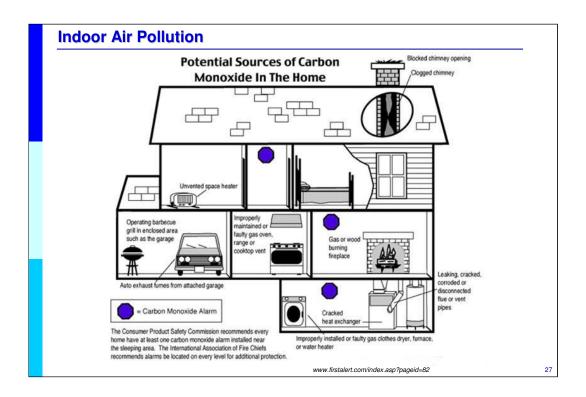
EPA

Any place where combustion is incomplete

2

Incomplete oxidation during combustion in gas ranges and unvented gas or kerosene heaters may cause high concentrations of CO in indoor air. Worn or poorly adjusted and maintained combustion devices (e.g. boilers and furnaces) can be significant sources, especially if the fuel is of an unsuitable size, or if the system is blocked, or leaking. Car, truck, or bus exhaust from attached garages, nearby roads, or parking areas can also be a source. CO is one of the components of secondhand tobacco smoke.

Picture: www.epa.nsw.gov.au/woodsmoke/heateruse.htm



In a a single-family house in the <u>industrialized</u> world, CO can come from many sources. Figure: www.firstalert.com/index.asp?pageid=82, Used with Copyright permission.

PREVENTION OF EXPOSURE TO CO

- Keep fuel-burning appliances in good working condition
- Check heating systems, chimneys and vents regularly
- Never burn charcoal indoors
- Never leave a car running in a closed garage
- Consider CO detectors

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Prevention is the key to avoiding carbon monoxide poisoning.

Primary prevention of carbon monoxide (CO) poisoning requires limiting exposure to known sources. Proper installation, maintenance, and use of combustion appliances can help to reduce exposure to CO.

The US Environmental Protection Agency (EPA) has set harm levels of:

- •50 ppm (8-hour average)
- •75 ppm (4-hour average)
- •125 ppm (1-hour average).

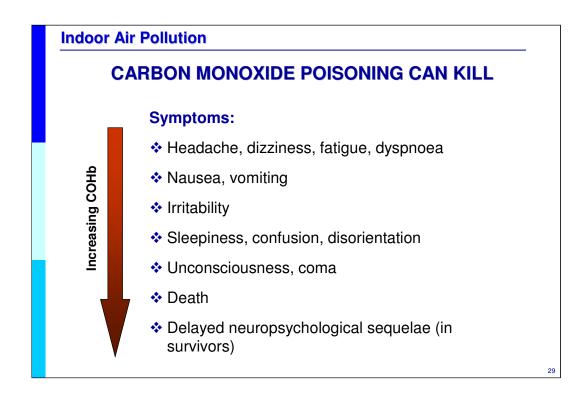
Exposure to these levels can lead to COHb levels of 5 to 10% and cause significant symptoms in sensitive individuals. The current US air quality standards for CO, intended to keep COHb below 2.1%, recommend levels of not more than 9 parts per million (ppm) for 8 hours and 35 ppm for 1 hour for outdoor air. No standards for CO have been agreed for indoor air.

Smoke and CO detectors may provide early warning and prevent unintentional CO-related deaths. They should, however, be in good working condition and should not substitute for other prevention measures (cleaning the chimney, etc.)

For more information on CO detectors, go to: www.epa.gov/iaq/pubs/coftsht.html COHb = carboxyhaemoglobin

Ref:

www.pinefire-85.org/Fire_safety_pages/CARBON_MONOXIDE/carbon_monoxide.html



The route of exposure is through inhalation. Unintentional exposure to carbon monoxide (CO) can be attributed to smoke inhalation from inadequately vented combustion appliances, and from vehicles and tobacco smoke.

The clinical features of CO poisoning are highly variable and symptoms vary from mild to very severe. Acute effects are due to the formation of carboxyhaemoglobin in the blood, which inhibits oxygen intake. At moderate concentrations, angina, impaired vision, and reduced brain function may result. At higher concentrations, exposure to CO can be fatal. Delayed neuropsychological sequelae have been reported in adults and children; these usually occur 3 to 240 days following exposure and are estimated to occur in 10 to 30% of victims.

Ref:

- California Poison Control System. Poisoning and drug overdose. Olson ed. Appleton and Lange, 1999.
- •Carbon monoxide. In: Pediatric Environmental Health, 2nd ed. Etzel RA Ed. Elk Grove Village, IL: American Academy of Pediatrics. 2003.
- •Hon KLet al. Neurologic and radiologic manifestations of three girls surviving acute carbon monoxide poisoning. J Child Neurol. 2006 Sep;21(9):737-41.

We report the neurologic and radiologic manifestations of three adolescent girls with acute carbon monoxide poisoning. The girls were found collapsed and unconscious in a bathroom where liquid petroleum gas was being used as heating fuel. As hyperbaric oxygen therapy was not available locally, they only received oxygen supplementation via nasal cannula (4 L/minute) as treatment in the first 2 days. On transfer to a tertiary center in Hong Kong, evolving neurologic manifestations of visual acuity and field deficits, confusion, and focal motor weaknesses were observed. Focal infarctions were evident in cerebral computed tomography in one patient and cortical lesions on magnetic resonance imaging in all three patients. [18F]Fluorodeoxyglucose (FDG) positron emission tomography (PET) revealed additional decreased metabolism in the basal ganglia in two patients, which was typical of carbon monoxide poisoning. The neurologic deficits resolved completely at

3 weaks after the exposure but psychologic symptoms succeeded. This report

MANAGEMENT

Diagnosis:

- Measurement of COHb
 - nonsmokers 1–3 %
 - smokers 3–8%
- Not useful:
 - Pulse oximeter
 - Arterial blood gases

Treatment:

- Remove patient from CO source
- Life support
- ❖ Oxygen 100%
- Hyperbaric oxygen?
- Monitoring

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The measurement of COHb confirms that exposure has occurred, but the severity of poisoning is not correlated to COHb levels. Measurements of oxygen saturation by pulse oxymetry and arterial blood gas are not helpful for diagnosis because they are normal, although metabolic acidosis may be present. Normal levels of COHb range from 1 to 3% in nonsmokers and 3 to 8% in smokers.

Blood tests have to be done as soon as possible after exposure. The gas company can also complement the measurements.

Treatment of poisoning consists of supplemental oxygen, 100%, ventilatory support and monitoring cardiac disrhythmias. Elimination half-life of COHb is approximately 4 hours in room air, 1 hour with provision of oxygen, 100%, and 20–30 minutes with hyperbaric oxygen.

Hyperbaric oxygen is a treatment that is usually reserved for severe CO poisoning.

COHb = carboxyhaemoglobin

CHRONIC EXPOSURE

Acute or chronic exposure to low levels of carbon monoxide (research in animals and humans):

- Linked to development of arteriosclerosis
- ?Aggravation of cardiovascular diseases
- ?Poor performance on certain psychomotor tasks
- Limited exercise capacity

3

The main areas of concern that have arisen from acute or chronic exposure to low levels of carbon monoxide in experimental and epidemiological research in animals and man are: (a) its role in the genesis of arteriosclerotic vascular diseases; (b) its role in the aggravation of symptoms of cardiovascular diseases; (c) its contribution to performance deficits in certain psychomotor tasks; and (d) its role in limiting the working capacity of exercising man

Cardiovascular system: Development of atherosclerotic cardiovascular disease

Extensive experimental work has been carried out over many years on animals, mainly rabbits, showing that prolonged exposure to moderate levels of carbon monoxide can produce atherosclerotic changes, especially in the presence of high cholesterol levels (1-2%) in the diet. The relevance of this work for man has not been established. However, other animal work, and some epidemiologic studies of prolonged human exposures to elevated carbon monoxide levels through smoking, occupation, or both, such as those carried out in Denmark, Finland, and Japan, indicate the need for further investigation of the possible role of carbon monoxide in the genesis of atherosclerotic vascular changes in animals and man. The degree of intermittency of exposure at various levels should be taken into account as well as the possible contribution of other agents such as nicotine and high-fat diets. There is some evidence of adaptation, but such changes may not be entirely beneficial.

Acute effects on existing heart illness

The few existing epidemiologic studies on the possible effects of carbon monoxide on the severity or fatality of coronary occlusion are insufficient to allow any conclusions.

Acute effects on existing vascular disease

One study has been carried out on patients with intermittent claudication from peripheral vascular disease. Effects on pain with exercise were observed in the same exposure range as with angina i.e., at carboxyhaemoglobin concentrations of 2.5-3.1%, with a mean of 2.8%.

Nervous system

As for the role of carbon monoxide in affecting psychomotor functions, no definite conclusions can be drawn from the existing data. The behavioural functions tested in such studies include vigilance and psychomotor performance, visual acuity and sensitivity, the ability to estimate time intervals, complex motor coordination as tested by driving simulators, and different perceptual and mental operations. Some workers observed detrimental effects at carboxyhaemoglobin levels as low as 2%, whereas others were unable to detect significant impairment even at levels from above 5% to about 20%. In evaluating these discrepancies, it should be mentioned, that these behavioural functions are easily influenced by a number of other factors besides carbon monoxide-induced hypoxia, e.g., degree of sensory deprivation, compensatory abilities, drugs, temperature, time of day, competition, etc.

Work capacity

That elevated carboxyhaemoglobin levels affect work capacity has long been known. Levels of 40-50% will usually prevent working entirely. Recent studies in the laboratory, on man, using maximum work capacity or maximum aerobic capacity as indicators of performance, have been carried out in relation to carboxyhaemoglobin levels. Here, dose-response data are available for maximum effort. The limitation appears at a carboxyhaemoglobin concentration of about 4% and increases at higher levels. Lower exposure levels have been studied and do not produce this effect. It should be noted that while levels of carboxyhaemoglobin of 2.5-4%, did not reduce maximum work capacity, they did reduce the length of time for which such effort could be carried out. It is not known what specific levels of carboxy- haemoglobin will reduce the capacity of individuals to perform at ordinary work levels, such as 30-50% of their maximum capacity, for prolonged periods of time.

Notes from IPCS INCHEM: www.inchem.org/documents/ehc/ehc/ehc013.htm#SubSectionNumber:9.2.1

CHRONIC EXPOSURE

Can lead mainly to:

- Cardiovascular effects.
- Central nervous system effects
- Increased susceptibility to acute respiratory infections

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Carbon monoxide can cause harmful health effects by reducing oxygen delivery to the body's organs (like the heart and brain) and tissues.

Cardiovascular Effects. The health threat from lower levels of CO is most serious for those who suffer from heart disease, like angina, clogged arteries, or congestive heart failure. For a person with heart disease, a single exposure to CO at low levels may cause chest pain and reduce that person's ability to exercise; repeated exposures may contribute to other cardiovascular effects.

Central Nervous System Effects. Even healthy people can be affected by high levels of CO. People who breathe high levels of CO can develop vision problems, reduced ability to work or learn, reduced manual dexterity, and difficulty performing complex tasks. At extremely high levels, CO is poisonous and can cause death.

Notes taken from EPA: www.epa.gov/air/urbanair/co/hlth1.html

Estrella B et al. Acute respiratory diseases and carboxyhemoglobin status in school children of Quito, Ecuador. Environmental Health Perspectives (2005) 113 (5): 607-11.

Outdoor carbon monoxide comes mainly from vehicular emissions, and high concentrations occur in areas with heavy traffic congestion. CO binds to hemoglobin, forming carboxyhemoglobin (COHb), and reduces oxygen delivery. We investigated the link between the adverse effects of CO on the respiratory system using COHb as a marker for chronic CO exposure. We examined the relationship between acute respiratory infections (ARIs) and COHb concentrations in school-age children living in urban and suburban areas of Quito, Ecuador. We selected three schools located in areas with different traffic intensities and enrolled 960 children. To adjust for potential confounders we conducted a detailed survey. In a random subsample of 295 children, we determined that average COHb concentrations were significantly higher in children attending schools in areas with high and moderate traffic, compared with the low-traffic area. The percentage of children with COHb concentrations above the safe level of 2.5% were 1, 43, and 92% in low-, moderate-, and high-traffic areas, respectively. Children with COHb above the safe level are 3.25 [95% confidence interval (CI), 1.65-6.38] times more likely to have ARI than children with COHb above the safety level, children are 1.15 (95% CI, 1.03-1.28) times more likely to have an additional case of ARI. Our findings provide strong evidence of the relation between CO exposure and susceptibility to respiratory infections.

FIRE INJURIES

- From inhaled toxic chemicals and/or thermal burns.
- The lungs and airways are affected in these ways:
 - tissue irritation
 - heat damage
 - Asphyxiation
 - potential cyanide poisoning

Make the home safer:

- Beware of matches and lighters
- Install smoke alarms
- Have a home fire escape plan

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Fire injuries can result from inhaled toxic chemicals and/or thermal burns.

Smoke inhalation means breathing in the harmful gases, vapors, and particulate matter contained in smoke. Smoke inhalation impairs the body from acquiring oxygen from the environment and its ability to deliver and use oxygen at every step of respiration. Those caught in fires may suffer from smoke inhalation whether or not they present skin burns. However, the incidence of smoke inhalation increases with the percentage of total body surface area burned. The lungs and airways are affected in three ways: heat damage, tissue irritation, and oxygen starvation of tissues (asphyxiation).

How to make the home safer:

- 1) Beware of matches and lighters around the house.
- -Store them out of reach and sight.
- -Teach toddlers to tell you when they find one and explain to them that these tools are only for adults.
- -Never use them as an amusement. Children may imitate you.
- -Practice and teach fire safe behaviours in your home: keep small children away from stoves when cooking, have your heating systems checked annually, use deep ashtrays and soak ashes in water if you are a smoker (or better: stop smoking!).
- -Install smoke alarms

Prepare a home fire escape plan.

- -Draw a basic diagram of the house and mark all exits.
- -Consider different fire scenarios and develop different escape plans.
- -When escaping, crawl low under the smoke. Touch doors before opening: if they are hot, use an alternative route.
- -Teach children NEVER to go back inside the house
- -Practice the fire escape plans and teach children how to cover their nose and mouth to reduce smoke inhalation.
- -If there are babies and toddlers: keep a harness by the crib to be able to carry the baby and keep hands free at the same time. Keep the child's bedroom closed.

Ref

•FEMA, A fact sheet on fire safety for babies and toddlers: usfa.fema.gov/downloads/usfaparents/508/USFA FireFacts 508.pdf

SECONDHAND TOBACCO SMOKE SCOPE OF THE PROBLEM

Children whose mothers smoke:

- 70% more respiratory problems
- ❖ Pneumonia and hospitalization in year 1 is 38% higher
- ❖ Infant mortality is 80% higher
- 20% of all infant deaths could be avoided if all pregnant smokers stopped by the 16th week of gestation
- ❖ 5 times higher risk of sudden infant death syndrome (SIDS)

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- *Children whose mothers smoke have an estimated 70% more respiratory problems than children whose mothers do not smoke.
- Pneumonia and hospitalization in the first year of life is 38% more frequent in children whose mothers smokes.
- ❖Infant mortality was 80% higher in children born to women who smoked during pregnancy than in children of nonsmokers.
- ❖An estimated 20% of all infant deaths could be avoided if all pregnant smokers stopped by the 16th week of gestation.
- Infants of mothers who smoke have an almost 5 times higher risk of sudden infant death syndrome (SIDS) than infants of mothers who do not smoke.

Smoke released from cigarettes, cigars and pipes is composed of more than 3800 different substances. Airborne particulate matter is 2–3 times higher in homes of smokers. Exposure may occur at home, school, in child care settings, in relatives homes and other places. The importance of the need to reduce exposure to second-hand smoke justifies prohibiting smoking at home, in schools and in child care facilities.

SHTS is covered extensively in a separate module.

Refs:

- •Etzel RA. Indoor air pollutants in homes and schools. Pediatric Clinics of North America, 2001, 48:1153.
- •Wisborg K et al. Exposure to tobacco smoke in utero and the risk of stillbirth and death in the first year of life. American Journal of Epidemiology, 2001, 154:322-7.

The authors examined the association between exposure to tobacco smoke in utero and the risk of stillbirth and infant death in a cohort of 25,102 singleton children of pregnant women scheduled to deliver at Aarhus University Hospital, Aarhus, Denmark, from September 1989 to August 1996. Exposure to tobacco smoke in utero was associated with an increased risk of stillbirth (odds ratio = 2.0, 95% confidence interval: 1.4, 2.9), and infant mortality was almost doubled in children born to women who had smoked during pregnancy compared with children of nonsmokers (odds ratio = 1.8, 95% confidence interval: 1.3, 2.6). Among children of women who stopped smoking during the first trimester, stillbirth and infant mortality was comparable with that in children of women who had been nonsmokers from the beginning of pregnancy. Conclusions were not changed after adjustment in a logistic regression model for the sex of the child; parity; or maternal age, height, weight, marital status, years of education, occupational status, and alcohol and caffeine intake during pregnancy. Approximately 25% of all stillbirths and 20% of all infant deaths in a population with 30% pregnant smokers could be avoided if all pregnant smokers stopped smoking by the sixteenth week of gestation.

SECONDHAND SMOKE INCREASES RISK OF:

- Respiratory tract illness
- ❖ Asthma
- Middle ear effusions
- Prenatal complications and low birth weight
- Fire-related injuries
- Sudden infant death syndrome (SIDS)
- Cancers (childhood leukemia and others)

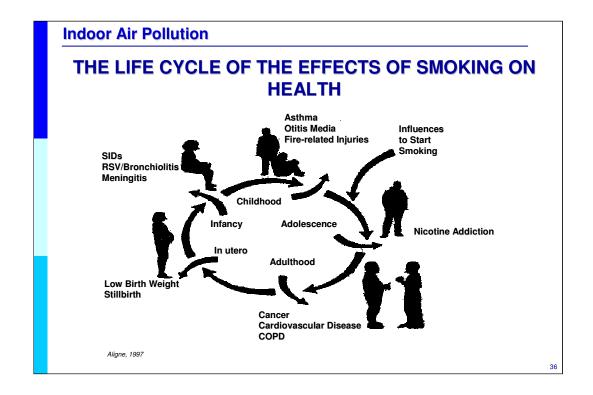
35

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Sudden infant death syndrome (SIDS): death of an apparently healthy infant, usually before the age of 1 year, that is of unknown cause and occurs especially during sleep.

Refs:

- •Brondum J et al. Parental Cigarette Smoking and the Risk of Acute Leukemia in Children. Cancer. 1999; 85:1380-1388.
- •Ji BT et al. Paternal Cigarette Smoking and the Risk of Childhood Cancer Among Offspring of Non-Smoking Mothers. J Natl Cancer Inst. 1997; 89:238-244.
- •Shu XO et al. Parental Alcohol Consumption, Cigarette Smoking, and Risk of Infant Leukemia: A Children's Cancer Group Study. J Natl Cancer Inst. 1996; 88:24-31.



This graphic depicts the life-cycle of the effects of tobacco smoking on health beginning *in utero* and continuing throughout adulthood. Pregnant women will have babies with lower birth weight as well as greater chances of stillbirth.

Children with parents who smoke will be more to likely develop respiratory problems, bronchiolitis, meningitis, asthma and otitis media and are at a higher risk of fire-related injuries. Furthermore, exposure to tobacco smoke damages the respiratory epithelium and decreases the ability to combat the respiratory syncytial virus (RSV), the leading cause of hospital admissions of children under 1 year of age.

Adolescence represents a high-risk period for taking up smoking behaviour.

As adults, children of smokers have a greater likelihood of developing cancer, chronic obstructive pulmonary disease (COPD) and cardiovascular diseases (CVD) than children with non-smoking parents. Also, children who have a parent who smokes are more likely to smoke as adults, so the cycle continues from one generation to the next.

SIDS: sudden infant death syndrome

COPD: chronic obstructive pulmonary disease

Figure: Aligne A et al. Tobacco and children: An economic evaluation of the medical effects of parental smoking. Arch Pediatr Adolesc Med, 1997, 151:652. Copyright (1997), American Medical Association. Used with permission.

Ref:

- Aligne A et al. Tobacco and children: An economic evaluation of the medical effects of parental smoking. Arch Pediatr Adolesc Med, 1997, 151:652.
- •Tager IB et al. Longitudinal study of the effects of maternal smoking on pulmonary function in children. N Engl J Med, 1983, 309:699-703.
- •WHO Report on the Global Tobacco Epidemic 2008: The MPOWER Package, 2008.

PESTICIDES

- Spraying pesticides at home / school:
 - Higher concentrations near the floor
 - Persistence in some surfaces (carpets, soft toys)
 - Overuse and misuse
- Children's behaviour and inhalation of pesticides
 - Crawling
 - Playing close to the floor
 - Plush toys
 - Hand-to-mouth
 - Object-to-mouth

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Spraying pesticides in the home results in increased risks to children because of higher concentrations near the floor and persistence of insecticides in carpets and soft toys. The typical activities of young children also contribute to their higher exposure.

Pesticides are covered extensively in a separate module.

Ref:

Reigart R et al. Pesticides in children. Pediatric Clinics of North America, 2001, 48:1185.

Children are exposed to a wide range of pesticides, including insecticides, herbicides, fungicides and rodenticides. They differ from adults in their exposures and responses to exposures. Acute and chronic toxicity are discussed, and important chronic effects, such as carcinogenesis, endocrine disruption, and neurodevelopmental effects are reviewed. Laws and regulations are also discussed. Recommendations are made to pediatricians regarding treatment and advising families regarding avoidance of pesticide exposures and their effects.

PESTICIDES: INSECTICIDES

Classes commonly used for insect control indoors:

- Pyrethroids: allergenic, CNS toxicity at high levels
- Cholinesterase inhibitors: neurotoxicants, neurodevelopmental toxicants
- ❖ Insect repellents (DEET)
- Mosquito coils

Health effects:

- Acute poisoning
- Allergic and general symptoms

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Classes of insecticide commonly used for insect control indoors include the following:

- Pyrethroids: these are very allergenic and can lead to central nervous system (CNS) toxicity at high levels of exposure.
- Cholinesterase inhibitors: these are neurotoxicants and neurodevelopmental toxicants (organophosphosphates, carbamates).
- •Insect repellents: diethyltoluamide (DEET).
- Mosquito coils.

The effects on health of exposure to these insecticides include:

- •acute poisoning usually related to accidental ingestion in children;
- •allergic and general symptoms
 - headache, nausea, vomiting;
 - cough, rhinitis, bronchitis, asthma and other allergic symptoms.

MOSQUITO COILS

Household use in Africa, Asia, South America

- Major active ingredient pyrethrins
- Long-term exposures linked to asthma and wheezing



ehp.niehs.nih.gov/members/2003/6177. 6177.html

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Mosquito coils may represent a serious potential threat to children's health. Prolonged use has been associated with increased incidences of asthma and persistent wheezing in children. Although the active ingredient is usually small amounts of pyrethrins (considered a low-toxicity insecticide), over 99% of the mass of the coil is so-called "inert" ingredients. When analysed, the smoke from coils was found to be entirely composed of respirable-sized particles, some quite small. The particles contain numerous polycyclic aromatic hydrocarbons (PAH) and carbonyl compounds including formaldehyde. One recent analysis found that the burning of one mosquito coil for 2 hours allowed a steady state of particulate matter to develop, and that the PM2.5 produced was the equivalent of that from burning 75–137 cigarettes (the formaldehyde produced was the equivalent of 51 cigarettes).

Ref:

•Liu W et al. Mosquito coil emissions and health implications. *Environ Health Perspect*, 2003, 111:1454-60.

Burning mosquito coils indoors generates smoke that can control mosquitoes effectively. This practice is currently used in numerous households in Africa, Asia and South America. However, the smoke may contain pollutants of health concern. We conducted the present study to characterize the emissions from four common brands of mosquito coils from China and two common brands from Malaysia. We used mass balance equations to determine emission rates of fine particles (particulate matter < 2.5 µm in diameter; PM2.5), polycyclic aromatic hydrocarbons (PAHs), aldehydes and ketones. Having applied these measured emission rates to predict indoor concentrations under realistic room conditions, we found that pollutant concentrations resulting from burning mosquito coils could substantially exceed health-based air quality standards or guidelines. Under the same combustion conditions, the tested Malaysian mosquito coils generated more measured pollutants than did the tested Chinese mosquito coils. We also identified a large suite of volatile organic compounds, including carcinogens and suspected carcinogens, in the coil smoke. In a set of experiments conducted in a room, we examined the size distribution of particulate matter contained in the coil smoke and found that the particles were ultrafine and fine. The findings from the present study suggest that exposure to the smoke of mosquito coils similar to the tested ones can pose significant acute and chronic health risks. For example, burning one mosquito coil would release the same amount of PM2.5 mass as burning 75-137 cigarettes. The emission of formaldehyde from burning one coil can be as high as that released from burning 51 cigarettes.

Picture: ehp.niehs.nih.gov/members/2003/6177/6177.html. NIEHS

SOLVENTS AND VOLATILE ORGANIC COMPOUNDS

Alkanes, aromatic hydrocarbons, alcohols, aldehydes, ketones

Sources:

- Solvents, fabric softeners, deodorizers and cleaning products
- Paints, glues, resins, waxes and polishing materials
- Spray propellants, dry cleaning fluids
- Pens and markers
- Binders and plasticizers
- Cosmetics: hair sprays, perfumes



www.epa.gov/oppfead1/cb/10_tips/

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Organic chemicals are widely used as ingredients in household products including paints, varnishes, wax, cosmetics, degreasing agents, wood preservatives, aerosol sprays, cleansers, disinfectants, moth repellents, air fresheners and hobby products. Fuels are also made up of organic chemicals.

All of these products can release organic compounds while they are being used, and, to some degree, when they are stored.

The average levels of several organic compounds in indoor air are 2–5 times higher than in outdoor air. During certain activities, such as paint-stripping, and for several hours immediately afterwards, levels may be 1000 times higher than outdoor levels.

These notes are taken from the US EPA Website www.epa.gov/iaq/voc.html

Picture: www.epa.gov/oppfead1/cb/10_tips/

VOLATILE ORGANIC COMPOUNDS: HEALTH EFFECTS

Acute:

- Irritation of eyes and respiratory tract
- General: headache, dizziness, loss of coordination, nausea, visual disorders
- Allergic reactions, including asthma and rhinitis

Chronic:

- Damage to liver, kidney, blood system and central nervous system (CNS)
- Some may cause cancer in humans (formaldehyde)

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Volatile organic compounds (VOC) vary greatly in their health effects: some are highly toxic, whereas some have no known effects on health.

As with other pollutants, the extent and nature of the effects on health will depend on many factors including level of exposure and duration of exposure.

The immediate symptoms experienced after exposure to VOC may include eye, nose and throat irritation; headaches; loss of coordination; nausea; dizziness; and visual disorders. Memory impairment and damage to liver, kidneys and central nervous system (CNS) may also occur. Little is yet known about what effects on health occur from exposure to the levels of organics usually found in homes. Many organic compounds are known to cause cancer in animals; some are suspected of causing, or are known to cause, cancer in humans.

Steps to reduce exposure

- •Use household products according to manufacturer's directions.
- •Make sure to provide plenty of fresh air when using these products.
- •Throw away unused or little-used containers safely; buy in quantities that can soon be used.
- •Keep out of reach of children and pets.
- •Never mix household care products unless directed on the label.

These notes are taken from the US EPA website www.epa.gov/iag/voc.html

FORMALDEHYDE

Sources: differ according to country

Developing countriesUse of solid fuels indoors

- Mosquito coils
- Furniture (pressed wood)

Industrialized countries

- Household cleaners and deodorizers
- Glues and resins
- Tobacco smoke
- Carpeting
- Furniture and dyed materialsPressed wood products
- Urea formaldehyde insulating foam (UFFI)
- Others

Ref:

www.epa.gov/iaq/formalde.html

FORMALDEHYDE: HEALTH EFFECTS

- Irritation of eyes, nose and throat
- Breathing difficulties
- Skin rash
- Asthma and other allergic reactions
- May be a sensitizer
- May cause cancer

Prevention:

- Reduce exposure
- Provide adequate ventilation
- Maintain moderate temperature and humidity levels

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Formaldehyde, a colourless, pungent-smelling gas, can cause watery eyes, burning sensations in the eyes and throat, nausea and difficulty in breathing (wheezing and coughing) in some humans exposed to levels above 0.1 parts per million. High concentrations may trigger attacks in people with asthma. There is evidence that some people can develop a sensitivity to formaldehyde. It has also been shown to cause cancer in animals and may cause cancer in humans.

Reducing exposure to formaldehyde in homes

- •Ask about the formaldehyde content of pressed wood products, including building materials and furniture before purchasing them.
- •Maintain moderate temperature and humidity levels and provide adequate ventilation.

The rate at which formaldehyde is released is accelerated by heat and may also depend somewhat on the humidity level. Therefore, the use of dehumidifiers and air conditioning to control humidity and to maintain a moderate temperature can help reduce formaldehyde emissions.

These notes are taken from the US EPA website www.epa.gov/iaq/formalde.htm

BIOLOGICAL POLLUTANTS

Biological pollutants are/were living organisms:

Animal dander, dust mites, moulds, infectious agents, pollen

Sources of biological agents:

- Water-damaged surfaces and materials
- Humidifiers and stagnant water
- Water vapour from cooking and showering
- Air conditioning systems
- Mattresses, upholstered furniture and carpets
- Dirt

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Dust mites, fungi and bacteria require moisture to proliferate. Permeation of rain or groundwater into a building and condensation on cold interior surfaces can promote proliferation of microbes. Water vapour is produced by people and pets, cooking and showering and requires sufficient air exchange to prevent moisture problems. Mattresses, upholstered furniture and carpets are reservoirs for dust mites.

Moulds have been associated with three types of effects: infections, allergic reactions and toxic effects. Toxic effects may be caused by inhalation of mycotoxins.

These notes are taken from the US EPA website www.epa.gov/iaq/pubs/bio 1.html

DUST MITES

- Feed on human dander
- Prefer warm, humid environments
- Sources:
 - bedding, carpets, upholstery, soft toys

Prevention:

- Encasing mattress and pillows
- Washing bedding in hot water
- Frequent vacuuming / damp mopping
- Decreasing clutter
- Removing carpets



CDC

Dust mites are acarians (Dermatophagoides spp.)

The effectiveness of prevention measures against dust mite sensitization has been studied in a European multicentre randomized controlled trial. Intervention was a combination of education and mattress encasement. Of 566 preschool-aged children in the study's first-year follow-up (mean age = 3.1 years), the incidence of sensitization to mite allergens was 10 (3%) of 330 in the intervention versus 20 (6.5%) of 306 in the comparison (control) group.

Likewise, in 213 school age children, 3 (2.56%) of 117 children in the intervention group and 9 (9.38%) of 96 in the comparison (control) group developed sensitization to dust mite.

Refs:

- •Tsitoura S et al. Randomized trial to prevent sensitization to mite allergens in toddlers and preschoolers by allergen reduction and education: one-year results. Arch Pediatr Adolesc Med, 2002, 156:1021-7.
- •Arshad SH et al. Prevention of sensitization to house dust mite by allergen avoidance in school age children: a randomized controlled study. Clin Exp Allergy, 2002, 32:843.
- •Arshad SH et al. Prevention of allergic disease during childhood by allergen avoidance: the Isle of Wight prevention study. J Allergy Clin Immunol. 2007 Feb;119(2):307-13.

BACKGROUND: Early life allergen exposure may increase the risk of childhood allergy, but the protective effect of reduction in allergen exposure remains uncertain. OBJECTIVE: To evaluate the effect of reduction in food and house dust mite (HDM) allergen exposure in infancy in preventing asthma and allergy. METHODS: Infants, at higher risk because of family predisposition, were recruited prenatally and randomized to prophylactic (n = 58) and control (n = 62) groups. Prophylactic group infants were either breast-fed with mother on a low allergen diet or given an extensively hydrolyzed formula. Exposure to HDM was reduced by the use of an acaricide and mattress covers. The control group followed standard advice. Development of allergic diseases and sensitization to common allergens (atopy) was assessed blindly at ages 1, 2, 4, and 8 years in all 120 children. RESULTS: Repeated measurement analysis, adjusted for all relevant confounding variables, confirmed a preventive effect on asthma: adjusted odds ratio (OR), 0.24; 95% CI, 0.09-0.66; P = .005; atopic dermatitis, OR, 0.23; CI, 0.08-0.64; P = .005; rhinitis, OR, 0.42; CI, 0.19-0.92; P = .03; and atopy, OR, 0.13; Cl. 0.05-0.32; P < .001. The protective effect was primarily observed in the subgroup of children with persistent disease (symptoms at all visits) and in those with evidence of allergic sensitization. CONCLUSION: Allergic diseases can be reduced, for at least the first 8 years of life, by combined food and HDM allergen avoidance in infancv. CLINICAL IMPLICATIONS: Strict food and HDM allergen

ANIMAL ALLERGENS

- Cat dander (most allergenic)
- Dog dander
- Birds
- Cockroach parts and faeces

Prevention:

- Remove animals from indoors
 - Allergens persist for many months after removal of source
- Clean environment and pet(s) frequently
- Ventilate adequately
- Control dust and moisture

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- Cats are most allergenic.
- ■Dogs are the most common household pet allergy to dogs is uncommon.
- ■Birds (harbour dust mites).
- Cockroach parts (keratin) and faeces are an important cause of asthma morbidity.

Prevention

- Avoid exposure: allergens persist for many months after removal of their source.
- Clean the environment and pet(s) frequently.
- Ensure adequate ventilation.
- Control dust and moisture.
- •Use an air filtration system.

Refs:

- •McConnell R et al. Cockroach counts and house dust allergen concentrations after professional cockroach control and cleaning, *Ann Allergy Asthma Immunol*, 2003, 91:546-52.
- •Becker AB. Primary prevention of allergy and asthma is possible. *Clin Rev Allergy Immunol*, 2005, 28:5-16.

MOULDS

- A frequently undetected environmental problem
- Occur in damp indoor areas
- Allergies and nonspecific symptoms are common, but infections are rare

Mold growing on a wooden headboard in a room with high humidity.



FPA

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Ref.

•Etzel RA et al. Indoor mold and children's health. Environ Health Perspect, 1999, 107(Suppl)3:463. Reactive airways disease in children is increasing in many countries around the world. The clinical diagnosis of asthma or reactive airways disease includes a variable airflow and an increased sensitivity in the airways. This condition can develop after an augmented reaction to a specific agent (allergen) and may cause a life-threatening situation within a very short period of exposure. It can also develop after a long-term exposure to irritating agents that cause an inflammation in the airways in the absence of an allergen. (paragraph) Several environmental agents have been shown to be associated with the increased incidence of childhood asthma. They include allergens, cat dander, outdoor as well as indoor air pollution, cooking fumes, and infections. There is, however, increasing evidence that mould growth indoors in damp buildings is an important risk factor. About 30 investigations from various countries around the world have demonstrated a close relationship between living in damp homes or homes with mould growth, and the extent of adverse respiratory symptoms in children. Some studies show a relation between dampness/mould and objective measures of lung function. Apart from airways symptoms, some studies demonstrate the presence of general symptoms that include fatigue and headache and symptoms from the central nervous system. At excessive exposures, an increased risk for haemorrhagic pneumonia and death among infants has been reported. The described effects may have important consequences for children in the early years of life. A child's immune system is developing from birth to adolescence and requires a natural, physiological stimulation with antigens as well as inflammatory agents. Any disturbances of this normal maturing process will increase the risk for abnormal reactions to inhaled antigens and inflammagenic agents in the environment. The knowledge about health risks due to mould exposure is not wi

MOULD-RELATED DISEASES

- Airway and conjunctival irritation
- Headache
- Difficulty in concentrating
- Hypersensitivity reactions: asthma, rhinitis
- Systemic infections (immunosuppressed child)
- Acute exposure associated with pulmonary haemorrhage in infants

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Hypersensitivity to moulds is immediate (type 1) and includes: acute asthma, allergic rhinitis and urticaria (hives) (which is uncommon).

Colonization associated with chronic asthma is rare and serious (allergic bronchopulmonary mycosis and allergic mycotic sinusitis).

Deep fungal infections are uncommon: serious, life-threatening diseases are not usually caused by common household moulds. The major exception is aspergillosis. Mould infection occurs only in an immunocompromised children.

Ref:

- •Etzel RA. Indoor mold and children's health. Environ Health Perspect, 1999, 107(Suppl 3):463.
- •Etzel RA et al. Acute pulmonary hemorrhage in infants associated with exposure to Stachybotrys atra and other fungi. Arch Pediatr Adolesc Med. 1998 Aug;152(8):757-62

BACKGROUND: A geographic cluster of 10 cases of pulmonary hemorrhage and hemosiderosis in infants occurred in Cleveland, Ohio, between January 1993 and December 1994. STUDY DESIGN: This community-based case-control study tested the hypothesis that the 10 infants with pulmonary hemorrhage and hemosiderosis were more likely to live in homes where Stachybotrys atra was present than were 30 age- and ZIP code-matched control infants. We investigated the infants' home environments using bioaerosol sampling methods, with specific attention to S atra. Air and surface samples were collected from the room where the infant was reported to have spent the most time. RESULTS: Mean colony counts for all fungi averaged 29 227 colony-forming units (CFU)/m3 in homes of patients and 707 CFU/m3 in homes of controls. The mean concentration of S atra in the air was 43 CFU/m3 in homes of patients and 4 CFU/m3 in homes of controls. Viable S atra was detected in filter cassette samples of the air in the homes of 5 of 9 patients and 4 of 27 controls. The matched odds ratio for a change of 10 units in the mean concentration of S atra in the air was 9.83 (95% confidence interval, 1.08-3 X 10(6)). The mean concentration of S atra on surfaces was 20 X 10(6) CFU/g and 0.007 x 10(6) CFU/a in homes of patients and controls, respectively, CONCLUSION: Infants with pulmonary hemorrhage and hemosiderosis were more likely than controls to live in homes with toxigenic S atra and other fungi in the indoor air.

CHEMICAL AGENTS PRODUCED BY MOULDS

Mycotoxins are associated with human disease and cause acute and chronic effects

- Mycotoxins
 - Aflatoxins
 - Tricothecenes
 - Ochratoxins and citrinin
 - Hundreds of others
- Glucans (cell wall components)
- Volatile organic compounds (irritating)

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Mycotoxins are associated with human disease.

Tricothecenes inhibit protein synthesis and have many acute effects, including anemia and infant pulmonary haemorrhage.

Ochratoxins and citrinin cause nephropathy and immunosuppression.

Aflatoxins are hepatotoxins and are carcinogenic.

(See module on Mycotoxins)

Refs:

•Etzel RA. What the primary care pediatrician should know about syndromes associated with exposures to mycotoxins. Curr Probl Pediatr Adolesc Health Care. 2006;36(8):282-305.

Disease associated with exposure to mycotoxins is known as the "Great Masquerader" of the 21st century because of its complex natural history involving different tissues and resembling different diseases at each stage in its evolution. It can present with a variety of nonspecific clinical signs and symptoms such as rash, conjunctivitis, epistaxis, apnea, cough, wheezing, nausea, and vomiting. Some cases of vomiting illness, bone marrow failure, acute pulmonary hemorrhage, and recurrent apnea and/or "pneumonia" are associated with exposure to mycotoxins. Familiarity with the symptoms of exposure to the major classes of mycotoxins enables the clinician to ask pertinent questions about possible fungal exposures and to remove the infant or child from the source of exposure, which could be contaminated food(s), clothing and furniture, or the indoor air of the home. Failure to prevent recurrent exposure often results in recurrent illness. A variety of other conditions, including hepatocellular and esophageal cancer and neural tube defects, are associated with consumption of foods contaminated with mycotoxins. Awareness of the short- and long-term consequences of exposures to these natural toxins helps pediatricians to serve as better advocates for children and families.

•Novak M et al. Beta-glucans, history, and the present: immunomodulatory aspects and mechanisms of action. J Immunotoxicol. 2008 Jan;5(1):47-57.

The present paper represents a comprehensive up-to-date review of beta -glucans, their chemical and biological properties, and their role in immunological reactions. beta -D-Glucans belong to a group of physiologically active compounds called biological response modifiers and represent highly conserved structural components of cell walls in yeast, fungi, or seaweed. Despite almost 150 years of research, the exact mechanisms of their action remain unclear. The present review starts with the history of glucans. Next, attention is focused on sources and structure, comparing the effects of physicochemical properties, and sources on biological effects. As glucans belong to natural products useful in preventing various diseases, they have been highly sought after throughout human history. Based on extensive recent research, this paper explains the various mechanisms of effects and the ways glucans mediate their effects on defense reactions against infections. Despite the fact that predominately pharmacological effects of glucans are positive, their unfavorable and potentially toxic side effects were not overlooked. In addition, attention was focused on the future research, possible alternatives such as synthetic oligosaccharides, and on clinical applications.

INDOOR AIR QUALITY: BUILDING COMPONENTS

The importance of:

- Materials in houses and schools:
 - Asbestos, wood preservatives, paints and others
- Ventilation
- Heating
- Use (overuse) and location of electric appliances
- Air conditioning

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Building components may be a source of indoor air pollution. The materials used in the construction of a house or a school may release toxicants into the air. Indoor air pollutants may have an effect on the health and performance of children in schools. Lack of ventilation (and hygiene) increases the risk of exposure, which may be further enhanced by heating and faulty air conditioning.

Knowledge of this issue has fueled interest in "green houses" and "green schools".

The "built environment" refers to the way a community is built, including how people get from home to school, work, stores, places of worship. houses, offices and other manmade structures in which people live, work and play.

Refs:

- •Etzel RA. Indoor air pollutants in homes and schools. Pediatric Clinics of North America, 2001, 48:1153.
- •Mendell MJ et al. Do indoor air pollutants and thermal conditions in schools influence student performance? A critical review of the literature. Indoor Air 2005,15:27-52.

SICK BUILDING SYNDROME

What is this syndrome?

- Discomfort not related to specific illness
- Effects appear to be linked to time spent inside the building
- Cause of symptoms is unknown
- Most complaints relieved soon after leaving the building
- An evolving area

Building related illness: symptoms of identified illness attributed to airborne contaminants in the building

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When energy prices soared in the 1970s there was a movement to make buildings "tight" to preserve heat in winter and air conditioning in summer. The unexpected consequence, in some buildings, was an increase of indoor pollution due to inadequate ventilation. Pollutants from off-gassing from building materials together with other indoor pollutants were trapped in some structures and built up to levels that caused symptoms in sensitive individuals. "Sick building syndrome" is the name that was given to this phenomenon.

Ref:

- •Indoor Air Facts N°4: Sick Building Syndrome. www.epa.gov/iaq/pubs/sbs.html
- •Wang BL et al. Symptom definitions for SBS (sick building syndrome) in residential dwellings. Int J Hyg Environ Health. 2008 Mar;211(1-2):114-20.

The potential risk factors for sick building syndrome (SBS) in newly built dwellings were investigated. Two different definitions for SBS were used, a narrow definition (symptoms related to home environment and continuously occurring in the last 3 months were regarded as positive) and another relatively broad definition (symptoms related to home environment and either continuously or sporadically occurring in the last 3 months were regarded as positive). With both definitions indoor air chemicals, especially TVOC, and high stress during work were found to be significantly associated with SBS symptoms. Allergic history was more associated with narrow-sense symptoms and odor perception with broad-sense symptoms. The results indicate that the broad definition be preferred to find more potential risk factors.

SICK BUILDING SYNDROME

Possible causes:

- Inadequate building design
- Occupant activities
- Remodelled buildings operating in a manner inconsistent with their original design
- Inadequate ventilation
- Inadequate maintenance
- Chemical and biological contaminants

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<<READ SLIDE.>>

SICK BUILDING SYNDROME

Symptoms:

- ❖ Headache
- Irritation of eyes, nose or throat
- Dry cough
- ❖ Dry or itchy skin
- Difficulty in concentrating
- ❖ Fatigue
- Sensitivity to odours

Solutions:

- Remove source of pollutant
- ❖ Increase ventilation
- ❖ Air cleaning: filters
- Education and communication

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<<READ SLIDE.>>

RADON

- Radioactive gas released from soil and rocks
- Second leading cause of lung cancer (in adults)
- Geology of the area can predict levels in soil and water
- Concentrations indoors depend on construction site and building materials
- Highest levels occur in basements and on the ground floor

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Radon is a radioactive gas that comes from the soil. Exposure to radon gas it the second-leading cause of lung cancer (after smoking) in the United States. About 14 000 adults die each year in the USA from radon-related lung cancer.

Radon is produced from the natural breakdown of thorium and uranium found in most rocks and soils. As it further breaks down, radon emits atomic particles. These particles are in the air we breathe and can be deposited in the lungs. The energy associated with these particles can alter DNA, leading to an increased risk of lung cancer.

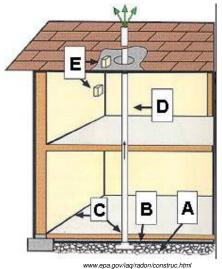
Radon does not usually present a health risk outdoors because it is diluted in the open air. Radon can, however, build up to dangerous levels inside a house. Radon can enter a new house through cracks or pores in concrete flooring and walls or through openings in the foundations, floor—wall joints or loose pipes. The differences in air pressure between the inside of a building and the soil around it also play an important role in radon entry. If the air pressure of a house is greater than that of the soil beneath it, radon will remain outside. However, if the air pressure of a house is lower than that of the surrounding soil (which is usually the case), the house will act as a vacuum, sucking radon gas inside. Because radon comes from the soil, a knowledge of the geology of an area can help to predict the potential for elevated indoor radon levels.

These notes are taken from the US EPA website www.epa.gov/radon/index.html

RADON MITIGATION SYSTEM

- A Gas-permeable layer
- B Plastic sheeting
- C Sealing and caulking
- D Vent pipe
- E Junction box

It is recommended that homes be tested for radon on the lowest lived-in level - basement or ground floor



- A. Gas-permeable layer. This layer is placed beneath the slab or flooring system to allow the soil gas to move freely underneath the house. In many cases, the material used is a 4-inch layer of clean gravel.
- B. Plastic sheeting. Plastic sheeting is placed on top of the gas-permeable layer and under the slab to help prevent the soil gas from entering the home. In crawlspaces, the sheeting is placed over the crawlspace floor.
- C. Sealing and caulking. All openings in the concrete foundation floor are sealed to reduce entry of soil gas into the home.
- D. Vent pipe. A 3- or 4-inch gas-tight or polyvinyl chloride (PVC) pipe (commonly used for plumbing) runs from the gas permeable layer through the house to the roof to safely vent radon and other soil gases above the house.
- E. Junction box. An electrical junction box is installed in case an electric venting fan is needed later.

Testing is not necessary above the second story.

These notes are taken from the US EPA website www.epa.gov/iag/radon/construc.html.

RADON IN SCHOOLS



www.epa.gov/radon/pubs/schoolrn.html

Schools should also be tested for radon:

❖ Levels above 4 pCi/L call for action to reduce exposure

5

A USA survey of radon levels in schools estimated that nearly one in five schools has at least one schoolroom with a short-term radon level above 4 pCi/L (picoCuries per litre) — the level at which EPA recommends that schools take action to reduce the level. EPA estimates that more than 70 000 schoolrooms in use today have high short-term radon levels. *Ref:*

www.epa.gov/radon/pubs/schoolrn.html

ASBESTOS

Sources:

- Used for insulation and as fire-retardant: asbestos cement, floor and roof tiles, water pipes and others
- Levels increase if asbestos-containing materials are damaged
- Levels can be high in clothes of working parents

Health effects:

- No acute toxicity
- Asbestosis results from occupational exposure
- Main risk for children: long-term exposure may cause cancer in adulthood
 - Malignant mesothelioma
 - Lung cancer

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Asbestos is a fibrous mineral product and is classified into six types: amosite, chrysotile, crocidolite, tremolite, actinolite and anthophyllite. It is very resistant and almost indestructible and has been used widely in manufactured products and building materials. Inhalation of microscopic fibres is the major route of exposure. Fibres are liberated from deterioration, destruction or renovation of asbestos-containing materials. Asbestos produces no acute toxicity. Workers exposed to asbestos in industry may develop asbestosis. The main risk for children is the long-term exposure that may lead to cancers, such as lung cancer and malignant mesothelioma.

These notes are taken from the US EPA websites www.epa.gov/iaq/asbestos.html and www.epa.gov/asbestos/ashome.html

Ref:

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

OCCUPATION-RELATED CONTAMINANTS

Parents "take home" exposures related to work:

- Contaminated clothing, shoes and objects
- Contaminated skin and exhaled breath
- Empty containers (pesticide and others)

Children are directly exposed when:

- Visiting parents' workplaces
- Participating in work

Prevention

Change work clothes and shower before hugging or playing with children

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Clothing contaminated with pesticides and other chemicals can be an important source of exposure for children and a source of indoor air contamination. Exposures of family members to pesticides have occurred from contact with contaminated skin, clothing or shoes, contamination of the family car, and during visits to the workplace. Parents should avoid hugging children or playing with them after coming home from work until they have showered and changed their clothes.

CHILDREN WORKING INDOORS

Child labour and adolescent work: exposure to pesticides, solvents, cleaning agents and other chemicals indoors

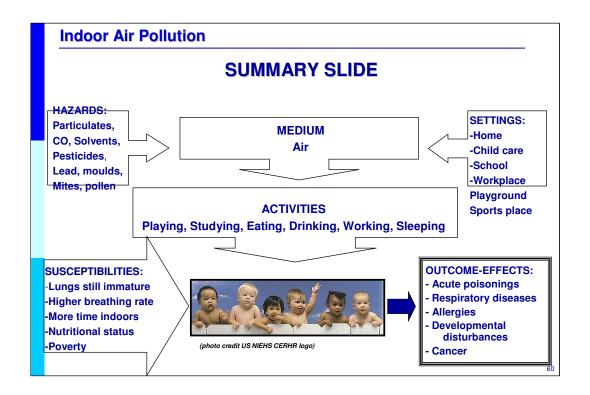
- Young children employed illegally
- Unregulated temporary work
- School-based vocational training
- Family businesses
- Cottage industries
- Indoor smoke from solid fuels (e.g. food stands)
- Volunteer service projects

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Children and adolescents are likely to work without proper training and protective equipment, leading to their being exposed to dangerous products and pesticides, mainly when they are being employed illegally or doing unregulated work.

Solvents and cleaning agents are important sources of exposure of employed adolescents. Their workplaces include fast-food restaurants, automotive services, retail stores and others. One of the most common types of exposure is to cleaning products containing ammonia or other airway irritants. Other common sources of exposures include paints, glues and solvents, caustic agents, hydrocarbons and bleach. Chemical burns have also been reported.

Another group of adolescents or younger children who may suffer potentially hazardous exposures to pesticides are the children of farm-workers working in the fields beside their parents.



Different hazards are presented by different settings and various children's activities. The effects of exposure are influenced by individual susceptibility which depends upon age, developmental stage, and social support. Pollutants in the indoor environment are potentially more hazardous to children than adults because their lungs are still growing and maturing; younger children breathe more air than older children or adults; and they spend more time indoors. All of these susceptibilities are modified by nutritional status and poverty.

APPROACHES TO REDUCE INDOOR AIR POLLUTION

- Eliminate or control the sources of pollution
 - Improved stoves
 - Clean fuels (kerosene, gas)
 - Venting stoves for cooking and heating
 - Regular maintenance of cooking, heating and cooling systems
 - Choose non-volatile, non-toxic building materials
 - Maintaining dry homes and schools
- Ventilation building design
 - Dilute and remove pollutants through ventilation with outdoor air
- 3. Air cleaning NOT air fresheners!
 - Air filters and ionizers may remove some airborne particles
 - Gas adsorbing material is used to remove gaseous contaminants

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It is always better to prevent rather than treat illness. To avoid problems due to indoor air quality, the first approach is source reduction and elimination, and the second, proper ventilation and maintenance of gas, oil and solid fuel cooking, heating and cooling systems. Air cleaning is the least effective, and most expensive. Air fresheners, which contain untested potentially harmful volatile organic compounds (VOCs), should not be used to cover up stale air or unpleasant smells.

Ref:

www.epa.gov/iaq/pubs/ozonegen.html

For more information on indoor air pollution, you can obtain a guide (Indoor pollution: An introduction for health professionals) recently published by the US EPA at: www.epa.gov/iedweb00/pubs/hpguide.html

Ref:

Meklin T et al. Effects of moisture-damage repairs on microbial exposure and symptoms in schoolchildren. Indoor Air. 2005;15 Suppl 10:40-7.

Effects of renovation on symptom prevalence and microbial status were studied in two moisture-damaged schools and in two non-damaged schools with longitudinal cross-sectional surveys before and after repairs. Over 1300 schoolchildren aged 6-17 returned questionnaires before and after repairs. After full renovation in one of the damaged schools, elevated concentrations and increased frequencies of indoor air fungi normalized and a significant decrease in the prevalence of 10 symptoms of 12 studied was observed among schoolchildren. No change in microbial conditions was seen after partial repairs in the other damaged school, and only slight improvement was observed in symptom prevalence. The change in the prevalence of symptoms in the reference schools was minor. The results suggest that increased symptom prevalence among schoolchildren in moisture-damaged schools can be managed with proper repair of the moisture damage. PRACTICAL IMPLICATIONS: This longitudinal intervention study showed the positive effects of the moisture and mold damage repairs of a school building on children's health. The success necessitates however, a thorough renovation including appropriate ventilation. Monitoring of

OZONE GENERATORS – CAUTION

- Ozone can be harmful to health
 - Chest pain, coughing, throat irritation
- Ozone is ineffective in controlling indoor air pollution below health standards levels
 - NIOSH / OSHA recommends an upper limit of 0.10 ppm ozone
- High concentrations are used to decontaminate unoccupied spaces from chemical and biological contaminants or odours

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Ozone generators have become popular in some industrialized countries. Ozone is a molecule composed of three atoms of oxygen. The third oxygen atom can detach from the ozone molecule, and re-attach to molecules of other substances, thereby altering their chemical composition. Scientific evidence shows that, at concentrations that do not exceed public health standards, ozone has little potential to remove indoor air contaminants: there is no approval for its use in occupied spaces.

When inhaled, ozone can damage the lungs. Relatively small amounts can cause chest pain, coughing, shortness of breath and throat irritation. Ozone may also exacerbate chronic respiratory diseases such as asthma and compromise the ability of the body to fight respiratory infections. People vary widely in their susceptibility to ozone.

Ozone has been extensively used for water purification, but ozone chemistry in water is not the same as ozone chemistry in air. High concentrations of ozone in air, when people are not present, are sometimes used to help decontaminate an unoccupied space from certain chemical or biological contaminants or odors (e.g., fire restoration). However, little is known about the chemical by-products left behind by these processes (Dunston et al, 1997). While high concentrations of ozone in air may sometimes be appropriate in these circumstances, conditions should be sufficiently controlled to insure that no person or pet becomes exposed. Ozone can adversely affect indoor plants, and damage materials such as rubber, electrical wire coatings, and fabrics and art work containing susceptible dyes and pigments (U.S. EPA, 1996a).

These notes are taken from the US EPA website www.epa.gov/iaq/pubs/ozonegen.html

Refs:

US EPA, *Air Quality Criteria for Ozone and Related Photochemical Oxidants*. Research Triangle Park, NC: National Center for Environmental Assessment-RTP Office; report nos. EPA/600/P-93/004aF-cF, 3v. NTIS, Springfield, VA; PB-185582, PB96-185590 and PB96-185608. 1996

Dunston et al. A Preliminary Investigation of the Effects of Ozone on Post-Fire Volatile Organic Compounds. Journal of Applied Fire Science, 1997, 6(3): 231.

EDUCATION AND ADVOCACY

Education of:

- Children
- Family and community
- Health care providers

Environment policy-making

- Framework Convention on Tobacco Control
- Clean indoor air regulations
- Community actions
- Research

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A multi-level approach to prevention of indoor air-related illness is required. Education, policy-making and research all have roles to play.

<<READ SLIDE>>

<<NOTE TO USER: Insert appropriate suggestions for tackling the specific indoor air problems in your area.>>

Refs:

Eriksen MP et al. The diffusion and impact of clean indoor air laws. *Annu Rev Public Health.* 2008;29:171-85.

Over the past quarter century, primarily as a result of scientific discovery, citizen advocacy, and legislative action, comprehensive clean indoor air laws have spread rapidly throughout the world. Laws that establish completely smoke-free indoor environments have many relative advantages including being low cost, safe, effective, and easy to implement. The diffusion of these laws has been associated with a dramatic and rapid reduction in population levels of serum cotinine among nonsmokers and has also contributed to a reduction in overall cigarette consumption among smokers, with no adverse economic impact, except to the tobacco industry. Currently, nearly half of the U.S. population lives in jurisdictions with some combination of completely smoke-free workplaces, restaurants, or bars. The diffusion of clean indoor air laws is spreading rapidly throughout the world, stimulated by the first global health treaty, the Framework Convention on Tobacco Control.

CRITICAL ROLES OF HEALTH & ENVIRONMENT PROFESSIONALS



WHO

- Diagnose and treat
- Do research and publish
 - · Detect sentinel cases
 - Inspire community-based interventions

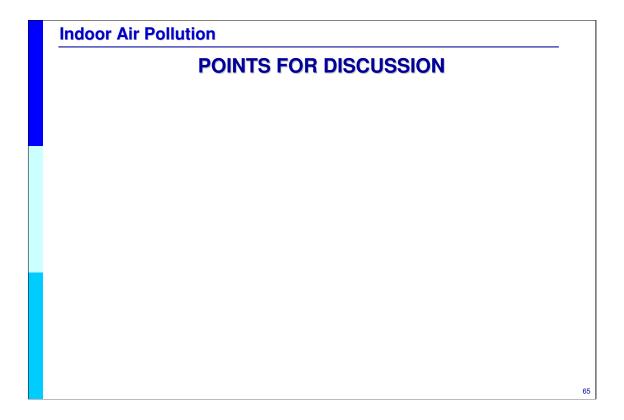
Educate

- 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 ensure children's health through a clean indoor environment.

- •At the patient level it is important to include indoor air as an environmental etiology or trigger of respiratory disease and in the preventive advice. Are the signs and symptoms possibly linked to air pollutant exposure? Are there any potential indoor sources of exposure?
- •Health care providers should be alert and detect the "sentinel" cases of indoor air pollutant exposure. Their detection and study will be essential for developing, proposing and supporting family and community-based interventions. Publication of cases and research studies allows the communication of knowledge and experience that will benefit other communities and countries.
- •It is important to inform and educate patients, families, colleagues and students didactically, on the possibility of exposure to indoor air pollutants and its potential impact in children. Also on how to avoid exposure and provide clean air.
- •Finally, we can become vigorous advocates for the protection of children's environments and prevention of exposure to indoor air pollutants. It is important to promote the measures that are crucial for eliminating or mitigating sources of exposure in children (and pregnant women!).
- •Professionals with understanding of both health and the environment are powerful role models. Their choices and opinions with respect to air pollutants and other environmental factors will be noticed by patients and communities.



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

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