Environmental Risk Assessment (ERA)

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- Environmental Risk Assessment is a process for estimating the likelihood or probability of an adverse outcome or event due to pressures or changes in environmental conditions resulting from human activities.

Risk Assessment and management

- Risk assessment is gathering of data that are used to relate response to dose
- Such dose-response data can then be combined with estimates of likely human exposure to produce overall assessments of risk
- Risk management is the process of deciding what to do. It is the decision making, **under extreme uncertainty**, about how to allocate national resources to protect public health and the environment
- Difficult decision making in risk management:
  - Is a one-in-a-million lifetime risk of getting cancer acceptable?
  - how do we go for it?
  - Zero risk achievement would cost infinite amount of money

Perspectives on Risks

Leading Causes of Death in the United States, 1992

Cause	Annual Deaths (thousands)	Percent
Cardiovascular (heart) disease	720	33
Cancer (malignant neoplasms)	521	24
Cerebrovascular diseases (strokes)	144	7
Pulmonary diseases (bronchitis, emphysema, asthma)	91	4
Pneumonia and influenza	76	3
Diabetes mellitus	50	2
Nonmotor vehicle accidents	48	2
Motor vehicle accidents	42	2
HIV/AIDS	34	1.6
Suicides	30	1.4
Homicides	27	1.2
All other causes	394	18
Total annual deaths (rounded)	2,177	100

Source: Kolluru et al., 1996.

Annual Risks of Death Associated with Certain Activities

Activity/Exposure	Annual Risk (Deaths per 100,000 Persons
Motorcycling	2,000
Smoking, all causes	300
Smoking (cancer)	120
Hang gliding	80
Coal mining	63
Farming	36
Motor vehicles	24
Chlorinated drinking water (chloroform)	0.8
4 tbsp peanut butter per day (aflatoxin)	0.8
3 oz charcoal broiled steak per day (PAHs)	0.5

Activities That Increase Mortality Risk by One in a Million

Activity	Type of Risk
Smoking 1.4 cigarettes	Cancer, heart disease
Drinking 1/2 liter of wine	Cirrhosis of the liver
Spending 1 hour in a coal mine	Black lung disease
Living 2 days in New York or Boston	Air pollution
Traveling 300 miles by car	Accident
Flying 1,000 miles by jet	Accident
Flying 6,000 miles by jet	Cancer by cosmic radiation
Traveling 10 miles by bicycle	Accident
Traveling 6 minutes by canoe	Accident
Living 2 summer months in Denver (vs. sea level)	Cancer by cosmic radiation
Living 2 months with a cigarette smoker	Cancer, heart disease
Eating 40 tablespoons of peanut butter	Liver cancer caused by aflatoxin
Eating 100 charcoal-broiled steaks	Cancer from benzopyrene
Living 50 years within 5 miles of a nuclear reactor	Accident releasing radiation

Source: Wilson, 1979.

Cigarette Smoking - In the US, 627 billion cigarettes were made in 1975 which is equivalent to 3,000 per person (including children).

It is estimated that in the US, 15% of all Americans (30% of all smokers) die from lung cancer or heart diseases due to smoking.

Average life time risk = 0.15

Annual risk = 0.15/70 = 0.002 (life expectancy in U.S. = 70 years)

Risk per cigarette = $0.02/3000 = 0.7 \times 10^{-6}$

Increased mortality risk per million = 1.4 cigarettes

Estimated Expenditures per Life-Year Saved for Selected Programs

Program	1990 U.S.$
Childhood immunizations	Direct savings
Eliminating lead in gasoline	Direct savings
Safety rules at underground construction sites	52,000
Hemodialysis at a dialysis center	56,000
Coronary artery bypass surgery	68,000
Front seat air bags in new cars	109,000
Dioxin effluent controls at paper mills	5,570,000

Source: Kolluru et al., 1996, based on data from the Harvard School of Public Health.

Perception of Risk

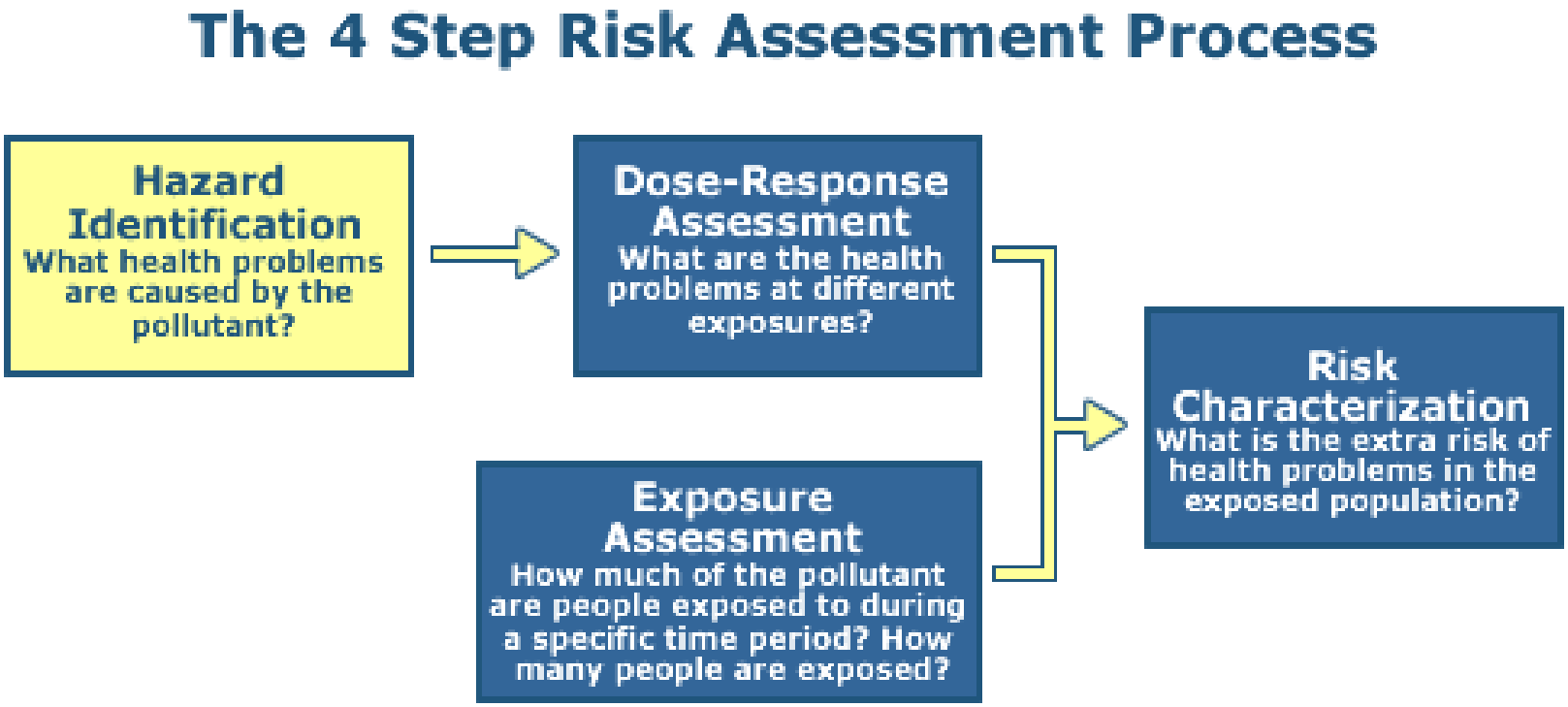
- Perception of risk as seen by an engineer/scientist familiar with the numbers are very different from those of an individual who lives next to a toxic waste site.

Some Characteristics That Change Our Perception of Risk

Attributes That Elevate the Perception	Attributes That Lower the Per
Involuntary	Voluntary
Exotic	Familiar
Uncontrollable	Controllable
Controlled by others	Controlled by self
Dread	Accept
Catastrophic	Chronic
Caused by humans	Natural
Inequitable	Equitable
Permanent effect	Temporary effect
No apparent benefits	Visible benefits
Unknown	Known
Uncertainty	Certainty
Untrusted source	Trusted source

Source: Based on Slovic, 1987, and Slovic et al., 1980.

Risk Assessment



http://www.epa.gov/risk_assessment/hazardous-identification.htm

Step 1 - Hazard Identification

- Hazard Identification is the process of determining whether exposure to a stressor can cause an increase in the incidence of specific adverse health effects (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans.

- In the case of chemical stressors, the process examines the available scientific data for a given chemical (or group of chemicals) and develops a weight of evidence to characterize the link between the negative effects and the chemical agent.

Step 1 - Hazard Identification

Sources of Data

- Epidemiological studies involve a statistical evaluation of human populations to examine whether there is an association between exposure to a stressor and a human health effect. The advantage of these studies is that they involve humans while their weakness results from generally not having accurate exposure information and the difficulty of teasing out the effects of multiple stressors.
- When data from human studies are unavailable, data from animal studies (rats, mice, rabbits, monkeys, dogs, etc) are relied on to draw inference about the potential hazard to humans. Animal studies can be designed, controlled, and conducted to address specific gaps in knowledge, but there are uncertainties associated with extrapolating results from animal subjects to humans.
- Statistically controlled clinical studies on humans provide the best evidence linking a stressor, often a chemical, to a resulting effect. However, such studies are frequently not available since there are significant ethical concerns associated with human testing of environmental hazards.

Toxic Effects

- Acute Toxicity happen very rapidly after a single exposure has occurred (food poisoning, breathing fumes from a chlorine spill). Sweating, nausea, paralysis, and death are examples of acute effects.
- Chronic Toxicity happen only after repeated long-term exposure (cigarette smoking, eating foods with low levels of contaminants, breathing polluted air). Cancer, organ damage, reproductive difficulties, and nervous system impairment are examples of chronic effects.
- These chronic effects fall into two categories: carcinogenic effects and non-carcinogenic effects.
- **Examples of non-carcinogenic chronic effects:**
  - **Organ damage:** cirrhosis of the liver from long-term alcohol consumption; emphysema from long-term tobacco smoking.
  - **Reproductive difficulty:** decreased fertility from the pesticide DBCP (di bromo chloro propane).
  - **Nervous system impairment:** mental retardation in people exposed to high levels of lead during early childhood.

Assessing Toxicity-Acute

- Most information about acute toxicity of chemicals to humans comes from accidental poisonings or exposures, such as drug overdoses or chemical spills. Physicians/researchers know or estimate the level of exposure and observe and document the effects.
- Scientists also use animal tests called LD_{50} (L-D-fifty) studies to assess acute toxicity. These studies determine the amount of a substance that will kill half the test animals in 14 days. This amount is called the LD50--Lethal Dose for 50% of the animals.
- LD50 is stated in milligrams per kilogram (mg/kg): milligram of chemical per kilogram of body weight.
- Lower the LD50-the lower the lethal dose-the more toxic the substance.

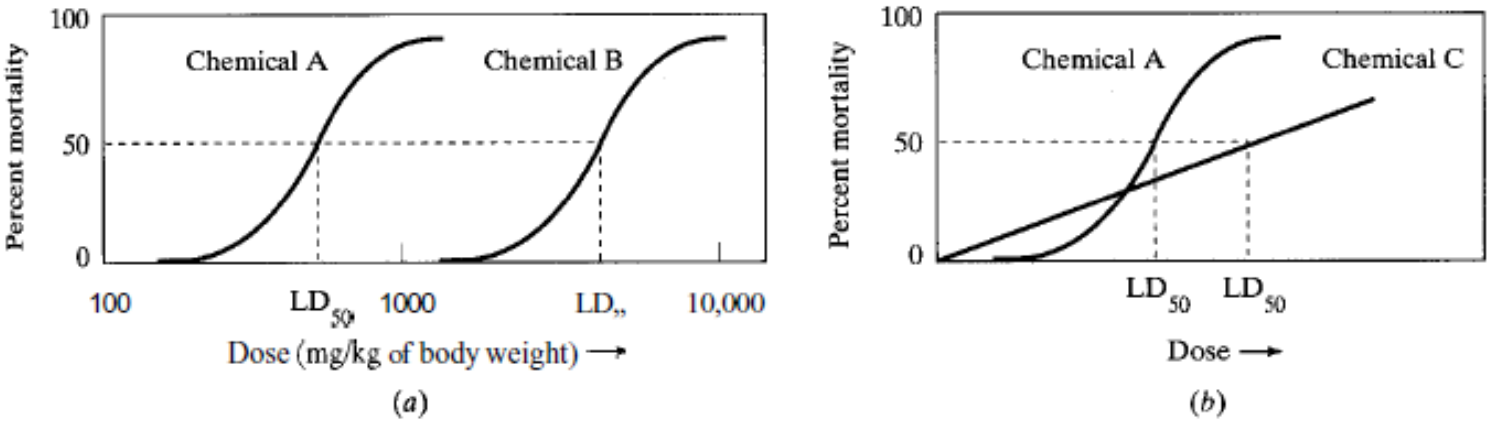


FIGURE 4.4 Dose-response mortality curves for acute toxicity: (a) Chemical **A** is always more toxic than B; (b) but Chemical **A** is less toxic than ***C*** at low doses even though it has a lower LD_{50}.

LD_{50} Comparison

Chemical	LD_{50} (mg/kg)
Ethyl Alcohol	10,000
Sodium Chloride	4,000
Ferrous Sulfate	1,500
Morphine Sulfate	900
Strychnine Sulfate	150
Nicotine	1
Black Widow	0.55
Curare	0.50
Rattle Snake	0.24
Dioxin (TCDD)	0.001
Botulinum toxin	0.0001

A conventional rating system for the acute toxicity of chemicals in humans

	Probable lethal oral dose for humans	
Toxicity rating	Dose (mg/kg of body weight)	For average adult
1. Practically nontoxic	more than 15,000	More than 1 quart
2. Slightly toxic	5,000–15,000	1 pint to 1 quart
3. Moderately toxic	500–5,000	1 ounce to 1 pint
4. Very toxic	50–500	1 teaspoon to 1 ounce
5. Extremely toxic	5–50	7 drops to 1 teaspoon
6. Supertoxic	Less than 5	Less than 7 drops

Assessing Chronic Toxicity

Non-Carcinogenic Assessment

- Scientists assess non-carcinogenic chronic toxicity by administering varying amounts of a substance (dose) to laboratory animals and noting the effects (responses), if any, at each dose.
- Essentially, the scientists look for the smallest dose that causes any detectable effect. This smallest dose is called the ***Lowest Observable Effect Level (LOEL)***.
- **To conduct these dose-response studies, scientists:**

  Administer different small doses of a substance to several groups of test animals every day over a lifetime.

  Periodically examine and finally autopsy the animals to determine if any effects have occurred. The effects may be:

  - damage to an organ,
  - behavioral modifications,
  - change in the level of an essential body chemical.

  Determine the smallest dose at which an effect occurs--**the Lowest Observable Effect Level (LOEL).**

  LOEL is measured in milligrams (mg) of substance per kilogram (kg) of body weight, or in parts per million (ppm) of substance in food.

Assessing Chronic Toxicity

Carcinogenic Assessment

- The prevailing carcinogenesis theory, that human cancers are initiated by gene mutations, has led to the development of short-term, in vitro (in glassware) screening procedures, which are one of the first steps taken to determine whether a chemical is carcinogenic.
- If a chemical can be shown to be mutagenic, then it ***may*** be carcinogenic, and further testing may be called for **Ames mutagencity test**

Ames mutagencity test

- subjects special tester strains of bacteria to the chemical in question.
- These tester strains have previously been rendered incapable of normal bacterial division so, unless they mutate back to a form that is capable of division, they will die. Bacteria that survive and form colonies do so through mutation; therefore, the greater the survival rate of these special bacteria, the more mutagenic is the chemical.
- **Intermediate testing procedures** involve relatively short-term (several months duration) carcinogenesis bioassays in which specific organs in mice and rats are subjected to known mutagens to determine whether tumors develop.

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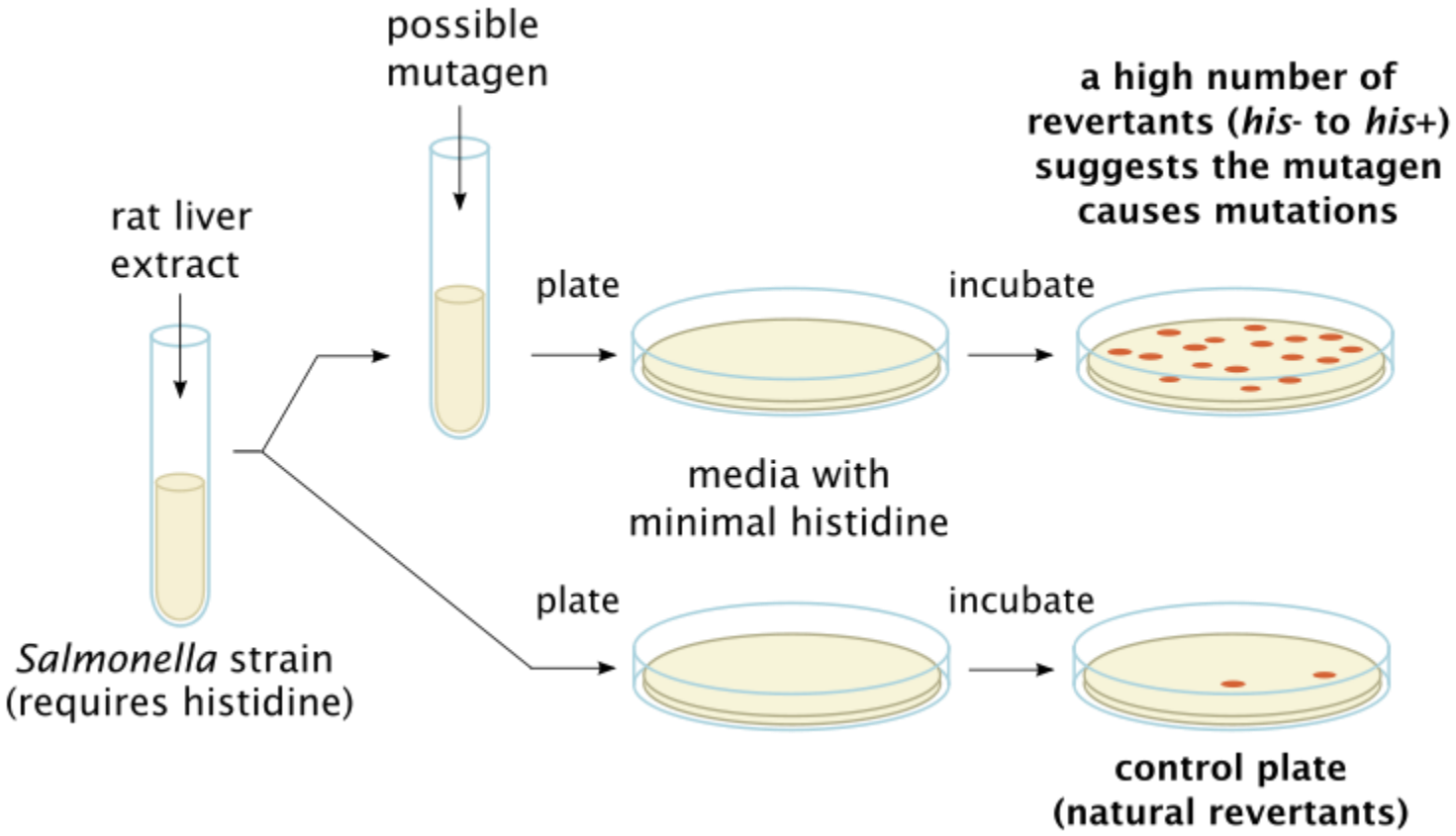

https://en.wikipedia.org/wiki/Ames_test