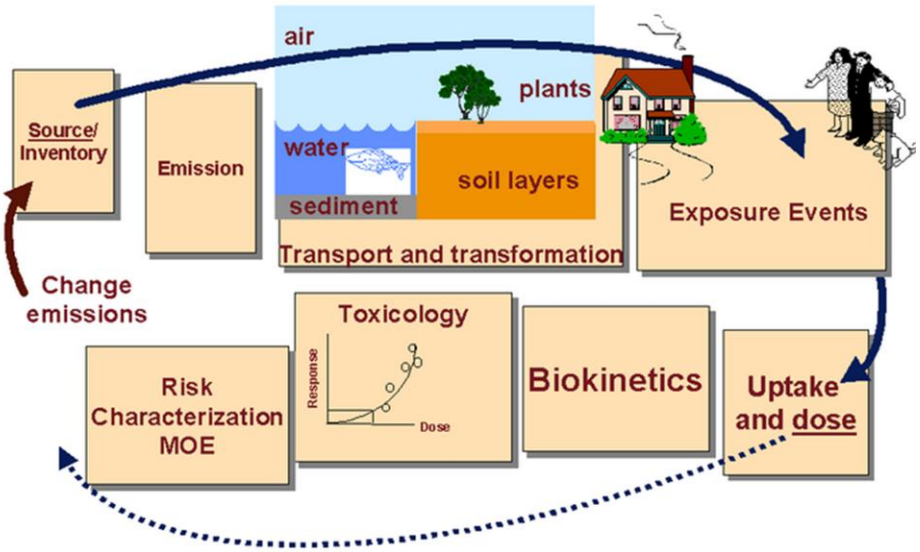

TOPIC V: ENVIRONMENTAL RISK ASSESSMENT

Content: Defining risk and environmental risk, methods of risk assessment, case studies

Source for graphic above: www.energy.lbl.gov

ABSTRACT

This module deals with the risk associated with hazardous waste, both to the environment as well as to human health.

IITM-EWRE

Solid and Hazardous Waste Management

What is Risk?

Risk can be quantified. Imagine a scenario where there is a source of hazardous waste, a pathway for its transport, and receptors such as human beings. Hazard is the intrinsic capability of the waste to cause harm. Hazard does not represent risk unless exposure or possibility of exposure occurs.

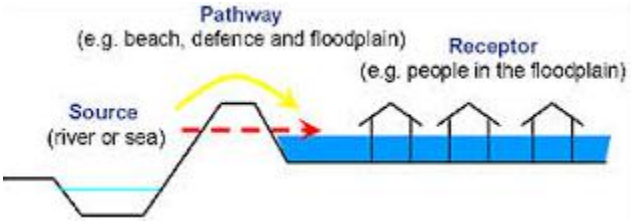

Fig. 1: Migration of contaminants
Source: Source-pathway-receptor-consequence model www.floodsite.net

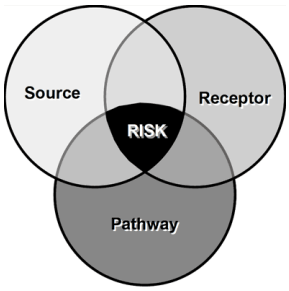

Fig. 2: Representing risk as a quantity
Source: www.landandgroundwater.com

How can risk be quantified?

- Hazard identification
  - Source of contamination
  - Properties of contaminants
  - Quantity and distribution of hazardous waste
- Exposure assessment
  - Transport and transformation of contaminants (pathways)
- Toxicity assessment
  - Receptor at source and along pathway/ at exposure
  - Response to exposure is studied
  - Based on response, critical concentrations (critical dose) of contaminants are identified
- Quantify risk
  - Based on above three

Applications of results of risk quantification:

- Selection of waste management options
- Remediation of contaminated sites
- Setting up of new waste treatment facilities
- Establishment of clean-up standards

Effect of toxicity on human body:

Susceptibility to toxic compounds follows a *Gaussian profile.*

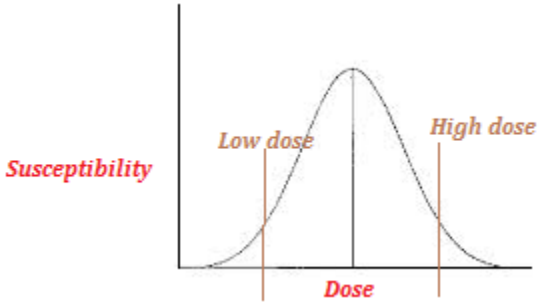

Fig. 3: Susceptibility vs. dose curve

A steep Gaussian curve leaves no doubt about the toxicity of a compound (e.g. – cyanide); whereas a compound such as ethanol has a flatter/ broader curve (difficult to define critical dose in this case).

Parameters for toxicity quantification:

- **Target organs**
  - Specific contaminants attack particular organs in the human body – for example, intake of lead damages the nervous system, and benzene can lead to blood cancer
- **Mode of entry into the body**
  - Inhalation – The contaminant goes directly into the alveoli and subsequently the blood steam. Example – fine particles of silica can be inhaled. Inhalation results in the worst impact.
    - $Inhalation\ rate = Contact\ rate\ \left(\frac{m^3}{d}\right) * Concentration\ \left(\frac{mg}{m^3}\right)$
  - Dermal contact – Toxic organic compounds are mostly nonpolar and soluble in lipids. Polar compounds can diffuse through the skin but are not soluble in lipids. Hence the lipid layer acts as a barrier. Dermal contact is a medium impact mode of entry.
    - The output of exposure assessment for dermal exposure is expressed as the amount of substance absorbed per kg body weight per day.
    - Soil Matrix (SM) - % of soil that is contaminated (ranges from 0.1 to 1).
    - ABS is the Absorption Constant or Absorption Rate.
    - Dust adherence (g/m^2) is the amount of soil adhering to the skin.
  - Ingestion – The contaminant affects the lining of digestive organs and excretory system. This is the least impact mode of entry.
    - $Ingestion\ rate\ (IR) = Contact\ rate * Concentration$
    - For water: $[IR] = \frac{L}{d} * \frac{mg}{L}$
    - For fish: $[IR] = \frac{g}{d} * \frac{mg}{g}$
- **Dosage**
  - $Dose = \frac{Mass\ of\ contaminant\ (mg)}{Body\ mass\ (kg)}$
  - Different target organs may have different thresholds (dosage levels) for them to be affected by the contaminant
  - Lethal Dose (LD_{50}) – Dose at which 50% of the population is affected, resulting in death sometimes; follows log normal distribution
  - LC_{50} – Concentration of contaminant in water (in mg/m^3 or mg/L) equivalent to LD_{50} in air

Table 1: Lethal dose of selected chemicals for human populations

Contaminant	LD_{50} (mg/kg)
Parathion	2
Ethanol	13000
Table salt	3800
Aspirin	1500
Nicotine	50-60
Dioxin -Pig -Rat -Hamster	0.02-0.05 0.001 0.02-0.05 5

- **Elimination mechanism** – The rate at which a toxic substance is removed from the body determines whether it will have a toxic effect. The main way a contaminant is removed is through urine. The kidneys thus eliminate the greatest amount of toxins from the body. Lungs eliminate chemicals in the gaseous phase (CO_2). Liver removes substances like lead and DDT by excreting them into bile, which is then carried to the small intestine from where it is removed as feces. Skin, hair and breast milk are other minor pathways for elimination.

Exposure Pathway

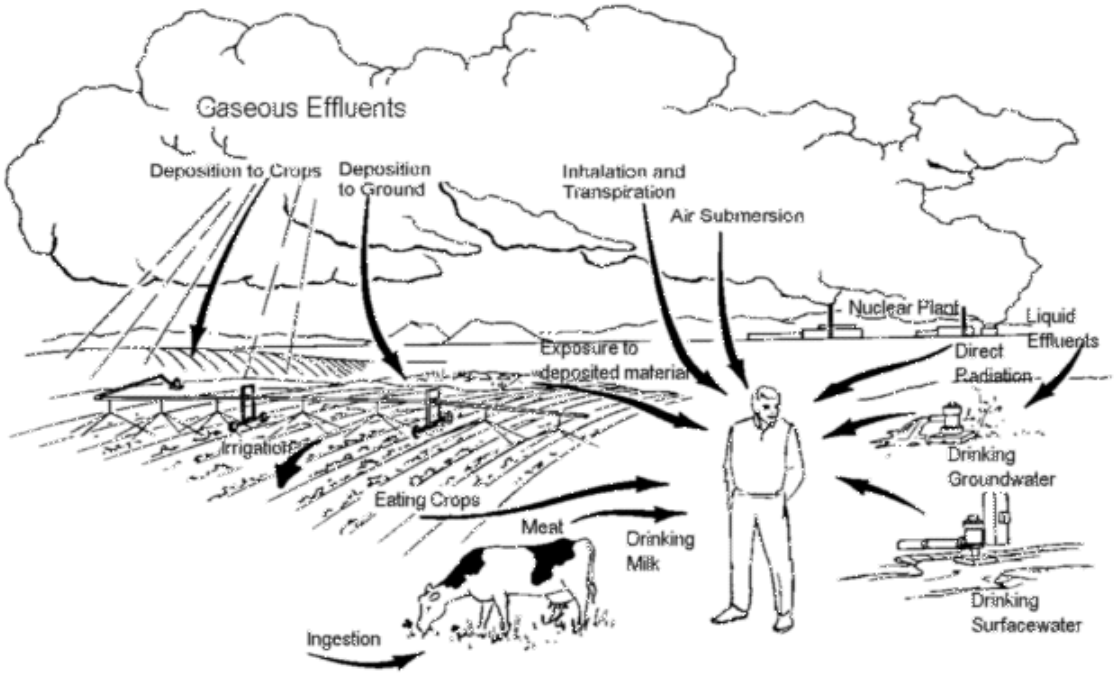

Fig. 4: Components of an exposure pathway
Source: What is environmental health? www.ehib.org

An exposure pathway should specifically examine the following points:

- The contaminant source or release – Sources may include storage containers, landfills, and so on.
- Environmental fate and transport – Once released into the environment, contaminants move through different media.
- Exposure point or area – Specific locations where people may come into contact with contaminants.
- Exposure route – Route by means of which people physically contact environmental contaminants at the exposure point (inhalation/ ingestion/ dermal contact).
- Potentially exposed populations – Identifying and characterizing populations that may have come into contact with contaminants.

Types of exposure:

- Acute – Exposure to a chemical for 24 hours or lesser (high dose, short term) o
    - Less than 5% of human life span
- Chronic – More than 3 months (low dose, long term)
    - Greater than 20% of human life span
- Sub-acute – For 1 month or lesser
- Sub-chronic – Between 1 and 3 months

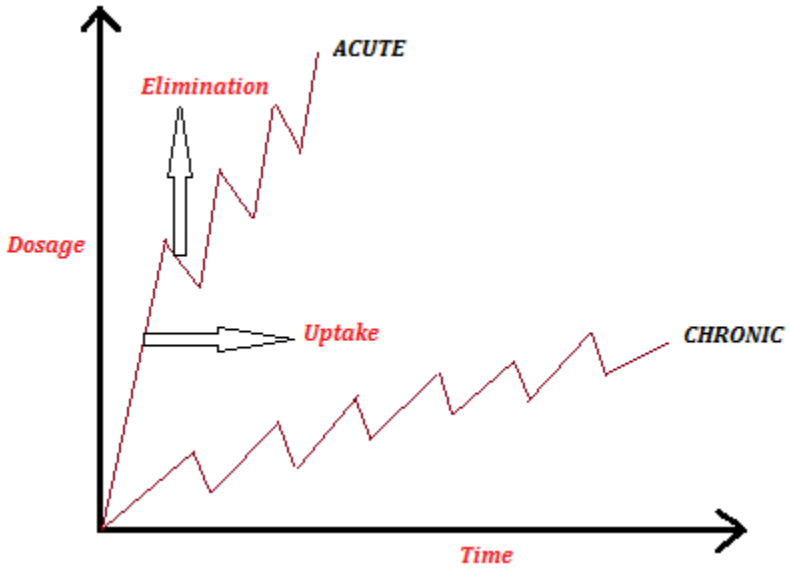

Fig. 5: Graph showing difference between chronic and acute exposure

Biomagnification:

Even though contaminants are never released directly to fish, animals, or plants, fate and transport processes sometimes make food-chain-pathway the most important. For instance, though the source of contamination at a site might be limited to wastewater discharge of PCBs to surface water, these contaminants can biomagnify resulting in relatively high concentrations in fish (subsequently eaten by humans). Presence of lipids enhance the absorption of these contaminations into the body. Polar compounds are readily excreted and often do not undergo transformations.

Compounds with high octanol-water partition coefficient undergo two-phase transformations:

Phase 1: *Oxidation, hydrolysis*

Phase 2: *Conjugation by addition of polar functional groups*

For example: Benzene → Benzene oxide → Phenol

Bio-transformation primarily takes place in liver and kidneys which have bio-activation enzymes. The intermediate products may be toxic.

Effects of exposure to toxic chemicals

Characterization of any chemical is done based on its partitioning coefficient across various media, its pathway or location, tracking the critical pathways involved and assessing the exposure level at receptor, and finally analyzing the impacts (carcinogenic or non-carcinogenic). This is an important procedure for any contaminant.

Table 2: Hazardous compounds and receptors in the human body

Compound	Receptor
Benzene	Bone marrow, stem cells
Hexane	Nervous system
PAHs	Lungs, skin
PCBs	Liver, lungs
Pb, Rd, F	Bones
Cd	Kidneys

Effects can be carcinogenic (cancer causing) or non-carcinogenic (non-cancer causing). While describing non-carcinogenic effects, the following terminologies are used:

- NOEL – No Observed Effect Level (dose at which there is no response)
- NOAEL – No Observed Adverse Effect Level
- LoEL – Low Effect Level (lowest dose tested for which effects were expressed)
- LoAEL – Low Adverse Effect Level
    - NoEL<LoEL<NoAEL<LoAEL
    - LoAEL is mostly used for fixing a concentration which necessitates clean-up of site.

Site-specific cancer doses and concentrations can be quantitatively found.

$$Theoretical\ cancer\ risk = Exposure\ of\ cancer\ risk\ (unitless) = Dose\ (or\ air\ conc.) * CSF\ (or\ IUR)$$

$$Dose = Site\ specific\ cancer\ dose\ \left(\frac{mg}{kg}\ per\ day\right) or\ concentration\ \left(\frac{\mu g}{m^3}\right)$$

Typically, the dose used is known as Chronic Daily Intake (CDI). It is an intake value (expressed in concentration units) averaged over 70 years. For example: CDI for phenol is 0.1 mg/kg/day; whereas for cyanide, it is 0.0003 mg/kg/day.

$$Cancer\ Slope\ Factor\ (CSF) or\ Inhalation\ Unit\ Risk\ (IUR) = \left(\frac{1}{\frac{mg}{\frac{kg}{day}}}\right)\ or \left(\frac{1}{\frac{\mu g}{m^3}}\right)$$

For example – an estimated cancer risk of $1*10^{-6}$ predicts the probability of one addition cancer over background in a population of 1 million.

Fixing of dosage levels based on experimental methods (Non-carcinogenic effects):

Acceptable Daily Intake (ADI) is a measure of the amount of a specific substance in food or drinking water that can be ingested on a daily basis without appreciable health risk.

$ADI = \frac{NoAEL}{SF}$, where SF is a Safety Factor of 100.

The safety factor is to account for the differences between test animals and humans (factor of 10) and possible differences in sensitivity between humans (another factor of 10).

Reference Dose (RfD) is the preferred toxicity value for evaluating non-carcinogenic effects from exposure to contaminated sites.

$RfD = \frac{NoAEL}{UF*MF}$, where UF is the Uncertainty Factor (usually 100) and MF is the Modifying Factor (also usually 100, based on professional judgment of data base of chemical).

$Noncancer\ hazard\ quotient = \frac{E}{RfD}$, Where E is the Exposure Level.

Calculating Risk

Risk is defined as the probability of suffering severe consequences.

$$Risk = Probability * Severity\ of\ consequence$$

Background risk is the risk posed in the absence of any particular source; whereas incremental risk is specifically caused by the source. Total risk is the sum of background risk and incremental risk.

$$CDI\ \left(\frac{mg}{kg.day}\right) = \frac{\left[C\ \left(\frac{mg}{L}\right) * CR * ED\ (years) * EF\ \left(\frac{day}{years}\right)\right]}{BW\ (kg) * AT\ (days)}$$

Where $CDI = Chronic\ daily\ intake$

$C = Concentration\ of\ dose$

$$CR = Contact\ rate\ \left[\frac{L}{day} for\ water, \frac{g}{day}\ for\ fish, \frac{m^3}{day} for\ air\right]$$

$ED = Exposure\ duration$

$EF = Exposure\ frequency$

$BW = Body\ weight\ (= 70\ kg\ if\ data\ not\ available)$

$AT = Average\ time\ (= 70\ years\ if\ data\ not\ available)$

For non-carcinogenic effect: $AT = ED\ (years) * 365 \frac{days}{year}$

For carcinogenic effect: $AT = Lifetime\ (years) * 365 \frac{days}{year}$

There is some variation in parameters used when mode of contact is different. For example, contact rate for dermal would be: $Dust\ adherence\ \left(\frac{mg}{cm^2}\right) * Area\ (cm^2) * Number\ of\ exposure\ events\ per\ day.$

Whereas the same contact rate for a situation where skin comes into contact with soil would be:
$Dust\ adherence * Soil\ matrix\ (\%) * Skin\ absorbance\ (\%).$

Some other parameters:

Threshold Limit Value (TLV) – It is used by industrial hygienists as the concentration of the chemical in air to which workers may be exposed safely over their occupational lifetime (i.e., for 8 hours a day, 5 days a week over a working life time). TLV has been established for over 100 chemical compounds using epidemiological data. The TLV for carcinogens is 0 – there is no threshold. TLV for mixtures can be calculated as follows:

$$TLV_{mix} = \frac{\sum_{i=1}^{n} C_i}{\sum_{i=1}^{n} \frac{C_i}{TLV_i}}$$

Maximum Contamination Level (MCL) – MCL is a standard set for drinking water quality. The limit is usually expressed in µg/L. It is indicative of how much of the contaminant may be present in the water body with no adverse health effects.

Example 1: Determine the CDI of a non-carcinogenic chemical in water, given that the concentration is 5 mg/L. Compare the CDI for an adult and child (both carcinogenic and non-carcinogenic risks involved). The following parameters have been given to you:

$$ED = 30\ yrs, BW = 70\ kg, EF = 365\frac{days}{yr}, CR = 2\frac{L}{day}\ for\ adult, CR = 1\frac{L}{D}\ for\ child$$

Solution:

Adult, non-carcinogenic:

$$CDI = \frac{\left[5\frac{mg}{L} * 2\frac{L}{d} * 30\ yrs * 365\frac{days}{yr}\right]}{70\ kg * \left(30\ yrs * 365\frac{days}{yr}\right)} = 0.143\frac{mg}{kg.day}$$

Child, non-carcinogenic:

$$CDI = \frac{\left[5\frac{mg}{L} * 1\frac{L}{d} * 30\ yrs * 365\frac{days}{yr}\right]}{16\ kg * \left(30\ yrs * 365\frac{days}{yr}\right)} = 0.313\frac{mg}{kg.day}$$

Adult, carcinogenic:

$$CDI = \frac{\left[5\frac{mg}{L} * 2\frac{L}{d} * 30\ yrs * 365\frac{days}{yr}\right]}{70\ kg * \left(70\ yrs * 365\frac{days}{yr}\right)} = 0.061\frac{mg}{kg.day}$$

Child, carcinogenic:

$$CDI = \frac{\left[5\frac{mg}{L} * 1\frac{L}{d} * 30\ yrs * 365\frac{days}{yr}\right]}{16\ kg * \left(70\ yrs * 365\frac{days}{yr}\right)} = 0.134\frac{mg}{kg.day}$$

Example 2: Soil is contaminated with chloroform (10 mg/g). The following data is given (for a male worker):

$Area\ of\ skin = 18150\ cm^2, 50\%\ of\ skin\ area\ is\ exposed$

$Absorption\ coefficient, ABS = 0.51$

$Soil\ matrix\ coefficient, SM = 0.06$

$2\ exposure\ events\ per\ day$

$Dust\ adherence = 70\frac{g}{m^2}$

Calculate CDI for dermal contact.

Solution:

Assuming he is forty years old and works six days a week. His exposure duration can thus be calculated (313 days in a year).

$$CDI = \frac{\left[10\frac{mg}{g} * \frac{18150 * 0.0001}{2} m^2 * \frac{2}{day} * 313\frac{days}{yr} * 40\ yrs * 0.51 * 0.06 * 70\ \frac{g}{m^2}\right]}{70\ kg * \left(70\ yrs * 365\frac{days}{yr}\right)}$$

$$= 0.27\frac{mg}{kg.day}$$

Example 3: Study in mice showed a LoAEL of 5 mg/kg for a certain contaminant. The quality of data is said to be high. However, there was no data available of NOAEL. What do you think the Reference Dose would be?

Solution:

$RfD = \frac{5}{100*100} = 5 * 10^{-4}\frac{mg}{kg}$, where the factors we divide LoAEL by are the Uncertainty Factor and Modifying Factor respectively.

Example 4: Determine the TLV for a worker exposed to the 42 ppm PCE, 38 ppm TCE, 86 ppm MEK and 35 ppm MIBK in the exhaust air of a solvent recycling operation. What is the TLV for the mixture?

Chemical	TLV (ppm)
PCE	50
TCE	50
MEK	200
MIBK	50

Solution:

$$TLV_{mix} = \frac{\sum_{i=1}^{n} C_i}{\sum_{i=1}^{n} \frac{C_i}{TLV_i}} = \frac{42 + 38 + 86 + 35}{\frac{42}{50} + \frac{38}{50} + \frac{86}{200} + \frac{35}{50}} = 73.6\ ppm$$

$$Actual\ TLV = 42 + 38 + 86 + 35 = 201\ ppm$$

Here, actual exposure is far greater than TLV of the mixture. Proper risk management system must be implemented immediately for the safety of workers.

Hazard identification

It is important to understand the chemicals present, their concentrations and spatial distributions, as well as their movement in the environment. There are numerous steps involved in the process of hazard identification.

Step 1: Initial screening

Chemicals are sorted according to their nature (carcinogenic and non-carcinogenic); and their movement in the environment (air, water, soil).

The data needed for this step includes site history, land use, contaminant levels in various media, exposure pathways, fate and transport of these chemicals, concentration at the receptor and toxicity of the contaminant.

Also, Reference Doses are identified. Toxicity Scores (TS) for contaminants are determined.

For non-carcinogens, $TS = \frac{C_{max}}{RfD}$

For carcinogens, $TS = C_{max} * SF$

Step 2: Exposure assessment

This step comprises of the following procedures: delineation of sources and spatial distribution of contaminants, how contaminant is released and how it migrates, identification of current and future receptor points, estimation of short and long term exposure.

Step 3: Environmental pathway

One example of a complete environmental pathway for a contaminant released into a lagoon is shown here.

Environmental Pathway
Source – Lagoon
Chemical release mechanism – Leaching
Transport mechanism – Groundwater flow
Transfer mechanism – Sorption
Transformation mechanism – Biodegradation
Exposure point – Well
Receptor – Residents
Exposure routes – Ingestion, inhalation, dermal
Potential population – Present and future

Some factors which are considered in addition to those listed are: fraction of time spent outdoors by the population under consideration, secondary exposure through crops grown using contaminated water, and so on.

Step 4: Development of exposure scenarios

There are different scenarios which vary depending on the situation: worker scenario, trespasser scenario, residential scenario, recreational scenario and construction scenario. Intake rates, exposure and its duration vary with each scenario.

Step 5: Dose calculation

The dose referred to here is CDI, and its calculation has been explained above.

Case Studies

Environmental risk assessment case studies are presented below.

I. Environmental and Safety Risk Assessment in Mines

In the case study on Ray Bachra coal mine (Saurabh Jain, 2007), the "probability, consequence and exposure" method was used to determine the applicability of risk assessment in this scenario. The matrix used for rating is presented below:

Table 3: Risk rating criteria

Consequence		**Exposure**		**Probability**	
Several dead	5	Continuous	10	Expected/ Almost certain	10
One dead	1	Frequent	5	Quite possible/ Likely	7
Significant chance of fatality	0.3	Weekly	3	Unusual but possible	3
One permanent disability	0.1	Monthly	2.5	Only remotely possible	2
Small chance of fatality	0.1	Yearly	2	Conceived but unlikely	1
Many lost time injuries	0.01	Once in 5 years	1.5	Practically impossible	0.5
One lost time injury	0.001	Once in 10 years	0.5	Virtually impossible	0.1
Small injury	0.0001	Once in 100 years	0.02		

$$Risk = Consequence * Exposure * Probability$$

$$Maximum\ risk\ rating = 500$$

$Risks \geq 20$ to be reported to management for action

An eight step risk management plan was devised:

1. Identification of all hazards associated with mine
2. Assessment of risk for prioritizing
3. Rank and identify principal hazards which need immediate action/ continuous management
4. Describe root causes for all hazards
5. Fine tune all control mechanisms
6. Document procedure for each process control
7. Assign responsibilities
8. Design auditing procedure

The hazards identified included: geological disturbances, chemical spills, coal stockpiles and spills, spontaneous combustion, residual explosives, limited or no ventilation and lighting for workers, fire from electrical shortages/ gas/ spilled lubricants.

This case study argues that environmental and safety risk assessment is not popular in India. This needs to change, considering the conditions prevalent in coal mines and other manufacturing sectors. Once the risks are quantified, the results can be applied to change existing operation and maintenance practices. Risk management is seen as a necessary tool in identifying hazards and the potential health effects these hazards could have on human health and the environment.

II. Environmental and Safety Risk Assessment Studies of Heavy Metal Contamination in the Industrial Area of Kattedan, India – A Case Study

Kattedan is an industrial area near Hyderabad with battery manufacturing, textile, electroplating and pharmaceutical industries. Twelve different locations when assessed in Kattedan, revealed high concentrations of heavy metals (Zn, Cu, Cr, Ni, Co, Pb, Hg, Cd, As) in soil, groundwater and surface water, and vegetation. A human exposure assessment conducted in the area revealed high amounts of Pb, Zn and Cr in the blood and urine samples of Kattedan residents (K. Chandra Sekhar et al., 2006). Concentration of heavy metals in blood and urine samples of residents shows a direct exposure pathway for the contaminants, which is potentially very harmful to both the environmental and human health.

Environmental hazard assessment was done in the following procedure:

- Sequential extraction of heavy metals from soil samples
- Soil properties analyzed:
    - pH
    - Composition of organic matter in soil
    - Clay minerals
    - Redox potential
    - Concentrations of salts in soil solutions
    - Fe, Mn, Al oxides and hydroxides
- Speciation studies for contaminants present in higher concentrations
- Groundwater and surface water collected from 12 locations and analyzed for heavy metals

Human exposure assessment was carried out in the following manner:

- Urine samples and venous blood samples collected from 120 residents across different age groups
- Samples analyzed for heavy metal content using ICP-MS

The results of the case study can be condensed as below:

Table 4: Concentration of metals of ecotoxicological importance

Sample	As	Cd	Hg
Soil (μg/kg)	100–210	80–160	20–55
Ground Water (μg/L)	55–80	60–100	40–180
Surface Water (μg/L)	40–90	70–120	25–90
Forage Grass (μg/kg)	20–66	40–66	15–40
Blood (μg/L)	8–35	20–40	10–40
Urine (μg/L)	11–38	10–40	8–18

*All the values are in ppb.

The study concludes that from the results obtained, it can be stated that human health risks associated with exposure to heavy metal contamination is a pressing and relevant issue for the residents of Kattedan. Risk assessment consists of four steps: source identification, hazard analysis, exposure assessment, and decision making. As of 2006, 13 of the polluting industries in the region were closed down.

Online Resources

- USEPA: Material on Risk Characterization (Chapter 8)
  - http://www.epa.gov/oswer/riskassessment/ragsa/pdf/rags_ch8.pdf
- ATSDR (Agency for Toxic Substances & Disease Registry): Public Health Assessment Guidance Manual (2005 Update) – Chapters 5-8
  - http://www.atsdr.cdc.gov/hac/PHAManual/ch5.html
- Saurabh Jain. *Environmental and safety risk assessment in mines*. National Institute of Technology, Rourkela. 2007. http://ethesis.nitrkl.ac.in/4260/1/%E2%80%9CEnvironmental_and_safety_risk_assessment_in_mines.pdf (Accessed 08-04-2015).
- K. Chandra Sekhar, N.S. Chary, C.T. Kamala, M. Vairamani, Y. Anjaneyulu, V. Balaram & Jan Erik Sorlie. Environmental and Safety Risk Assessment Studies of Heavy Metal Contamination in the Industrial Area of Kattedan, India – A Case Study. *Human and Ecological Risk Assessment: An International Journal* 12:2, 408-422, DOI: 10.1080/10807030500531513 (2006). http://www.tandfonline.com/doi/pdf/10.1080/10807030500531513 (Accessed 08-04-2015).