LECTURE NOTES

For Environmental Health Science Students

Air Pollution

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In collaboration with the Ethiopia Public Health Training Initiative, The Carter Center,
the Ethiopia Ministry of Health, and the Ethiopia Ministry of Education

August 2006

Funded under USAID Cooperative Agreement No. 663-A-00-00-0358-00.

Produced in collaboration with the Ethiopia Public Health Training Initiative, The Carter Center, the Ethiopia Ministry of Health, and the Ethiopia Ministry of Education.

PREFACE


Ethiopia Public Health Training Initiative • Ethiopia Public Health Training Initiative
EPHTI

ACKNOWLEDGEMENTS

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ABBREVIATIONS

CNS

COHb

DALYS

EPA

EPHTI

GCMHS

IR

LOAEL

M.P.H

PM

TSM

TSP

UOG-

UV

VOC

CHAPTER ONE

INTRODUCTION

1.1. Learning Objective

1.2. Introduction to the course

TROPOSPHERE

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-
-

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-

-
-

MESOSPHERE

What is air Pollution?

Tukul

1.3. Historical overview

Homo sapiens

1.4.4. Acute effects:

1.4.5. Delayed effect:

1.4.6. Aerosols: -

1.4.7. Dust

1.4.8. Smoke: -

1.4.9. Ash: -

1.4.10. Particulates: -

1.4.11. Fumes: -

1.4.12. Inhalable fraction: -

µ

Unit of measurement

μ

×

×

×

μ

1.7. Exercise question

Table 1.1: Exercise on the basic requirements for a healthy environment

Parameter	Air	Water	Food	Settlement
Degree of importance				
Degree of accessibility				
Magnitude of health problem				
Risk of pollution at the Global level				
Risk of pollution at the National level				
Manageability level: -Globally - National - Households				
Preventive and control measures: - At policy - At community - At households				
Other parameters that need to be consider				

CHAPTER TWO

METEOROLOGY AND AIR POLLUTION

2.1. Learning objective

2.2. Introduction to the chapter

Saturated adiabatic lapse rate, (Γs)

°

Example

°

°

Solution

°

° ° °

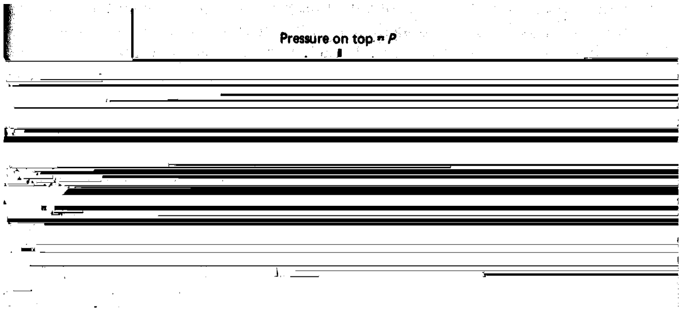
wind,

2.3. Temperature lapse rate and stability

adiabatic

$$\frac{dT}{dP} = \frac{V}{Cp} \text{-------------------} (2.2)$$

ρ ρ



$$P(z) = P(z+dz) + \frac{g\rho A dz}{A} ---------------------(2.3)$$

ρ

$$\frac{dT}{dZ} = \frac{dT}{dP} \times \frac{dP}{dZ} = \frac{V}{Cp}(-g\rho) -------------------(2.5)$$

ρ

ρ

$$\frac{dT}{dZ} = \frac{-g}{Cp} ---------------------------(2.6)$$

$$\frac{\quad}{\quad} \qquad \frac{\quad}{\quad} x \frac{J}{\quad} \qquad c \quad m$$

[illegible]

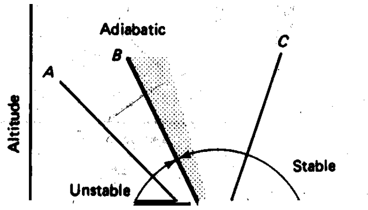
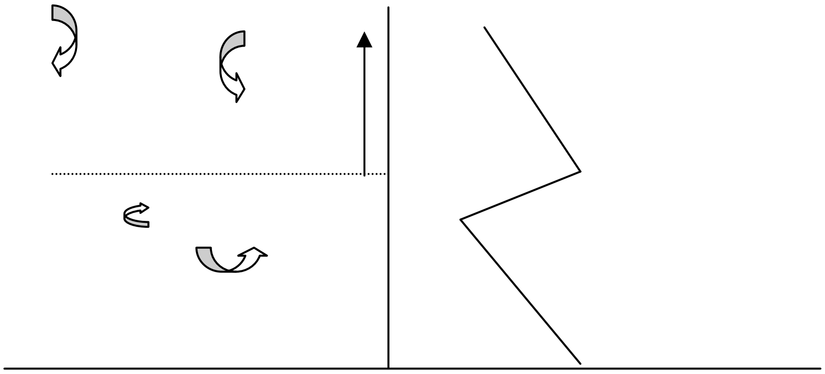
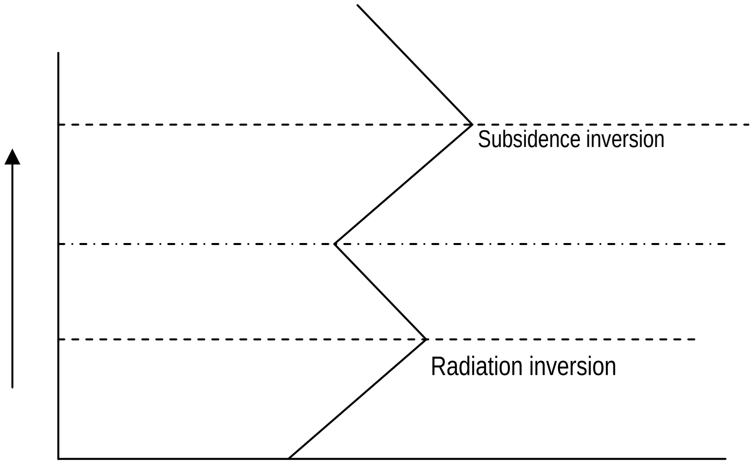


Figure 7.19 Temperature profiles to the left of the adiabatic lapse rate correspond to an unstable atmosphere (line *A*); profiles to the right are stable (line *C*). The dry adiabatic lapse rate is

Γ



Radiation Inversion



TOPOGRAPHICAL EFFECTS

2.4. Wind velocity and turbulence

$$\frac{U}{U1} = \frac{Z}{Z1}\ \alpha --------- (2.11)$$

α

α

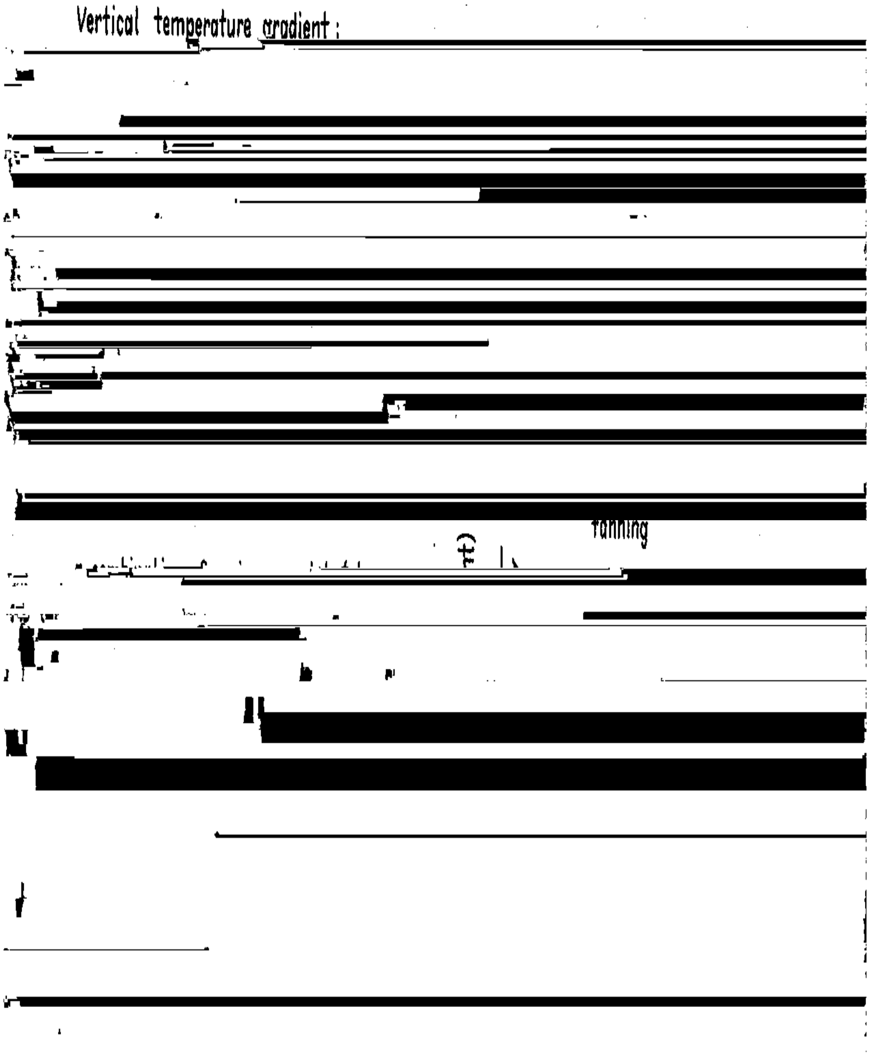
Table 2.1: Wind velocity in different topography

Surface configuration	Stability	α

2.5. Plume behavior

Classification of plume behavior

- **Looping:**


Vertical temperature gradient :
fanning

PLUME DISPERSION

(a)

$$\left[\rho A\right]\left(x \;\; y \quad H\right) \quad \frac{Q}{\pi\delta y\,\delta Z u} \qquad \frac{}{} \;\; \frac{y}{\delta y} \qquad \frac{}{} \;\; \frac{H}{\delta Z}$$

δ δ

$$[\rho A](0.5,0,60) = \frac{Q}{\pi \delta y \delta z u^{-}} . \exp \; \frac{-1}{2} \; \frac{H}{\delta z} \; {}^{2}$$

$$= \frac{18 g / \sec}{\pi \times 3.5 \times 18 \times 5} . \exp \; \frac{-1}{2} \; \frac{60}{18} \; {}^{2}$$

7.4μg

Example 2

A

(a) ρ

(b) ρ

(c) ρ

Solution

α α

δ δ

=

$$[\rho so2](1000,0,0,250) = \frac{500x10^6}{\pi(151)(108)6} \exp \frac{-1}{2} \frac{250}{108}^{2}$$

μ

$$\left[\rho so_2\right](\qquad\qquad) \quad \frac{Q}{\pi\delta y\delta zu} \qquad - \; \frac{y}{\delta y} \qquad - \; \frac{H}{\delta z}$$

Plume rise

Estimation of plume rise

1. Buoyant plumes

$$\Delta \mathrm{H} = \frac{Vs.Ds}{U^{-}} \; 1.5 + 2.68x10^{-3}\, pa.\frac{Ts - Ta}{TS}.Ds \quad ------(2.18)$$

Where: Vs = stack gas exit velocity, m/s

Pa = atmospheric pressure, mb

Ts = stack gas temperature, k

Ta =ambient air temperature

U - =wind speed, m/sec.

Ds = Diameter of stack out let, m

2. Plume rise under stable and calm conditions

$\Delta \mathrm{H}$

$$F = gvs\ \frac{Ds}{2}^{\;2} .\frac{Ts - Ta}{Ts}, \frac{m^4}{s^2} ----(2.20)$$

CHAPTER THREE

SOURCES, TYPES OF AIR POLLUTANTS AND THEIR EFFECTS

3.3. Common condition to which air pollution exposure may contribute

Table 3.1: Examples of Common Conditions to Which Air Pollution Exposure May Contribute

Disease or condition	How air pollution may affect it	Associated factors

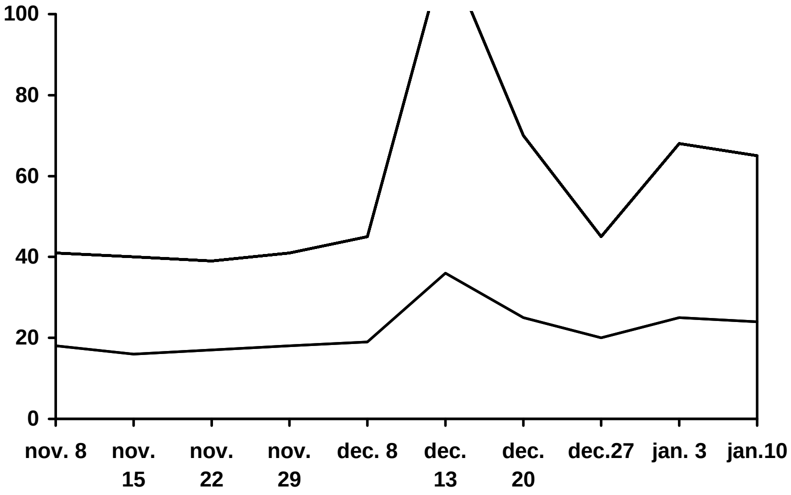
3.4. Types of pollutants

3.4.1 CONVENTIONAL

Sulfur Dioxide

BOX 5.1: London Fog

Figure 1: Deaths in London Administration County and the Outer Ring by Weeks. November 1952 – 10 June 1953



Nitrogen Dioxide

secondary pollutant

µ

µ

Table :- 3.2. Potential Human Effects of Nitrogen Dioxide

Health Effect	**Mechanism**
Increased incidence of respiratory infections	Reduced efficacy of lung defenses
Increased severity of respiratory infections	Reduced efficacy of lung defenses
Respiratory symptoms	Airways injury
Reduced lung function	Airways and possibly alveolar injury
Worsening of the clinical status of persons with asthma, Chronic obstructive pulmonary diseases or other chronic Respiratory conditions	Airways injury

Particulates matter

μ

μ

μ

Particulate matter (PM 10)

μ

Particulate matter (PM 2.5)

μ

μ

TYPEs of particles deposited

Hydrocarbons

3.4.2 NON-CONVENTIONAL

Asbestos

μ

Health effects

-
-
-

Mercury

Beryllium

μ

-
-
-
-

Fluorides

-
-
-
-
-

-
-
-

Ozone

μ

μ

Physical Forms of Pollutants

Aerosols

particulates.

polydispersed)

Fumes

μ

inhalable fraction

μ

μ **thoracic fraction**

μ

μ

μ **respirable range.**

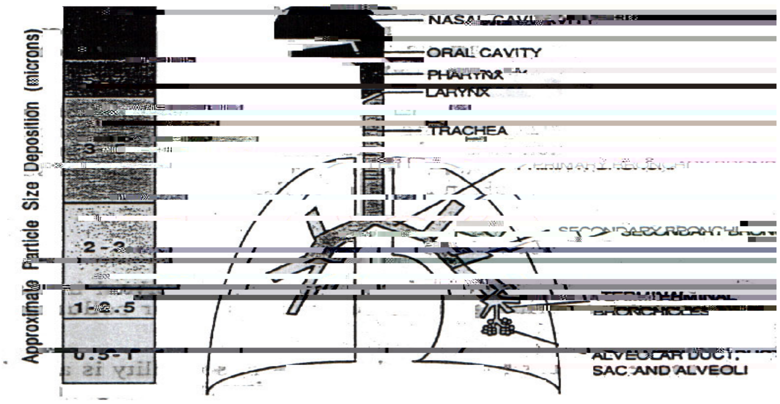


Figure 2: Deposition of Dust particles by size

fibers,

Liquid phase

droplets,

mist.

adsorption

hygroscopic.

vapor

3.5 Magnitude and Sources of Ambient Air Pollution

Table 3.3: Sources of Pollutant Emissions, United States, 1989

	Particulate Matter	Sulfur Oxides	Carbon Monoxide	Nitrogen Oxides	Volatile Organic
Transportation	25%	4%	66%	40%	35%
Fuel consumption	22%	80%	13%	56%	5%
Industrial	39%	16%	8%	3%	44%
Solid wastes	3%	0%	3%	1%	3%
Miscellaneous	12%	0%	11%	1%	14%

Table 3.4: Major types of occupational pulmonary disease

Pathophysiolog process	Occupational disease example	Clinical history	Physical examination	Chest x-ray	Pulmonary function pattern
Fibrosis	Silicosis	Dyspnea on exertion, shortness of breath	Clubbing, cyanosis	Nodules	
	Asbestosis	Dyspnea on exertion, shortness of breath	Clubbing, cyanosis, rales	Linear densities, pleural plawues, calcifications	
Reversible airway obstruction (asthma)	Byssinosis, isocyanate asthma	Cough, wheeze, chest tightness, shortness of breath, asthma attacks	Respiratory rate↑, wheeze	Usually normal	
Emphysema	Cadmium poisoning (chronic)	Cough, sputum, dyspnea	Respiratory rate ↑ ↑ expiratory phase	Hyperaeration bullae	
Granulomas	Beryllium disease	Cough, weight loss, shortness of breath	Respiratory rate ↑	low DLCO Usually decr042sd Tj0 -1.3759 TD.0026 Tc.uDLCO, Chypoxemia a	c - 3
Pulmonary edema	Smoke inhalation	Frothy, bloody sputum production	Coarse, bubble rales	Tw ( )Tj 0 -1.3802 TD .0020 Tc -.0038 Tw [(rDLf T7(COf T8(=)ffus	

Table 3.5: Common air pollutants, their sources and pathological effects on man

Ser. No.	Pollutants	Where they come from	Pathological effect on man
1	Aldehydes	Thermal decomposition of fats, oil or glycerol	Irritant nasal and respirator tract
2	Ammonia	Chemical processes dye-	

3.6. Exercise question

Study Exercise

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4.3. Types of Industrial Air Pollutants

Reducing

Table 4.1: Types of Air Pollution by Chemical Characteristics and Source

Type	Composition	Source
Reducing	Sulfur dioxide, particulates.	Stationary combustion sources, Such as fossil fuel power plants, industrial furnaces, home heating units.
Photochemical	Hydrocarbons and nitric oxide emitted by sources such as internal combustion engine undergo automobiles, fossil fuel power Complex photochemical reactions in the plants and oil refineries. Presence of sunlight, resulting in an atmosphere with significant concentrations of ozone, nitrogen dioxide, aldehydes, and organic nitrates.	Mobileemissions
Point Source	Specific to source of emission, e.g. lead Specific industries; near a smelter.	Industrial or transportation accidents.

Photochemical

point-source emissions.

BOX 2.

Bhopal – A Case Study of an International Disaster

Argualbly, the world's worst industrial cataclysm occurred on 2 December 1984 at the Union Carbide Plant in Bhopal, India, where a release of a gas cloud of methylisocyanate killed over 3800 people.

With respect to the historical facts leading up to the disaster, it is noteworthy that the post-World War II era witnessed a dramatic world-wide increase in the production of organic chemicals. The application of pesticides in particular was encouraged and became widely prevalent. Although the impacts on occupational and environmental health were beginning to be realized, the necessity for greater food production despite inadequate safety testing lead to the continued use of these chemicals in everyday life. Early in the 1970s governments of many developed countries recognized the need to adopt a proactive role for government intervention and regulation in this area. This, combined with the fact that markets in developed countries approached saturation, lead to the multinational corporations turning their attention to the developing world, where public health concerns for occupational and environmental health were low. These conditions lead to an increase in the international mobility of hazardous products, industry, and wastes to these lucrative markets of cheap labour, with only its costs and relative indifference to occupational and environmental health standards.

What was described in one report as a "normal accident" was apparently initiated by the introduction of water into the MIC storage tank, resulting in an uncontrollable reaction, with liberation of heat and escape of MIC and other decomposition products in the form of a gas. Safety systems were either not functioning or were inadequate to deal with large volumes of the escaping toxic chemicals.

Among the more than 200,000 persons exposed to the gas, the initial death toll within a week following the accident was over 25,000 by 1990, the Directorate of Claims in Bhopal had prepared medical folders for 361 966 of the exposed persons. Of these, 173382 had temporary injuries and 18922 had permanent injuries, with the recorded deaths totaling 3828.

One of the most important lessons of the Bhopal tragedy is how important it is to prevent these incidents by taking action in advance. Environmental legislation, preventive maintenance strategies,

worker-training programmes, environmental education programmes, research on intermediate products, development of systematic hazard-evaluation models, emergency planning, and disaster preparedness are all examples of such ac

4.5 Air Pollution in the Workplace

CHAPTER FIVE

GLOBAL ENVIRONMENTAL PROBLEMS DUE TO AIR POLLUTION

5.1. Learning Objective

5.2. Introduction to the chapter

5.3. Global warming

What is global warming?

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×

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×

×

×

×

Anthropogenic (man made) “green house gases” and their sources:

Potential health effects due to global warming

<u>General points:</u>

<u>Effects:</u> Direct and Indirect:

A/ Direct effects:

____

B/ Indirect effects:

5.4. OZONE DEPLETION

Identity

Its role

ü

ü

ü

ü

Mechanism for UV blocking:

ü

ü →

ü →

ü →

What causes Ozone depletion?

-
-
-

•

The reaction:

→

→

→

→

<u>Potential adverse effects:</u>

•

•

A/ Direct effect:

1. Skin damage and cancer:

•

•

•

Effects on eye:

3. **Effects on immune system**
    -

B/ Indirect effects:

-

-

INTERNATIONAL RESPONSE TO OZONE DEPLETION:

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Perspectives of operating in harmony with climate and weather

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5.5 Acid rain

ACID DEPOSITION

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-
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Extent of the problem

Formation of acid rain

Environmental effect of Acid Rain

1.

Plants

Aquatic Insects

Invertebrate on which fish dependent on food

2. Deterioration of buildings and monuments

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3. Mobilization of toxic metal

4. Damage to forest productivity

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5. visibility reduction

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5.6. Exercise question

EXERCISE QUESTIONS

CHAPTER SIX

INDOOR AIR POLLUTION

6.1. Learning Objective

6.2. Introduction to the chapter

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6.3. Environmental tobacco smoke

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6.4. Radon gas

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-

6.6. Asbestos

6.7. Lead

Table 6.2: Predicated Carboxyhaemoglobin Levels for Subjects Engaged in Different Types of Work

Carbon monoxide concentration		Exposure Time	Predicated COHb Level for those engaged in		
ppm	mg/m^3		sedentary work	light work	heavy work
100	115	15 minutes	1.2	2.0	2.8
50	57	30 minutes	1.1	1.9	2.6
25	29	1 hour	1.1	1.7	2.2
10	11.5	8 hours	1.5	1.7	1.7

Source: WHO, 1987a

carboxyhaemoglobin

Table 6.3: Human Health Effects Associated with Low-Level Carbon Monoxide Exposure: Lowest-Observed-Adverse-Effect Levels Carboxyhaemoglobin effects Concentration (%)

2.3 – 4.3	statistically significant decrease (3-7%) in the relation between work time and exhaustion in exercising young healthy men
2.9 – 4.5	statistically significant decrease in exercise capacity (i.e. shortened duration of exercise before onset of pain) in patients with angina and increase in duration of angina attacks
5 – 5.5	statistically significant decrease in maximal oxygen consumptions and exercise time in young healthy men during strenuous exercise
< 5	No statistically significant vigilance decrements after exposure to carbon monoxide
5 – 7.6	statistically significant impairment of vigilance tasks in healthy experimental subjects
5 – 17	Statistically significant diminution of visual perception, manual dexterity, ability to learn, or performance in complex sensorimotor tasks (e.g. driving)
7 – 20	Statistically significant decrease in maximal oxygen consumption during strenuous exercise in young healthy men

Source: WHO, 1987a

6.9. Biological Contaminants

6.10. Building materials, furniture’s and Chemical products

6.11. Sick Building Syndrome (SBS)

Table 6.4: Sources and exposure guidelines of indoor air contaminants

Pollutant and indoor sources	Guidelines, average concentrations
Asbestos and other fibrous aerosols Friable asbestos; fireproofing, thermal and acoustic insulation, decoration, Hard asbestos: vinyl floor and cement products.	0.2 fibers/ml for fibers longer than 5 µm
Carbon monoxide Kerosene and gas space heaters, gas stoves, wood stoves, fireplaces, smoking.	10 mg/m^3 for 8 hr, 40 mg/m^3 for 1 hr
Formaldehyde Particleboard, paneling, playwood,	120 µg/ m^3

carpets, ceiling tile, urea-formaldehyde foam insulation, other construction materials.	

6.12. Indoor air pollution in relation to developing countries

Indoor-air pollution in the context of developing countries

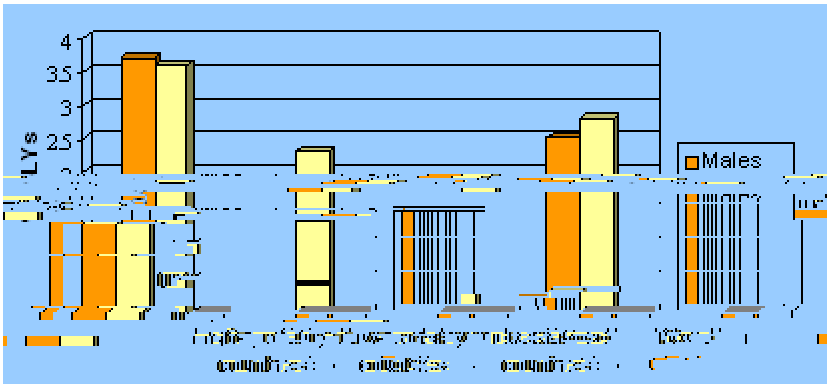
3.5 billion people

Threat to children's health

-
-

substantial technical and economic barriers

Disease burden by level of development



WHY DOES THE BURDEN PERSIST?

health **energy** **housing**
development

Interventions

Technical interventions

-

ü

ü

ü

cor pulmonale *cor pulmonale*

ü

ü

ü

HEALTH EFFECTS OF WOOD SMOKE

6.13. Exercise questions

CHAPTER 7

RISK ASSESSMENT

7.1. Learning Objective

Table7.1. Several of the Many Sources of Uncertainty in a Risk Assessment

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7.4.2. Study Methods

Table 7.2. Study Designs in Environmental Epidemiology That Use the Individual as the Unit of Analysis

Study design	Population	Exposure	Health effect	Confounders	Problems	Advantages
Descriptive study	Community or various sub-populations	Records of past measurements	Mortality and morbidity statistics; case registries; case registries; other reports	Difficult to sort out	Hard to establish exposure-effect relationships	Cheap, useful to formulate hypotheses
Cross-sectional study	Communities or special groups; exposed vs. non-exposed	Current	Current	Usually	Current	Can be done quickly; can use large populations; can estimate prevalence
Prospective cohort study	Community or special groups; exposed vs. non-exposed	Defined at outset of study (can change during study)	To be determined during study	Usually easy to measure	Expensive, tie consuming exposure categories can change; high dropout rate possible	Can estimate incidence and relative risk; can study many diseases in one study; can describe associations that suggest cause-effect relationships

Historical cohort study	Special groups e.g. workers, patients, insured persons	Records of past measurement	Records of past or current diagnosis	Often difficult because of retrospective nature; depends on disability of previously obtained data	Need to rely on records that may not be accurate	Less expensive and quicker than prospective study; can be used to study exposures that no longer exist
Case-control study	Diseased (cases)vs. non-diseased (control)	Records or interview	Known at start of study	Possible to eliminate by matching	Difficult to generalize; may incorporate biases; cannot derive rates	Relatively cheap and quick; particularly useful for studying rare diseases
Experimental (interventions study)	Community or special groups	Controlled /known already	To be measured during study	Can be controlled by randomization of subjects	Expensive; ethical consideration; study subjects' compliance required	Well accepted results; strong evidence for causality or efficacy or intervention

Source: WHO, 1991a

An Approach to classification of studies designs

Table 7.3. Assessing the Quality of Historical Cohort Studies

Figure 7.1:

RATE OF DISEASE:	$\frac{\text{Number of cases of disease in population at risk}}{\text{Number of persons in population at risk}}$
EXPRESSED AS:	$\frac{\text{Number of cases}}{\text{100 or 1000, etc. persons at risk}}$
EXAMPLE:	$\frac{\text{50 cases}}{\text{2500 persons at risk}} = \frac{20}{1000}$
RISK RATIO:	$\frac{\text{Rate of disease in population with the risk factor}}{\text{Rate of disease in population without the risk factor}}$

risk ratio

BOX 7.1

—

Attributable Fraction (Exposed) = $\frac{(E-U)}{E}$ =(through mathematics) $\frac{(RR-1)}{RR}$

Attributable Fraction (Population) = $\frac{I - U}{I}$ = $\frac{[p(RR-1)]}{[p(RR-1)+1]}$

Where U = incidence (or mortality) in the unexposed group;

E = incidence (or mortality) in the exposed group;

P = prevalence in the total population;

standardized mortality ratio (SMR)

$$\text{SMR} = \frac{\text{Observed number of deaths (or events) in the study populationx100\%}}{\begin{array}{l}\text{Expected number of deaths (or events) if the study population had the same}\\ \text{age and gender composition as the comparison (e.g. national) population}\end{array}}$$

confidence interval

confidence interval

Basic Epidemiology,

Environmental Epidemiology,

7.4.4. Study Difficulties and the Determinants of Causation

Environmental Epidemiology

association **causation**

Table 7.4: Tests of Causation

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α β

7.5. Hazard identification in the field

health hazard evaluations and hazard audits

walk-through

7.5.1. Occupational Environment

7.5.2. General Environment

marker

7.6. The relationship between dose and health Outcome

7.6.1. Dose-Effect and Dose-Response Relationships

Table 7.5: Range of Effects on Human Health Due to Environmental Exposure

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7.7. Human exposure assessment

7.7.1 Options in Approach

7.7.2. Personal Exposure Monitoring

7.7.3..Biological Monitoring

Table 7.6: Examples of Useful Markers of Exposure

Substance	Biological Marker

7.7.4. The Indirect Approaches to Estimation of Exposure

estimation

time activity diaries

observational

7.7.5. Estimating Inhalation Exposure

BOX 3.6

Estimating Lead Intake Via Inhalation by a Child

Task: estimate the cumulative dose of inhaled lead for an 11-year-old child who has been exposed for two hours per day every day since birth, to lead in outdoor air at a concentration of $8x10^{-5}$ mg/m^3. Exposure ended at age 12 when the family moved to another area.

The cumulative dose is calculated as follows:

The inhalation rate (IR) of contaminated air is a fraction of the total air breathed, in this case 2 hours of exposure /day x total daily amount of air inhaled. The total daily amount of air inhaled changes as a child grows (see Table 3.11). Multiplying each of these values by 2/24 (0.083) gives an IR of 0.166m^3

7.7.7. Principles of Environmental Sampling

7.7.8. Ensuring Adequate Sample Size

7.8. Health risk characterization

7.8.1. General Approach Summarized

Table 7.9: Consecutive steps in Health Risk Characterization

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-
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-

7.9. Health in environmental impact assessment (EIA)

7.10. Exercise questions

CHAPTER EIGHT

SAMPLING AND ANALYSIS

8.1. Learning Objective

8.2. Introduction

Table 8.3: WHO Air Quality Guidelines for Europe, Revised 1994

Compound	Guideline	Value	Averaging Time
Ozone	120›g/m^3	(0.06 ppm)	8 hr
Nitrogen dioxide	200›g/m^3	(0.11 ppm)	1 hr
	40 to 50›g/m^3	(0.021 to 0.026 ppm)	Annual
Sulfur dioxide	500 ›g/m^3	(0.175 ppm)	10 min
	125›g/m^3		

8.4. Exercise questions

CHAPTER NINE

AIR POLLUTION PREVENTION AND CONTROL

9.1. Learning Objective

9.2. Introduction to the course

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9.3 Control of Ambient Air Pollution

4. Respirable usage

Box 3

Motor vehicle Air Pollution: Health Effects and Control Strategies

Studies of human exposures to air pollution from motor vehicles have revealed the following:

- Concentrations of some air pollutants inside motor vehicles and along roadsides are typically higher than those recorded simultaneously at fixed-side monitors.
- Exposures tend to be higher inside automobiles than in buses and other vehicles used in public transit.
- Priority lanes used to afford speed advantages to buses and car pools tend to reduce air pollutant exposures.
- Concentrations of air pollutants in enclosed settings are similar to outdoor concentrations in the absence of indoor sources, but tend to lag behind the peak concentrations observed outdoors. (A notable exception is commercial buildings attached to inadequately ventilated parking garages.)
- Concentrations of motor vehicle air pollutants decline with greater distance from the road, suggesting that passengers and vehicles are at greatest risk, followed by pedestrians and street merchants along roadsides, and then the general urban population.

Motor vehicle emissions may be reduced by: 1) controlling vehicle performance, and 2) altering fuel composition. With respect to vehicle performance, this can be controlled by ensuring that vehicles are designed and built to meet standards. It is also necessary that they be properly maintained. Proper maintenance, in turn, can be promoted by providing incentives to car owners to obtain proper maintenance and by marketplace incentives. Requiring maintenance through a mandatory inspection and maintenance programme is considered by many to be the most effective incentive for car owners.

Fuel composition may be controlled as a direct means of controlling emissions, 01 Cg. d bucg emth6( )e leadontrotiv6.5( )] prlead asgaso6(l)1l

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9.4. Exercise question

Study Exercise

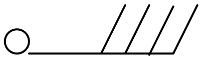
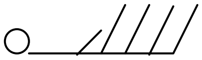
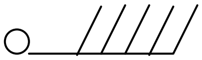
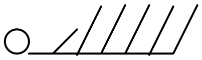
REFERENCES

APPENDIX

Annex-1

Weather- man wind measuring reports system

Beaufort numbers	Map symbol for wind speed and direction	Speed M.P.H	Description	Specification
0	0		calm	Smoke rises vertically
1		1 to 3	Light air	Wind direction shown by drift of smoke
2		4 to 7	Slight breeze	Wind flet on face; leaves rustle; flag stirs
3		8 to 10	Gentel breeze	Leaves & twigs in constant motion; wind extends light flags
4				

8		39 to 46	Fresh Gale	Leaves & twigs in constant motion; wind extends light flags
9		47 to 54	Strong gale	Slight damage to house
10		55 to 63	Whole gale	Trees uprooted; much damage to house
11		64 to 75	Storm	Widespread damage
12		Over 75	Hurricanes	Excessive damage

Annex-2

Some questions worth asking about fuel, cooking and ventilation

Type of fuel

Type of stove

Location

Uses

Ventilation

Fuel gathering

Annes-3

INDOOR AIR SAMPLING PROCEDURE

I. Gravimetric sampling technique

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