CE 213A
Introduction to Environmental Science

L19
Unit 3: Impact of Pollution
Particle Deposition in Respiratory Tract

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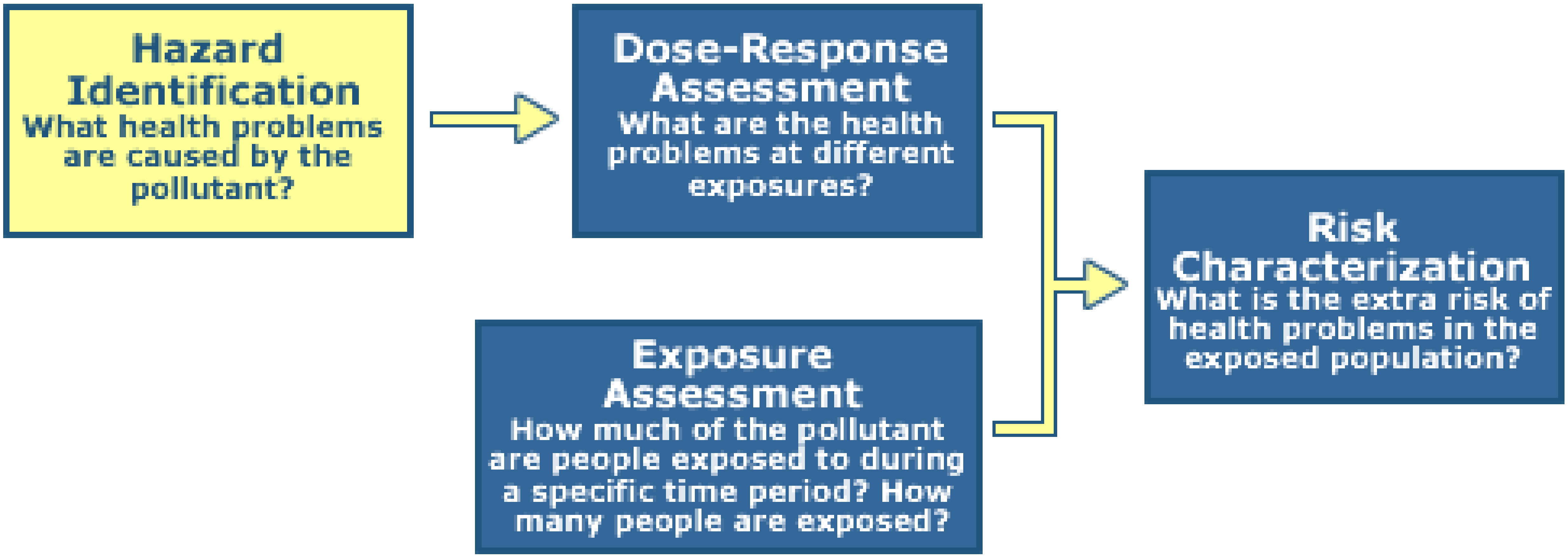
Schedule : LEC Mon Wed Fri 5:10 – 6 pm

Deposition of particles in human respiratory tract
Assessment of health impacts

The 4 Step Risk Assessment Process



Brief Statistics- Air Pollution

- **International Agency for Research on Cancer** (IARC), specialized cancer agency of the World Health Organization, in October 2013 **classified outdoor air pollution as Group 1 carcinogenic to humans**.
- An earlier study by the agency in 2010 on the link between air pollution and cancer and reported that 223,000 deaths from lung cancer worldwide resulted from air pollution[1].
- In 2012, indoor air pollution was linked to 4.3 million deaths globally, compared with 3.7 million for outdoor air pollution.
- Carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter, and sulfur dioxide are the 6 **criteria pollutants** present in ambient air
- Particulate matter causes most adverse impacts on human health as compared to other criteria pollutants
- A Study reported that Particulate matter alone is responsible for around 2.1 million of premature deaths per year globally.

HOW INDIA IS GETTING CHOKED

- **Delhi and Ghaziabad among five top critically polluted cities**
- Deaths caused by particulate air pollution increased by six times since 2000
- **Respiratory and cardiovascular diseases key reasons for air pollution-induced premature deaths: These diseases include stroke (25.48%), chronic obstructive pulmonary disease (17.32%), Ischemic heart disease (48.6%), lower respiratory infections (6.4%), and trachea, bronchus and lung cancer (2.02%)**
- Globally, air pollution-related deaths increased by 300 per cent since 2000. About 65 per cent of these deaths occur in Asia
- **A Global Burden of Disease report says that in 2010, about 6.2 lakh premature deaths occurred in India from air pollution-related diseases. This is up from 1 lakh in 2000 — a six-fold increase**
- One-fifth of global deaths due to pollution in India
- **CSE's quality analysis shows half of urban population breathes air with particulate levels that exceed the permissible limit**

World's most polluted

1. Delhi	11. Ferozabad
2. Patna	12. Doha
3. Gwalior	13. Kanpur
4. Raipur	14.Amritsar
5. Karachi	15. Ludhiana
6. Peshawar	16. Igdir
7. Rawalpindi	17 Narianganj
8. Khorramabad	18 Allahabad
9. Ahmedabad	19. Agra
10. Lucknow	20. Khanna

Straif et al., 2013; WHO, 2013

Health Impacts

- Particulate matter exposure has two types of health effects.
- **Acute effects** are immediate and short term due to high exposure concentration.
  - ✓Bronchospasm, pulmonary oedema, allergic alveolitis
  - ✓Runny nose,sore throat, wet cough
  - ✓Head cold, hay fever, red eyes sinusitis.
- **Chronic effects** are long-term ailments due to a certain trigger and serious conditions due to low exposure concentration but exposed constantly to long time.
  - ✓Chronic obstructive pulmonary diseases (COPDs) include bronchitis or emphysema
  - ✓Damage to lungs, to blood, nervous system, liver, kidneys, bones and skin
  - ✓Altered host defence mechanisms
  - ✓Cancer
  - ✓Respiratory problems
  - ✓low birth weight and infant mortality.

Particulate Matter and its Sources

- Combination of solid particles and liquid droplets in the air.
- Composed of different size particles like ultrafine, fine and coarse which are formed either by **dispersion or reaction/condensation mechanism**. These **sizes vary** from **place to place** and **time to time** depending on the **sources (natural or anthropogenic)**.
- Indoor sources
  - ✓ Combustion events; Resuspension during movement
  - ✓ Outdoor sources make way to indoor through ventilations, windows etc.
- Outdoor sources
  - ✓ Road traffic; Resuspension of road dust
  - ✓ Construction and agricultural activities
  - ✓ Power plants, industrial processes, biomass burning etc.,
  - ✓ Secondary pollutants formed through the photochemical transformations of gas phase species.
- Comprise of the primary Heavy metals and Poly-aromatic Hydrocarbons (PAH), Water soluble ions, Organic carbons etc.,

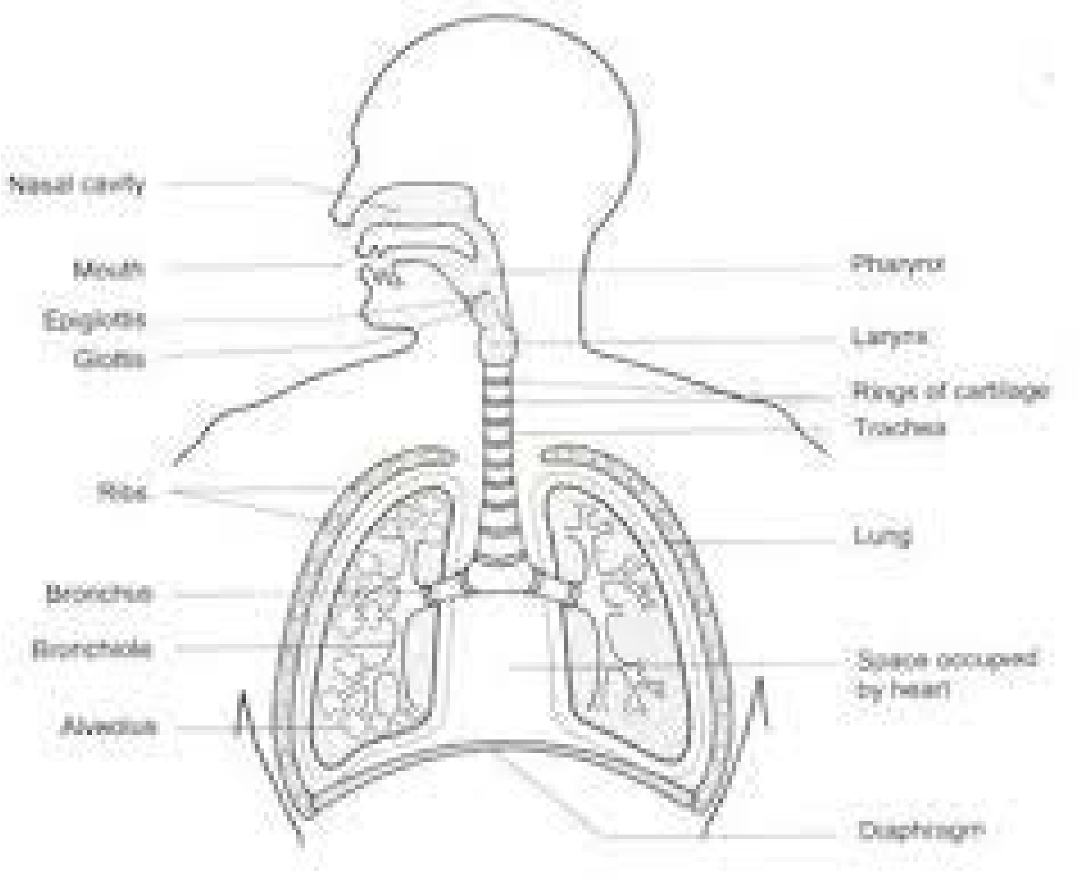
Why Particle Size a concern?

- Particle size have a significant influence on assessment of human risk, **as aerodynamic diameters decide where each particle is likely to deposit in the human respiratory system**[1].
- Several epidemiological studies have suggested a statistical association between health effects and ambient fine particle concentrations[2].
- It has been reported that most **of** the **toxic metals accumulate in the smaller particles** ($PM_{2.5}$ or less) which have been reported to cause carcinogenic and non carcinogenic impacts[3].
- **The United States Environment Protection Agency (US EPA) recommended in a recent report emphasised that submicron particles ($PM_1 \leq 1$ µm) could be used as the standard cut-off point for fine particles, as human respiratory symptoms are highly linked with PM_1 levels**[4].

Cao et al., 2012; Pope, 2000; Ravindra et al., 2008; Hieu and Lee, 2015

Human Respiratory System

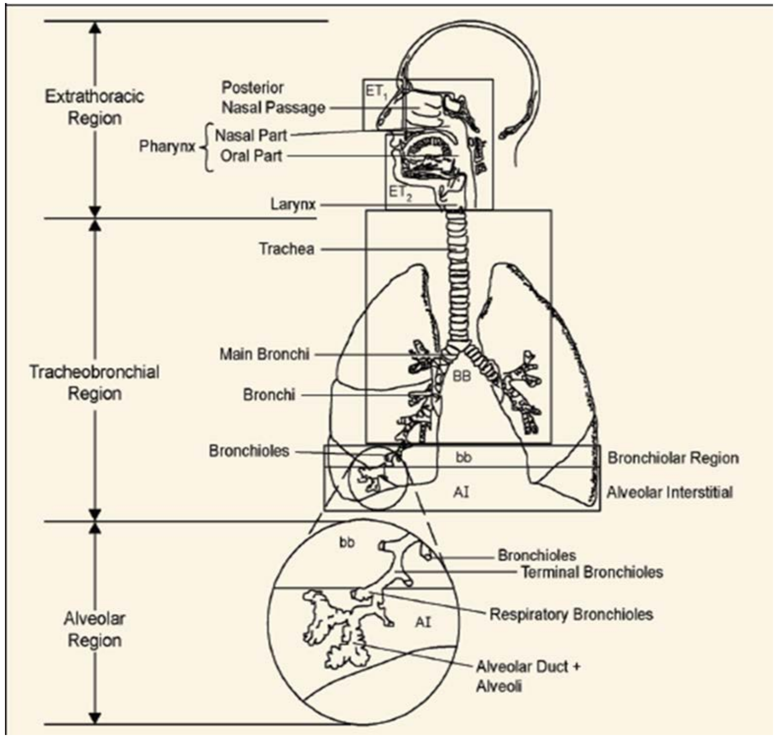
- https://www.youtube.com/watch?v=hc1YtXc_84A



Human respiratory system

Mechanisms of particle Deposition

- Different HRT regions imply different deposition mechanisms, with regard to different particle size.
- Head: impaction, sedimentation, electrostatic
- Tracheobronchial: impaction, sedimentation, diffusion
- Pulmonary: sedimentation, diffusion



Dominant mechanism:

- **Diffusion** is the deposition mechanism for small particles.
- **Sedimentation** occurs when gravitational force act on the particles.
- **Impaction** occurs when particle cannot follow the trajectory due to its **inertia** and hit the wall.

Impaction increases with particle size and flow rate.

Deposition Modelling

- **Experimental techniques** available today are **not sufficiently sensitive** for **quantitative assessment of local or regional deposition** in the Human respiratory tracts.
- However, several mathematical models have been developed over the years which are capable of predicting total and regional deposition.
- A deposition model is a computational scheme that includes: **morphometry of the respiratory tract, respiratory physiology, particle characteristics.**
- **These models** outputs including both total and regional deposition have been **compared to experimental studies, with a fair correlation** being obtained between model predictions and experimental measurements.

Health Risk Assessment

- Carcinogenic risk
- Non carcinogenic risk

ICRP, 1994

Earlier studies (Deposition of particles)

- In a study reported at South Texas, USA, for **school children** in different microenvironments, it was found that **maximum pulmonary deposition occurred and it gradually decreased with the age of children**[1].
- In another study done at Budapest, Hungary for women at different microenvironments for the particle size ranged from 6-1000 nm, it was reported that mean **deposition fraction** performed by MPPD model in the **extra-thoracic region decreased from 26% for sleeping to 9.4% for heavy exercise. Also, in pulmonary regions mean deposition fraction increased from 14.7% for sleep to 34% for heavy exercise**[2].
- In another study at Singapore, deposition fraction were calculated for particle size which were categorised into coarse (2.5-10 µm), fine (0.1-2.5 µm), ultrafine (0.01-0.1 µm) and nano (0.01-0.056 µm) particles. Results showed that **among all sizes coarser particles had highest deposition at head region (34 to 39 %) followed by finer particles (22 to 25 %). Further ultrafine (21 to 25 %) and nano particles (25 to 28 %) were found** to **be deposited at pulmonary region**[3]**.**
- **Also study reported at Barcelona, Spain, people commuting through subway and train had deposition of** $PM_{2.5}$ **around 68% extrathoracic region, 4% tracheobronchial region and 10**% at alveoli region[4].
- In a study done at USA, reported that while sleeping the combustion of woods at wintertime causes deposition in adults for $PM_{1.8}$ about 32% in pulmonary region and 6% in tracheo-bronchi region[5].

Patterson et al., 2014; Salma et al., 2015; Behera et al., 2014; Sanchez-Soberon et al., 2015; Ham et al., 2010

Exposure to particle bound metals

- The model used in this study to compute the exposure of humans to heavy metals in ambient particulate matter is based on those developed by **USEPA human health evaluation method**.
- Exposure pathways considered
  - Inhalation
  - Ingestion
  - Dermal Contact
- Exposure is defined in terms of daily dose and computed **individually for each metal and each exposure pathways**.
- $ADDing = \frac{C*IngR*EF*ED*CF}{BW*AT}$
- $ADDinh = \frac{C*InhR*EF*ED}{BW*AT*PEF}$
- $ADD_{derm} = \frac{C*SA*AF*EV*ABS*EF*ED*CF}{BW*AT}$

US EPA, 2007; US EPA, 2009

Parameter	Definition	Values
C	Concentration of metal in PM_1 (mg kg^{-1})	
IngR	Ingestion rate (mg day^{-1})	30 (adults), 60 (children)
InhR	Inhalation rate (m^3 day^{-1})	7.63 (adults), 20 (children)
EF	Exposure frequency	180 days/year
ED	Exposure duration (year)	24 (adults), 6 (children)
BW	Body weight (Kg)	70 (adults), 15 (children)
SA	Skin surface area (cm^2)	5700 (adults), 2800 (children)
AF	Adherence factor of soil to skin ($mg/cm^2/event$)	0.07 (adults), 0.2 (children)
EF	Events frequency	1events/day
ABS	Dermal absorption fraction	.001
PEF	Particle emission factor (m^3/kg)	$1.36 * 10^9$
AT	Averaging time for Non-carcinogens (days/year)	ED $*$ 365
CF	Conversion Factor (kg/mg)	10^{-6}.

Non Carcinogenic Health Risk

- Hazard Quotient (HQ) and Hazard Index (HI) are used to measure the non-carcinogenic health risk of heavy metals in ambient particles.

$$HQ = \frac{ADD}{RfD}$$

$$HI = \sum_{i=1}^{i=3} HQ_i$$

- Where RfD is the reference dose which is an approximation of maximum allowable risk on human population through daily exposure

If HI < 1
- No adverse impact on health

Else HI > 1
- Chances persist for adverse health impact

Zheng et al., 2010

Risk Assessment for Carcinogens

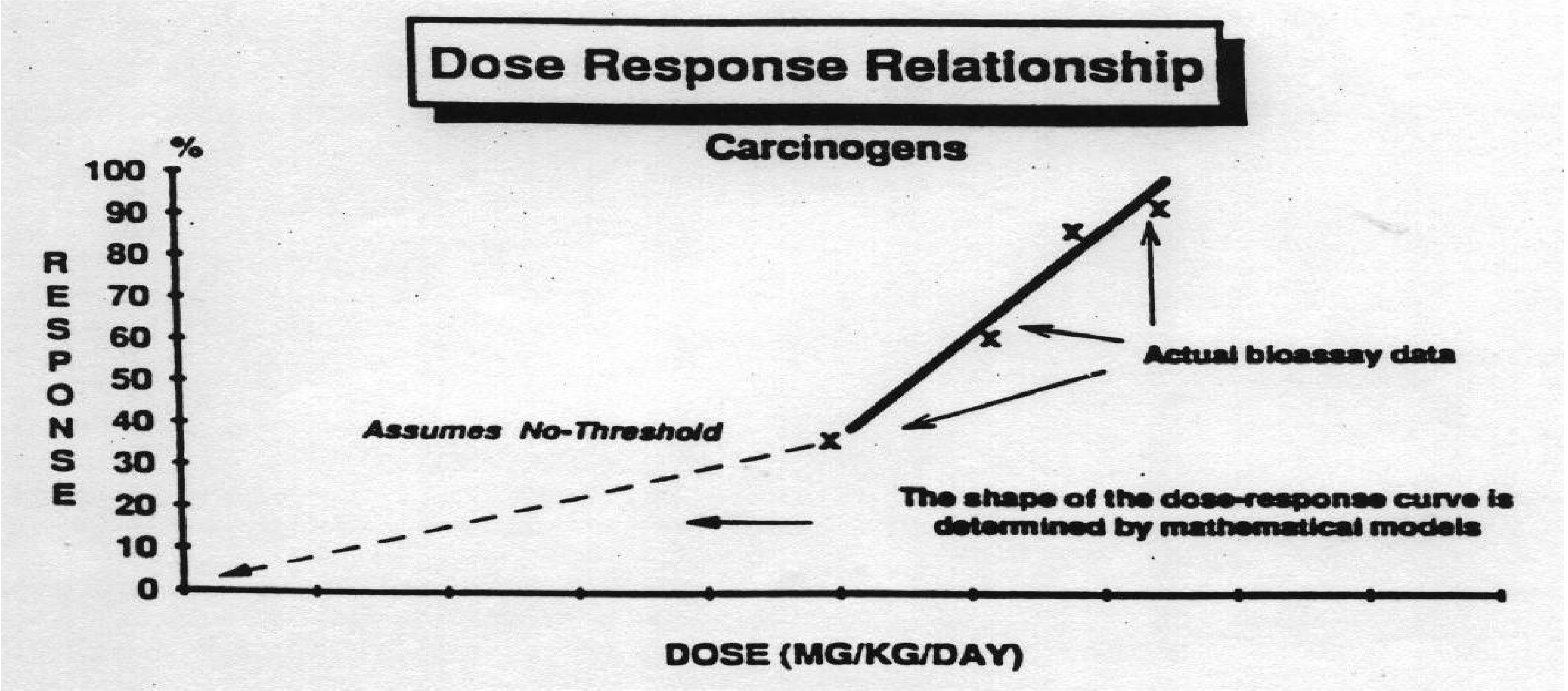
Home Assignment

Risk Assessment for Carcinogens

- https://www.epa.gov/fera/risk-assessment-carcinogens

b. Carcinogenic Effects

- Mathematical models are used to extrapolate from the high doses used in animal experiments to the low doses to which humans are normally exposed in a chronic setting.



Carcinogenic Effects - Continued

TABLE 21.5. Primary Models Used for Assessment of Nonthreshold Effects

Linearized multistage model	Assumes that there are multiple stages for cancer Fits curve to the experimental data Linear from upper confidence level to zero
One hit model	Assumes there is a single stage for cancer and that one molecular or radiation interaction induces malignant change Very conservative
Multihit model	Assumes several interactions needed before cell becomes transformed Least conservative model
Probit model	Assumes probit (log-normal) distribution for tolerances of exposed population Appropriate for acute toxicity; questionable for cancer
Physiologically based pharmacokinetic models	Incorporates pharmacokinetic and mechanistic data into the extrapolation Data rich requirements and, while promising, are currently of limited availability

Slope factor

- The key risk assessment parameter derived from the carcinogen risk assessment process is the “slope factor”. The slope factor is a toxicity value that quantitatively defines the relationship between dose and response.
- A plausible upper bound estimate of the probability that an individual will develop cancer if exposure is to a chemical for a lifetime of 70 years.

Slope factor

- Slope Factor = a plausible upper-bound estimate of the probability of a response per unit intake of chemical over a lifetime.
  - Risk per unit dose
  - Units of Risk $(mg/kg\text{-}day)^{-1}$
  - Symbol for Slope Factor = q_1^*

ILCR – Incremental Lifetime Cancer Risk

- For carcinogens, the estimated exposure will be multiplied by the appropriate *Cancer Slope Factor* or *Unit Risk* to derive an estimate of the potential Incremental Lifetime Cancer Risk (ILCR) associated with that exposure (Health Canada 2004).
- The ILCR is derived as:

ILCR = Exposure (µg/kg/d) x Cancer Slope Factor (µg/kg/day)$^{-1}$

Where pathway-specific slope factors or unit risks exist, the risks via inhalation and the risks via oral and dermal exposure should be estimated separately. In other cases, the cancer risks posed by simultaneous inhalation/dermal/oral exposure can be estimated.

ILCR – Incremental Lifetime Cancer Risk

Case: Persistent Organic Pollutants like DDT

- Cancer risks will be considered "essentially negligible" where the estimated ILCR is 1-in-100,000 ($\leq 1 \times 10^{-5}$) (Health Canada 2004).
- If the ILCR is greater than 1×10^{-5}, the risk assessment should either be refined and/or risk management measures should be taken.

http://www.popstoolkit.com/riskassessment/module/risk+characterization/ilcr.aspx

Effect in DF due to change in inhalation pathway

- Humans have the ability to breathe either nasally or orally or both.
    - ➢ oral (completely through mouth)
    - ➢ oronasal (50 percent through nose and rest through mouth).
- The DF for Heavy activity level at Play court has been considered to observe the variation.
- The DF at Head region followed the order as: Nasal>Oronasal>Oral. The higher deposition efficiency of nasal breathing is due to increased particulate matter removal by nasal hairs, impaction on pathway walls, and sedimentation.
- For the TB and P region DF order followed were: Oral>Oronasal>Nasal.
- Thus the route of breathing influences the quantity of inhaled contaminants delivered to human lungs.
- **Since particles deposition at pulmonary region is more prone for bad health effects due to its high retention time so it is preferable to inhale through nasal pathway most of time where the PM concentration is high.**