Chapter 9
Risk Assessment Applications: Exposure, Safety, and Security

Mrugesh H. Trivedi, Gautam V. Priyadarshi, Dipa Lalwani, and Saeid Eslamian

Abstract Risk assessment is a term which encompasses the entire process of hazard/risk identification, risk analysis, risk evaluation, and control. The term 'risk' is commonly believed to be associated with industrial operation; however, categorisation of risk reveals common elements, which can be applied to avoid hazards in other areas. Risk in a wider context is related to the probable harmful effects occurring to human health and/or ecological systems as a result of exposure to environmental stressors. The unsustainable use of natural resources creates stress and causes risks to ecology and human health that lead to social issues in surrounding communities, such as low quality of health and unemployment. Although natural events may be responsible for risk, anthropogenic activities are the major basis of risk to the environment and humans. Environmental risks present themselves as the probability of temporary or permanent changes to the atmosphere, hydrosphere, and lithosphere due to human activities that result in the loss of biodiversity, global warming, and climate change. The risk factor can be understood by calculating relative risk, attributable risk, and odds ratio. The risks occur primarily from exposure to factors such as occupational exposure, environmental exposure, biological exposure, and chemical exposure. The magnitude of the impact of risk on humans or wildlife depends on the path of exposure (inhalation, ingestion, and dermal), amount of exposure (dose), and duration of exposure. The severity of overall risk is dependent upon a range of factors and scenarios. Application of risk

---

M. H. Trivedi (✉) · G. V. Priyadarshi
Department of Earth and Environmental Science, KSKV Kachchh University, Bhuj, Kachchh, India
e-mail: drmrugesh.trivedi@gmail.com; gautamparghi@gmail.com

D. Lalwani
Department of Environmental Science and Technology, Institute of Science and Technology for Advance Studies and Research (ISTAR), Vallabh Vidhyanagar, India
e-mail: lalwani.dipa6@gmail.com

S. Eslamian
Department of Water Engineering, College of Agriculture, Isfahan University of Technology, Isfahan, Iran
e-mail: saeid@iut.ac.ir

J. N. Furze et al. (eds.), *Earth Systems Protection and Sustainability*,
https://doi.org/10.1007/978-3-030-98584-4_9

assessment enables taking care of safety and security in environmental, ecological, and social issues so that the well-being of humans and 'Mother Earth' can be managed. To ensure the safety and security of environmental sustainability, this chapter will focus on environmental contaminants and their risk to human health by projecting five key steps in the process of risk assessment.

Keywords Dose-response · Environmental exposure · Occupational exposure · Risk assessment · Management

9.1 Introduction

Risk is defined as the probability of something going wrong or there may be an unpleasantness that causes injury or loss. Risks revert from specific hazards or threats that occur from any livelihood and affect humans, animals, or the environment (Reynolds and Jennifer 2015). The magnitude of the overall risk is dependent upon various factors such as the potentiality of contaminants to cause damage and the mode and duration of exposure. The overall purpose of risk assessment is to characterise the nature and magnitude of health risks to humans (residents, workers, recreational visitors) and ecological receptors (birds, fish, and wildlife). Thus, risk assessment acts as a safeguarding action from physical, chemical, biological, or other stressors that may be present or anthropogenically introduced to the environment. Risk assessment (RA) is the systematic approach of confronting and expressing uncertainty in predicting the future. RA has been developed as an examination of workplace safety, to determine if adequate safeguards have been taken or more should be done to avert possible harm. To get the environmental clearance for any project, there is a provision to conduct an environmental impact assessment (EIA). Both RA and EIA have similar goals and provide complementary information. The objective is not limited to estimating quantitative risk and the impact on the environment. However, the rationale behind policy-making for conducting EIA and RA is intended to provide reason-based predictions of possible consequences of planned decisions to provide other comfortable alternatives and management plans (Andrews 1990). The United States Environmental Protection Agency (USEPA) divides risk assessment into two categories: (i) human health risk assessment and (ii) ecological risk assessment (USEPA 2019a). As per the USEPA definition, 'the environmental risk is the probability of harmful effects to human health and/or ecological systems due to exposure to that environment'. It is also known as 'ecological risk assessment' (ERA). In the twenty-first century, anthropogenic activities have contaminated all spheres of Mother Nature outside the industrial/research laboratories and commercial areas. Therefore, RA applications should go beyond the boundaries of industry and should cover all three components of sustainability, i.e., social, economic, and ecology. A common man may have some risk from the environmental contamination of the water he drinks, the food he consumes, or the air he used to breathe. The risk that affects humans, animals, or the environment after an accidental exposure or intentional release of a biological agent is known as biological risk. A person, who works directly with biological materials, faces numerous bio-risks, which also exist for a person who works indirectly with or near infectious agents (Reynolds and Jennifer 2015). The recent example of the coronavirus pandemic (christened by the World Health Organisation as "COVID-19") has further led to a risk of an outburst of pneumonia. It was first detected in Wuhan, Hubei Province, China. The virus has infected almost more than 248,098 out of which more than 10,000 death reported from 182 countries as of 20th March 2020 and continues to expand. The pandemic condition created by COVID-19 has forced us to rethink biosafety and security measures at all possible places to avoid or to reduce the bio-risk. To ensure the safety and security of humans and ecology for environmental sustainability, this chapter will further discuss the risk assessment process with several examples of environmental contaminants and risk assessment applications.

9.2 Risk Assessment Process

Risk assessment is a four-step process that utilises empirical information to assess health effects caused by exposure: The four steps are (1st) hazard identification, (2nd) exposure assessment, (3rd) dose-response assessment, and (4th) risk characterisation. While Carpenter (1995) has shown his consent for the four steps process, he further emphasised that attempts to quantify the risk to human health, economic welfare, and ecosystem from human activities and natural phenomena are incomplete without preparing to mitigate or eliminate unacceptable risks asserting the fifth step of risk management. These steps are illustrated in Fig. 9.1.

The five steps of risk assessment are detailed in subsections of Sect. 9.2.

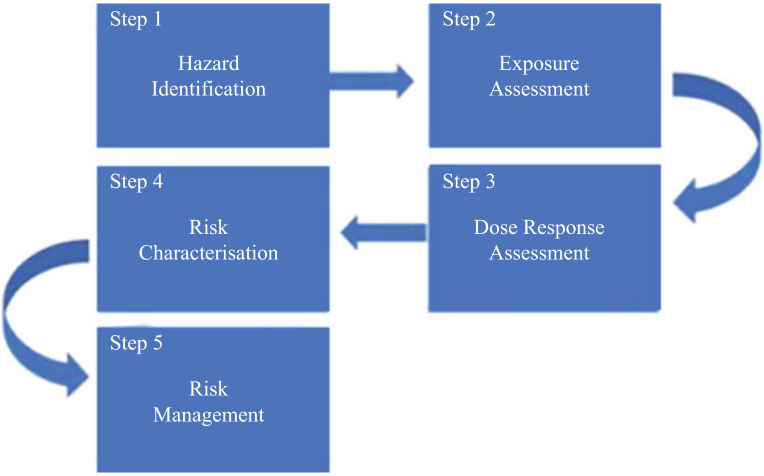

Fig. 9.1 The five steps of the risk assessment process (USEPA 2019b, c; Lohani et al. 1997)

9.2.1 Hazards Identification (Step One)

Hazard identification determines whether environmental exposure of pollutants or contaminants can lead to an increase in the occurrence of certain negative health effects. It lists the possible sources of harm, usually identified by experience elsewhere with certain technologies, materials, or conditions. Exposure to environmental pollutants may cause different adverse effects on human populations such as carcinogenesis, neurotoxicity, cytogenetic damage, and endocrine disruption in addition to developmental, reproductive, and immunological disorders (Tchounwou et al. 2012; Coster and Larebeke 2012). Recent studies reveal that widely used plastic products have contaminated the world with a number of different environmental endocrine-disrupting chemicals (EDCs). EDCs may also lead to deleterious impacts on human blood through the generation of reactive oxygen species (ROS) (Trivedi et al. 2020), in addition to toxicological dysregulation in embryonic reproductive tissue differentiation and development (Rich et al. 2016), which may further lead to gender dysphoria in humans. The EDCs exposure to aquatic animals may cause vitellogenin induction in fish (Thorpe et al. 2001) that may lead to a negative effect on fish breeding and consequently on fish catch. Natural contamination such as the high level of fluoride in drinking water produces neurotoxicity in school children of 12–14 years of age, in addition to known impacts of dental and skeletal fluorosis (Trivedi et al. 2007; Ding et al. 2011; Seraj et al. 2012; Choi et al. 2012; Trivedi et al. 2012; Razdan et al. 2017; Duan et al. 2018). The USEPA (2019b and Damania et al. 2019) have issued threshold guidelines and frequently update the lists of highly toxic chemicals based on new research outcomes. These thresholds indicate the minimum concentration of chemicals involved in causing harm if present at any location in a particular form. In addition to guidelines, environmental indicators provide information on pollution. For example, intensive anthropogenic activities result in the disappearance of foraminifera from the ocean (Bhatt and Trivedi 2018), which is one of the most significant natural indicators of anthropogenically disturbed coast.

Hazard identification is a systematic approach to identify risks and determine their scope, impact, and vulnerability effect in our surroundings. Hazard identification can be more thoroughly understood by the relationship between exposure to any hazardous compounds and the risk of causing adverse consequences (Masters and Ela 2008). That can be identified by using a simple 2 × 2 matrix as given in Table 9.1, where rows divide people by exposure and columns divide them by disease.

Table 9.1 A 2 × 2 matrix for epidemiologic rate comparison

	With disease	Without disease
Exposure	a	b
Non-exposure	c	d

The rows split the populations among those who have been exposed to the risk factor and those who have not. The columns are based on the numbers of people with the disease and without the disease. Relative risk is thus defined as

$$\text{Relative risk} = \frac{a(a+b)}{c(c+d)} \tag{9.1}$$

Here we should note that the numerator is the fraction of those exposed who have the disease and the denominator is the fraction of those not exposed but have the disease. If those two ratios are the same, the probabilities of having the disease would not depend on contact with risk factor. Above 1.0, the higher relative risk shows an association between exposure and risk. The attributable risk is defined as

$$\text{Attributable risk} = \frac{a}{a+b} - \frac{c}{c+d} \tag{9.2}$$

In Table 9.1, the difference between the odds of having the disease with and without exposure is known as attributable risk. Attributable risk with the value of 0.0 shows no relationship between exposure and risk.

The cross product of the entries in the matrix is known as odds ratio.

$$\text{Odds ratio} = \frac{ad}{bc} \tag{9.3}$$

Numbers greater than 1.0 indicate that there is a link between exposure and risk.

An example of hazard identification using the above formula would be an examination of personnel data for employees of a vinyl chloride manufacturing firm revealing that 15 of 200 workers had liver cancer. A control group of persons with smoking histories similar to the exposed employees and who were unlikely to have come into contact with vinyl chloride had 24 people acquire liver cancer and 450 people did not develop liver cancer. To reveal the relationship between exposure and risk, the data may be summarised as shown in Table 9.2.

Solving for each measure yields

$$\text{Relative risk} = \frac{15/(15+185)}{24/(24+450)} = \frac{0.075}{0.05} = 1.5.$$

$$\text{Attributable risk} = \frac{15}{200} - \frac{24}{474} = 0.024$$

Table 9.2 A 2 × 2 matrix for epidemiologic rate comparison

	Liver cancer	Non-cancer worker
Exposure	15	185
Non-exposure (control)	24	450

$$\text{Odds ratio} = \frac{15 \times 450}{185 \times 24} = 1.52$$

In the example, the relative risk and the odds ratio both are above 1.0, so they show that there is a relationship between exposure and risk. For those who were exposed, the risk of cancer has increased by 0.024 (the attributable risk) over that of their cohorts who were not exposed (Masters and Ela 2008). Based on all three measures, we can identify the risk, and further study of risk assessment can give guidance to reduce and manage the risk.

9.2.2 Exposure Assessment (Step Two)

The process of assessing or estimating the size, frequency, and duration of exposure to an environmental agent or stressor is known as an exposure assessment. Contaminant sources, release methods, distribution, and transformation characteristics are all essential considerations in determining exposure. Exposure assessment also includes the determination of any exposure of contaminants through inhalation, ingestion, and dermal in the group of population. There are various ways to identify exposure such as diagnosis of disease including analysis of blood samples, urine samples, saliva, tissue samples, or a swab from nasal or buccal cavity. There are two types of exposure, viz., (i) occupational exposure and (ii) environmental exposure.

9.2.2.1 Occupational Exposure

Occupational exposure can be explained as the exposure to any stressor, either chemical or biological, at a workplace, which might harm the employee/workers. For example, workers in mining or road construction have the risk of accident or mine collapse. Risk identification and analysis for occupational exposure include identifying unfavourable occurrences that result in a hazard, analysing the hazard process of the unfavourable event, and estimating the extent, size, and frequency of detrimental consequences (Paithankar 2011). Due to high-risk procedures, hazardous sectors witness substantial deaths connected to workers and workplaces (Gul and Ak 2018). Hazardous industries are facing serious fatalities related to work, workplaces, and workers because of their high-risk processes (Gul and Ak 2018).

Recent studies revealed that chronic exposure to petrochemical and chemical industry pollutants such as suspended particles, polycyclic aromatic hydrocarbons (PAH), volatile organic compounds (VOCs), SO_x, ozone (O_3), and NO_x are linked with detrimental health effects including reduced lung capacity, increased respiratory illnesses, and increase in rate of mortality. These are particularly found in industrial workers (WHO 2006). Additionally, handling and working on the premises of the above mentioned chemical causes allergies and sever respiratory effects such as asthma and chronic obstructive pulmonary disease (COPD) (Ramirez et al. 2012).

Apart from the chemical industries, some workers with biological exposure to aquaculture or food production processes may also have a risk of occupational diseases and injuries. Ngajilo and Jeebhay (2019) revealed occupational exposure characterisation for noise and chemicals such as formaldehyde and microorganisms such as *E. coli* bacteria in fish ponds. The occupational diseases related to the aquaculture work included musculoskeletal disorders (MSD), respiratory symptoms and asthma, skin infections, dermatitis and urticarial, and some occupational infections such as leptospirosis. The farmers associated with agricultural activities are also susceptible to the risk of various chemical-based fertilisers and pesticide exposure.

Recently, global studies have shown negative health effects of commonly used agrochemicals in farming. Various diseases ranging from respiratory effects to cancer have been identified among farmers due to exposure to such agrochemicals (Dhananjayan and Ravichandran 2018). Pesticide exposure at work is related to an increased risk of obstructive lung diseases including chronic bronchitis and COPD (Pourhassan et al. 2019). The agrochemical-based industrial occupation has the same types of risk and occupational disease. Therefore, several countries have legislation to conduct risk assessment studies as a part of their EIA for environmental clearance, before starting any industry.

Risks can be identified, and the severity can be measured based on risk assessment analysis. Precautions can be taken to minimise the risk by using risk assessment methods. In most cases, the occupational health-related risk is well managed and given more concern for environmental clearance. The compensation mechanism has encouraged employers to improve the safety of workers at the workplace. The compensation insurance system may reduce occupational accidents and rises the risk-based employer’s payment (Shin et al. 2011). In the present era, there are various legislation and guidelines, throughout the world, including the factory act, the mine act, the fire act, the insecticide act, OECD guidelines, and the environmental protection act that provide occupational safety in addition to the well-being of the worker at their workplace.

9.2.2.2 Environmental Exposure

Approximately 25% of global deaths occur due to exposure to environmental pollutants, including those in household and ambient air pollution, ultraviolet radiation, and various chemicals (Pruss-Ustun et al. 2016). According to the United Nations Development Programme (UNDP), every 2 s someone aged between 30 and 70 dies prematurely from non-communicable diseases including cardiovascular and chronic respiratory diseases, diabetes, or cancer. The causes of diseases are often unknown environmental pollution. Every year, seven million people worldwide die as a result of exposure to fine particles in contaminated air (UNDP 2019a). The UNDP has set 17 sustainable development goals in which the 3rd goal focuses on

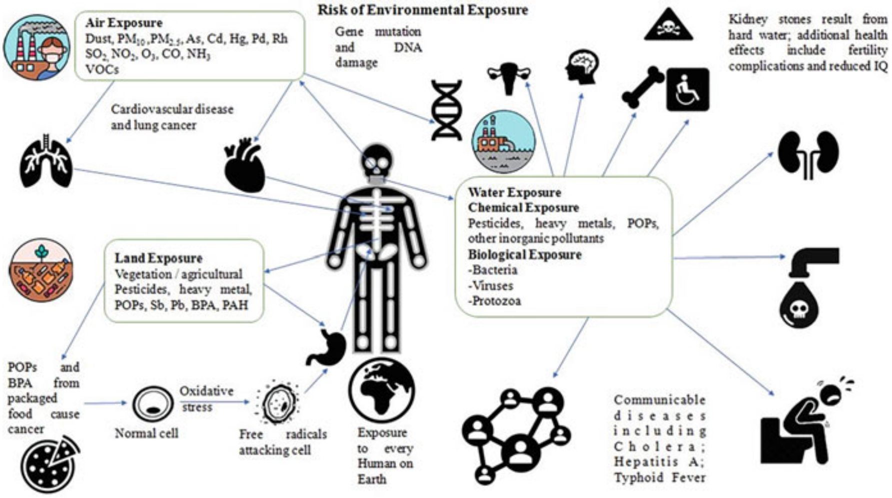

Fig. 9.2 Risk of environmental exposure from the air, water, and land with its health effects

"Good Health and Well-Being." To achieve this, it is essential to have a comprehensive understanding of risk assessment to environmental contaminant exposure. The majority of hazardous chemicals and pollutants and their adverse health effects are summarised in Fig. 9.2.

Due to modern lifestyle and industrialisation, there is a continuous emission of toxic pollutants from various anthropogenic sources; these pollutants have become part of our routine life. Sadly, there is a scarcity of awareness among people towards health risks associated with exposure to environmental pollutants. To understand the same, first, there is a need to understand the intake of pollutants using the universal formula as follows:

$$\text{Intake}\,(I) = \frac{C \times \text{CR} \times \text{EFD}}{(\text{BW} \times \text{AT})} \tag{9.4}$$

where

I = Intake – the amount of the chemical taken [mg (kg body weight)$^{-1}$ × day^{-1}]

C = average concentration of the pollutant over the exposure period, [mg/dm^3 water], [mg/kg soil], [mg/m^3 air]

CR = Contact rate, the quantity of contaminated medium contacted per unit time, [mg soil/day], [m^3 water /day], [m^3 air/day]

EFD = Exposure frequency and duration. It describes how often and how frequently exposure occurs [hours], [days], [years]

BW = Average body weight [kg]

AT = Averaging time, the period of exposure [days]

Environmental exposure may occur in diverse ways, such as inhalation of polluted air, ingestion of contaminated water and food, and exposure to biological pathogens due to unhygienic routine habits. Common environmental exposures to humans are discussed hereafter in brief.

The environmental exposure of pollutants or contaminants occurs through three main pathways: inhalation, ingestion, and dermal exposure (De Miguel et al. 2007). The amount of exposure to the pollutants via their routes can be calculated using different equations, where accurate data of the concentration of the pollutant in the particular matrix is the most important, as the error in the concentration of the pollutants may lead to false-positive or false-negative exposure results. Dose concentration may be calculated by using the following equations:

Inhalation of resuspended particles or toxic gases through mouth and nose from the environment:

$$D_{\text{inhalation}} = \frac{C \times \text{Inh}\,\text{R} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times \text{PEF}} \tag{9.5}$$

Direct ingestion of environmental contaminants from the consumption of food and water:

$$D_{\text{ingestion}} = \frac{(C \times \text{Ing}\,\text{R} \times \text{EF} \times \text{ED})}{(\text{BW} \times \text{AT})} \times 10^{-6} \tag{9.6}$$

Dermal absorption of contaminants adhered to exposed skin:

$$D_{\text{dermal}} = \frac{(C \times \text{SA} \times \text{SL} \times \text{ABS} \times \text{EF} \times \text{ED})}{(\text{BW} \times \text{AT})} \times 10^{-6} \tag{9.7}$$

Where:

D = exposed dose in (mg kg^{-1} day^{-1}) through inhalation ($D_{\text{inhalation}}$), ingestion ($D_{\text{ingestion}}$), and dermal (D_{dermal}) contact
C = Concentration of contaminants in mg kg^{-1}
Inh R = Inhalation rate (USEPA 2011)
Ing R = Ingestion rate
EF = Exposure frequency (site-specific)
ED = Exposure duration (site-specific)
BW = Average body weight
AT = Average exposure time
PEF = Particle Emission Factor (USEPA 2014)
SA = Exposed skin area (USEPA 2008)
SL = Skin adherence factor
ABS = Dermal absorption factor

A few examples of environmental pollutant exposures are given in the remainder of this section.

9.2.2.3 Risk Related to Exposure to Polluted Air

As per USEPA (2019d), there are six most common pollutants in the atmosphere, also known as ‘criteria pollutants’. These are also considered as a standard of ambient air quality for most of the countries, including airborne particulate matter (PM), carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), and lead (Pb). Evidence from epidemiological studies has demonstrated that both outdoor and indoor air pollution are well-known major problems of public health (Bruce et al. 2000). A recent human study revealed that short-term exposure to traffic-related air pollution (TRAP) alters some extracellular micro-RNAs. TRAP is a dynamic combination of pollutants such as PM_{10}, $PM_{2.5}$, NO, NO_2, CO, CO_2, black carbon, and ultrafine particulate matter. TRAP increased the risk of a variety of illnesses including various forms of cancer, lung, cardiovascular, and neurological disorders, and, more recently, diabetes mellitus (Krauskopf et al. 2019).

9.2.2.4 Particulate Matter (PM) and Gaseous Phase

Particulate matter is classified as PM_{10}, further known as suspended particulate matter (SPM), and $PM_{2.5}$ and respirable particulate matter (RSPM). $PM_{2.5}$ and $PM_{<1.0}$ are of major risk due to their ultrafine size, which can penetrate respiratory systems deeply. Such ultrafine particles are associated with increased mortality and morbidity due to various side effects on humans including cardiovascular disease, respiratory syndrome, COPD, decreased lung function, and premature mortality (Pope et al. 2004; Samoli et al. 2008; Halonen et al. 2009; Guaita et al. 2011; Perez et al. 2012). Liu et al. (2019b) have found that maternal exposure to $PM_{2.5}$ may increase susceptibility to foetal distress. Kim et al. (2017) have found a positive correlation between asthma hospitalisation and the concentration of PM in residential areas. In addition to the fine size, persistent organic chemicals (POPs) attached to the $PM_{<1.0}$ and $PM_{2.5}$ directly penetrate human lungs via inhalation and are also a matter of concern. Recent studies on emerging pollutants give evidence of chemicals attached to particulate matter, being present in the gaseous phase of the atmosphere risking human health (Ge et al. 2017; Guo et al. 2018; Müller et al. 2012; Ruan et al. 2019).

9.2.2.5 Carbon Monoxide (CO)

Carbon monoxide (CO) is a colourless, odourless, tasteless, and fatal gas that is released from incomplete combustion. It is highly toxic when inhaled. The cooking and heating appliances are the sources of CO in our houses due to poorly maintained appliances, clogged flues, vents, or chimneys. Especially in rural areas, wood burners and other solid fuel heating systems are being used that potentially increase the risk of CO poisoning (HES 2019). Cushen et al. (2019) have reported a local area of a European country (Wales) has experienced an incident of carbon monoxide exposure due to faulty installation of at least 541 wood burners. In developed and developing countries, open burning of municipal waste is practised leading to the emission of CO gas.

9.2.2.6 Nitrogen Oxides (NO_x)

Oxides of nitrogen, a group of nitrogen oxides (NO_x), are highly reactive gases. In the anthropogenic form, they are largely emitted from vehicular emission. NO_x is emitted from natural sources such as lightning and the natural combustion of biomass. The concentration of NO_x is directly related to acid rain, photochemical smog, and ozone in the atmosphere (Liu et al. 2019b). If a high amount of NO_x is inhaled by the human body, it causes damage to lung function and disturbs respiratory systems. Many researchers study the impact of NO_x on human health risks in metro cities where vehicular traffic is highest (Neuberger et al. 2002; Samoli et al. 2007; Boningari and Smirniotis 2016).

9.2.2.7 Sulphur Oxides (SO_x)

Oxides of sulphur, a group of sulphur oxides (SO_x), are also highly reactive gases. The majority of SO_x comes from anthropogenic sources, namely, urban transportation and industrial activity, by the burning of coal (Ielpo et al. 2019). In an Italian epidemiological study related to SO_x exposure, it was revealed that people who reside beside an industrial area have a high risk of adverse health consequences and were suffering from critical situations in terms of short-term health effects and excess mortality (Gianicolo et al. 2016). A researcher from China has found a correlation between exposure to SO_2 and adverse short-term effects on total health with specific cardiovascular disease (CVD) and related hospital admission (Amsalu et al. 2019). Several studies reveal that a high risk of acid rainfall is related to the high atmospheric concentration of SO_x which can deteriorate historically/archaeologically significant monuments such as the Taj Mahal in India.

9.2.2.8 Ozone (O_3)

Ozone is a bluish explosive gas found in the Earth's atmosphere and is considered a major pollutant when present in the troposphere. Ozone is one of the secondary pollutant generated in the atmosphere due to the reaction between two primary pollutants. It is an irritating and toxic pollutant, even at low concentrations. Various VOCs including benzene, ethylbenzene, toluene, and xylene are emitted from vehicular emission and combustion of fossil fuels which is popularly known as BETX. These aromatic compounds play a vital role in photochemical reactions forming peroxyl radicals (RO_2), by reacting with the hydroxyl radicals (OH°) from ozone molecules (Ceron-Breton et al. 2015). Peroxyl radicals (RO_2) also react with nitric oxide (NO) to form nitrogen dioxide (NO_2). In the presence of sunlight, NO_2 molecules react with various VOCs to generate toxic stratospheric ozone molecules (Geng et al. 2008; Garg and Gupta 2019). Hackney et al. (1975) have experimented and found that a low level of O_3 exposure leads to the development of decreases in pulmonary function.

9.2.2.9 Lead (Pb)

Lead is a well-known highly toxic heavy metal found in nature as well as anthropogenic sources. It can be released directly into the air in the form of particulate matter from vehicular and industrial emissions. Exposure to atmospheric lead occurs mainly through inhalation of dust and aerosol particles containing lead and vapours of paints (Tchounwou et al. 2012). Mrugesh et al. (2011) found the cytotoxic effects of Pb at low concentration on human red blood corpuscle (RBC) under in vitro conditions. The United States has tackled the problem of lead poisoning in children by attempting to eliminate sources of exposure, including gasoline, solder in water pipes, cans, and industrial emissions. Hence, a dramatic reduction was found in the number of children with elevated blood lead levels in the last 20 years (Meyer et al. 2003). Long-term exposure of lead from paint and gasoline is also a positive correlation in violent crime along with the murder rates, which is consistent with findings indicating that children with the higher bone lead often seem to have more impulsive and aggressive behaviour (Nevin 1999). From the 1st January 1996, the US Clean Air Act banned the sale of leaded fuel for use in on-road vehicles. Boskabody et al. (2018) have reviewed and concluded that exposure to lead may increase the likelihood of having a detrimental health effect on respiratory, neurologic, digestive, cardiovascular, and urinary disorders and also explained the mechanism of these effects.

9.2.2.10 Risk Related to Exposure to Polluted Water

Every living creature on the planet requires water to sustain. Only 1% of the water on earth is available for drinking and other daily activities including industrial utility, washing, bathing, and cooking. Following industrialisation, the quality of various water bodies has been deteriorating due to the release of unwanted/foreign chemicals into water bodies. According to UNDP (2019b), 80% of the industrial/municipality wastewater goes into waterways untreatably. In accordance, to get good quality water and for the conservation of natural water bodies, UNDP has set a sustainable development goal (no. 6) as ‘clean water and sanitation’. Polluted water contains toxic and hazardous chemicals, radioactive material, and microorganisms which may harm human health.

9.2.2.11 Microbial Exposure Through Contaminated Water

Microbes grow fast in drinking water; hence, water is a huge source of microbial contamination, including bacteria, viruses, and protozoa. Drinking water is mainly contaminated through poor sanitation and discharge of untreated municipal wastewater. More than 500 waterborne pathogens are found in drinking waters, identified by the USEPA through its candidate contaminant 'CCL 3 Universe' list (USEPA 2019b). Nine out of ten children are dying due to infectious diarrhoea. In most cases, pathogens such as *Rotavirus*, enterotoxigenic *Escherichia coli*, *Campylobacter Jejuni*, *Vibrio cholerae O1*, *Shigella* spp. and possibly *Aeromonas* spp., enteropathogenic *E. coli*, *Aeromonas* spp., *V. cholerae* O139, enterotoxigenic *Bacteroides fragilis*, *Clostridium difficile*, and *Cryptosporidium parvum* are the main culprits of waterborne disease (Ashbolt 2004).

9.2.2.12 Chemical Exposure Through Contaminated Water

Various foreign chemicals flow into the water because of man-made activities and act as contaminants of water. On consumption of such water, the risk of adverse health impact is increased. Water pollutants can be further classified into two types: organic pollutants and inorganic pollutants, viz., VOCs, POPs, pesticides, and heavy metals. Understanding risk in relation to the consumption of contaminated water is discussed based on the origin of the pollutants and its source as per World Health Organisation guidelines (WHO 2011).

9.2.2.13 Industrial Source and Human Dwelling

Mining (extractive industries), manufacturing units, processing industries, sewage (including several contaminants of emerging concern), municipal wastes, fuel leakages, and urban runoff may cause water contamination by changing the physiochemical properties of water. Bungling central effluent treatment plants (CETP) or municipality sewage plants results in a high rate of surface and groundwater contamination. On consumption of contaminated water for extended periods, it either mimics or alters hormonal activities in humans and causes adverse consequences.

9.2.2.14 Agricultural Activities

Excessive use of fertilisers and all classes of pesticides such as cholinesterase inhibitors, organophosphorus compounds, and carbamates is introduced into surface or groundwater through water runoff during monsoon or irrigation practices.

Residues of pesticides on food enter into food webs. The use of vegetables and fruits without proper cleaning may pose the risk of adverse effects on human health.

9.2.2.15 Water Treatment or Material in Drinking Water

Coagulants in piping materials are high in Fe, Mn, Zn, Pd, Cu, and other biological contaminants. The Flint (MI, USA) 2016 water crisis was the major lesson that taught the world the seriousness of increasing health risks related to water contaminants like lead. The contaminants forced governor Rick Snyder to declare an emergency condition in Flint on January 5, 2016. The study conducted by Laidlaw et al. (2016) revealed a meaningful conclusion, i.e., 'In Flint, Michigan, USA, a public health crisis occurred in April 2014 when the water supply was switched from Lake Huron to a more corrosive source from the Flint River, causing lead to leach from water pipes'. On one occasion, former President Barack Obama tweeted, *The mentality is just as corrosive to democracy as that substance that puts lead in your water.* He stated: *It is not enough to just repair the water. We must change the culture of neglect.* He also blamed the Flint water issue on a belief that *less government is the ultimate good*, which he claimed has resulted in disinvestment in low-income communities. Such incidences may increase the risk of exposure of toxic metals to the community.

9.2.2.16 Pesticides Used for Public Health (Other Than Agriculture)

Larvicides such as chlorinated organics, DDT, HCCH, and chlorinated cycloidians are being used to control vectors of diseases that will pose a risk to health and safety of human being. Due to continuous application, microbial resistance develops tolerance against low doses. Such consequences force the application of high doses that lead to public health being increasingly vulnerable.

9.2.2.17 Naturally Occurring Risks

Geogenic sources such as rocks, soil, and sand and eutrophic water bodies (also influenced by sewage inputs and agricultural runoff) including As, Pb, Cu, Fe, Cl^-, F^-, and NO_3^- may contaminate the groundwater and increase consumption related risk.

9.2.2.18 Risk Related to Exposure to Polluted Land

The land is a solid surface, which is not permanently covered by water. The land is a medium, which provides strength and nutrients to grow plants. For thousands of years, humans practised farming to satisfy food requirements. The ever-increasing

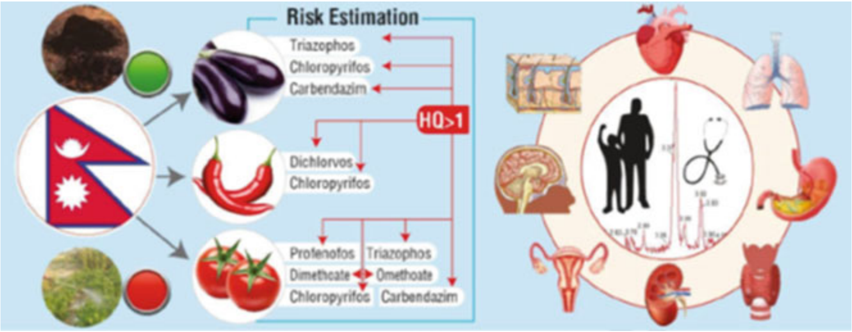

Fig. 9.3 Graphical presentation for risk estimation of pesticides from vegetables (Bhandari et al. 2019)

population generates more pressure on land to increase agriculture yield, which ultimately leads to the increased utilisation of chemical fertilisers and pesticides (Foley 2011). The excessive use of chemical pesticides in developed and developing countries may be hazardous to individuals and the environment. Application of pesticides such as alachlor, atrazine, simazine, and paraquat results in insecticide-resistant in bugs (WRI 1998). Figure 9.3 shows the recent pesticide residue-related risk to human health calculated by Bhandari et al. (2019).

Other than agriculture, mining activities and industrial effluents also cause land degradation with concurrent risk to human health. Most naturally occurring contaminants come from geogenic sources such as weathering and erosion of rocks or mountains, forest fire, and volcanic eruption. The amount of contaminants is lesser than anthropogenic sources. As natural contaminant sources were present from the beginning of life, many life forms may grow by adaptation to the nearby environment, whereas anthropogenic pollutants are added in large quantities over much shorter time scales due to increasing population and industrialisation to compensate for the demands of living beings. Industrialisation activities lead to the extreme use and release of chemicals in the environment which ultimately poses risk to all life forms. Pesticide residue from food, soft drinks, and drinking water poses more risk to human health. The risk from exposure to POPs should be given more attention due to their unique chemical properties, bio-accumulative nature, and relevant toxicity.

9.2.3 Risk Characterisation (Step 3)

Risk characterisation is an important constituent of the risk assessment procedure for both ecological and human health. The purpose of risk characterisation is to interpret the risk calculations based on existing data coming from observations and clarify their meaning for the health of populations. Risk characterisation is an essential element of the risk assessment process that supports decision targeting at the risk abatement arrangements. Potential health risks can be characterised using hazard quotient (HQ) and hazard index (HI). Data related to the rate of ingestion, rate of inhalation, dermal exposure, exposure frequency, exposure duration, exposed skin area, and skin adherence factor for various age groups are available on USEPA's reports and guidelines (USEPA 2008, 2011). Based on the dose of any contaminants and dose-response relationship, the health effect due to exposure of a particular contaminant can be calculated. The risk of any contaminants can be calculated using a hazard quotient (HQ) and hazard index (HI) following Chabukdhara and Nema (2013):

$$\mathrm{HQ} = \frac{\mathrm{Dose}}{\mathrm{RfD}} \tag{9.8}$$

where

HQ = Hazard quotient
Dose = The concentration of contaminants exposed through inhalation, ingestion, or dermal
RfD = Reference dose (of the daily exposure to human)

There are two types of exposure: (i) acute/short-term and (ii) chronic/long-term exposure

For acute/short-term HQ assessment known as aHQ, the calculation is as follows:

$$\mathrm{aHQ} = \frac{\mathrm{ESTI}}{\mathrm{ARfD}} \times 100\% \tag{9.9}$$

where

ESTI = Estimated short-term intake can be express as the highest level of contaminant × Dose
ARfD = Acute references dose, which can avail from WHO and USEPA database

For chronic/long-term HQ assessment known as cHQ, the calculation is as follows:

$$\mathrm{cHQ} = \frac{\mathrm{EDI}}{\mathrm{ADI}} \times 100\% \tag{9.10}$$

where

EDI = Estimated daily intake that can be expressed as

$$\left(\frac{\text{Mean level of contaminants} \times \text{dose}}{\text{Bodyweight}}\right) \tag{9.11}$$

ADI = Acceptable daily intake (which can be taken from the WHO and USEPA database)

The HQ is used to estimate the potential risk due to an individual contaminant, while HI is a cumulative risk that takes into account multiple contaminants (Posthuma and Suter 2011). The HI is the total sum of the HQs. The HI is based on the cumulative effect of any contaminant with similar mechanisms of adverse health effects. The long-term exposure of contaminants causes the same physiological effects in terms of size and nature in relation to the time of exposure. Therefore, the HI was calculated by adding up the cHQi of contamination of a group and expressed as a hazard index (HI) as follows:

$$\mathrm{HI} = \sum\nolimits_{i=1}^{n} \mathrm{cHQi} \tag{9.12}$$

An HQ or HI > 1 denotes potential risk to human health, while an HQ or HI ≤ 1 indicates no risk.

The above equations can be derived from authentic published literature available on risk assessment studies of specific contaminants such as risk assessment of pesticides, trace metal from the soil, and food contamination; equations have been derived and demonstrated (Darko and Akoto 2008; Chabukdhara and Nema 2013; Akoto et al. 2013; Sun and Chen 2018) for various environmental pollutants or cumulative risk assessment.

9.2.4 Dose-Response Assessment (Step 4)

This step of risk assessment determines the correlation between the dose and the toxic effect among the concerned population. Dose-response can be described as the amount of any contaminant or pollutant that is responsible for a certain adverse health effect. A database of USEPA and WHO may be used for the identification of environmental pollutants regarding health effects and dose-responses. For the dose-response assessment, toxicity databases have been determined and published by different authorities. The most useful are integrated risk information systems (IRIS), risk assessment information system (RAIS), agency for toxic substances and disease registry (ATSDR), and health effects assessment summary tables (HEAST). The use of these databases significantly simplifies the assessment procedure. When no data are available, arduous, time-consuming, and very costly toxicological and epidemiological investigations are needed, which in many cases makes the assessment impossible. Every contaminants has initial symptoms that depend on its dose of exposure. In our daily routine life, there are several products that we use which may contain such chemicals in concentration that may cause adverse health effects. For example, exposure to fluoride at more than 1.5 mg/l concentration may show spotted teeth and a decrease in the IQ level of children. However, dose-response is also dependent largely on an individual's immune system and age.

9.2.5 Risk Management (Step 5)

As described in the EPA's risk characterisation handbook, risk management is a process that evaluates how to protect public health from the evaluated risk. Examples of risk management actions include deciding the releasing standards for particular pollutants. Determining the optimum standards for certain contaminants is an example of risk management activity. The national ambient standard along with the permissible limits, acceptable limits for releasing of treated effluent into any waterbody, and set-up of the permission levels for handling, storage, and transport of any hazardous material with its guidelines is also part of risk management. Risk characterisation parameters such as HQ and HI can be used for the management of the risk. At present, no such international standards are available for assessing the risk of environmental exposure (Chartres et al. 2019). Once the international standards have been set for each environmental pollutant, it can be implemented through a proper legislative body of the country. In the absence of standards, published literature might be used to manage the risk. Risk management reduces possible risks, not in terms of environmental safety, and provides economic and social safety. Therefore, the application of risk assessment helps in making the project more sustainable. There are some case studies related to the risk assessment in environmental exposure that will give more insight into the risk assessment application.

9.3 Case Studies Related to the Risk of Environmental Exposure

Various kinds of risks are involved with environmental exposure due to low quality of air, water, and land. Anthropogenic activities pose risk to humans and their environment. Risks of biological agents, toxic chemicals, or radioactive material are commonly found in our habitat. Few landmark case studies in history reveal the significance of studying the risk of environmental exposure from industrial activities that damage the ecological, social, and economical aspects of sustainability. Herein, we describe three case studies related to environmental exposure and its adverse health effects due to contamination of air, water, and land.

9.3.1 Yellowknife Gold Mine, Canada

The extraction of resources has the potential of economic benefit but also results in high contamination of the local environment. The gold mines of the Yellowknife, Northwest Territories of Canada, are a classic example. The mine was operational between 1949 and 1999 and released 237,000 tonnes of toxic waste in the form of arsenic trioxide (As_2O_3) dust, and methyl mercury (Houben et al. 2016). The gold was found in ores of arsenopyrite (FeAsS); consequent roasting was required for conversion of As and S to As_2O_3 and sulphur dioxide (SO_2). These gases were emitted into the environment during the smelting procedure. The toxicity of As is determined by the chemical species and chemical form, rather than the overall amount of As in the environment. For example, arsenobetaine ($(CH_3)_3As^+CH_2COO^-$) is found in marine animals and mushrooms and is less toxic than arsenous acid or arsenite ($As(OH)_3$) (Koch et al. 2000). Ore roasting increases the solubility, toxicity, and bio-accessibility of As by converting sulphide-hosted As to oxide-hosted As. It is more important than the conversion of As as it increases the concentration of dissolved As in groundwater and surface waters. Therefore, it is potentially more toxic due to its high viability. Arsenic trioxide posed a health risk to beings who drank from the tainted water and potentially those that lived near, streams, lakes, and puddles contaminated by falling arsenic dust. Arsenic trioxide dust also settled on local sources of food, especially berries, and vegetables gathered or grown in the Yellowknife area. Various health impacts of arsenic are dose-dependent; arsenic trioxide kills human beings at 70–180 mg. Ingestion of dose levels below the lethal threshold produces a range of health effects that include vomiting, diarrhoea, muscle pain, rashes, parenthesis, and keratosis. Lower exposure over the year can also produce hyperpigmentation (Sandlos and Keeling 2012). Arsenic pollution proved dangerous, even deadly for the Yellowknife's Dene community, in the earliest operating years of the giant mine. Although Yellowknife residents were exposed to arsenic in their water supply, the risk posed to local communities was even greater than the native communities simply due to the proximity of the area they lived in, near the roasting facility, as well as the reliance of people on the polluted snow and lake water for their consumption. The gravity of the situation that the pollution caused can be understood by studying the impacts on animals such as sled dogs, cattle, and chickens dying from drinking arsenic-laden water. Though the Yellowknife gold mine gave a boost to the economy, the pollutants have no boundaries. It increased the risk to the surrounding human health and ecosystem outside the mining area (Jamieson 2014).

9.3.2 Bhopal Gas Disaster, India

Bhopal gas tragedy, the world's deadliest industrial catastrophe, occurred in Union Carbide India Limited (UCIL), Bhopal, India. On the 3rd December 1984, an explosion at the UCIL, a pesticide manufacturing company, released 41 tonnes of methyl iso-cyanate (MIC), a toxic gas that resulted in the death of more than 3000 people, injuring hundreds of thousands more (Koplan et al. 1990). Multitudes of victims in Bhopal were exposed to levels of MIC, with respect to their distance to the plant, and atmospheric factors such as wind velocity and direction; there were more than 500,000 registered survivors of the tragedy (Mishra et al. 2009). According to the Indian council of medical research (ICMR) report, nearly 75% of the deaths occurred within 3 days of the incident. The survivors in the exposed population continued to be chronically ill with diseases of the respiratory, gastrointestinal, reproductive, musculoskeletal, neurological, and other systems (ICMR 2010). Studies related to the reproductive health effects of the event revealed that many years after the disaster, the females of Bhopal exposed to the MIC suffer from problems of menstrual abnormalities, vaginal discharge, and premature menopause. Investigative research was carried out to assess the effect of MIC exposure in pregnant women, including two groups of women who were and were not exposed in Bhopal gas tragedy. It showed 24.2% spontaneous miscarriages in pregnant women who were exposed to MIC, while the control area showed 5.6% spontaneous miscarriages (Bhandari et al. 1990; ICMR 2010). Six serious accidents in the last 4 years before the 1984 tragedy also occurred in the UCIL, which resulted in the death of workers. However, management authorities failed to take action regarding the analysis of the situation and safety measures (Gupta 2002). The local government of Bhopal failed to act in earlier accidents and ignored the newspaper reports predicting disaster. The abovementioned points revealed that major tragedies could occur in any country irrespective of the level of development. The effect of chemical contamination of Bhopal gas is not restricted to air exposure, but it has caused groundwater contamination of various toxic chemicals. The UK-based Bhopal medical appeal and the Sambhavna clinic of Bhopal said, ‘Water contamination is worsening as chemicals leach through soil into the aquifer’ (Goodman 2009). The incident affected people beyond the boundary of industries and caused disaster to the entire local community. It may affect their health, culture, wealth, properties, and ultimately the entire socioeconomic environment, important stakes of sustainability.

9.3.3 Lanzhou Region, Yellow River, China

The Yellow River is the 2nd longest river of China and provides water to millions of people; it is highly contaminated at present. On 25th November 2008, a report furnished by The Guardian, a British daily newspaper, claimed that the river has suffered from severe pollution and that one-third of China’s Yellow River is unusable for drinking, agricultural, or even industrial use, due to factory discharges and sewage from fast-expanding cities. The report on the state of the environment in China revealed that waste and sewage water discharged into the system in 2006 totalled 4.29 billion tonnes. Industry and manufacturing made up 70% of the discharge into the river, with households accounting for 23% and just over 6% coming from other sources (SoE China 2007). Zhang et al. (2018) and Cheng et al. (2019) have found that the metals in sediments of the Yellow River have high concentrations of Cd and Hg among other heavy metals and the pollution and the ecological risk of Hg and Cd are serious. Six types of alkyl esters of p-hydroxybenzoic acids (parabens) were determined in surface water and sediment from the Yellow River, China. The parabens cause adverse effects on the male reproductive system in rats. Besides, parabens were related to the incidence of breast cancer due to the extensive use of cosmetics containing the toxic class (Oishi 2001;

Darbre et al. 2004; Feng et al. 2019). In 2004, 16 different polycyclic aromatic hydrocarbons (PAHs) were detected with concentrations of 23–370 ng/L soluble and 36–3700 ng/g dry weight in the particulate phase (Yu et al. 2009; Feng et al. 2018). These contaminants are cancer-causing and were released from industrial wastewater and household waste.

9.4 Conclusion

The effect of contaminants is not restricted to occupational workers but also covers the entire communities who are directly or indirectly dependent on any of the ecological services in the proximity of industrial intervention such as water supply by the river, pond, or borewell and agricultural practice. The role and responsibility of risk assessment and its management by all industries should extend up to the community level, not only during construction and operational stages but even after the shutdown. Risk assessment application should be imposed in a way that provides safety and security to environmental, ecological, and economical areas to cover all aspects of sustainability.

References

Akoto O, Andoh H, Darko G et al (2013) Health risk assessment of pesticides residue in maize and cowpea from Ejura, Ghana. Chemosphere 92(1):67–73. https://doi.org/10.1016/j.chemosphere.2013.02.057

Amsalu E, Guo Y, Li H et al (2019) Short-term effect of ambient sulfur dioxide (SO_2) on cause-specific cardiovascular hospital admission in Beijing, China: a time series study. Atmos Environ 208:74–81. https://doi.org/10.1016/j.atmosenv.2019.03.015

Andrews RNL (1990) Environmental impact assessment and risk assessment: learning from each other. In: Wathern P (ed) Environmental impact assessment: theory and practice. Routledge. https://doi.org/10.4324/9780203409978

Ashbolt NJ (2004) Microbial contamination of drinking water and disease outcomes in developing regions. Toxicology 198(1–3):229–238. https://doi.org/10.1016/j.tox.2004.01.030

Bhandari N, Syal A, Kambo I et al (1990) Pregnancy outcome in women exposed to toxic gas at Bhopal. Indian J Med Res 92:28–33

Bhandari G, Zomer P, Atreya K et al (2019) Pesticide residues in Nepalese vegetables and potential health risks. Environ Res 172:511–521. https://doi.org/10.1016/j.envres.2019.03.002

Bhatt K, Trivedi M (2018) Molecular studies on foraminifers: past, present, and future. J Foraminiferal Res 48(3):193–209. https://doi.org/10.2113/gsjfr.48.3.193

Boningari T, Smirniotis P (2016) Impact of nitrogen oxides on the environment and human health: Mn-based materials for the NO x abatement. Curr Opin Chem Eng 13:133–141. https://doi.org/10.1016/j.coche.2016.09.004

Boskabady M, Marefati N, Farkhondeh T et al (2018) The effect of environmental lead exposure on human health and the contribution of inflammatory mechanisms, a review. Environ Int 120:404–420. https://doi.org/10.1016/j.envint.2018.08.013

Bruce N, Perez-Padilla R, Albalak R (2000) Indoor air pollution in developing countries: a major environmental and public health challenge. Bull World Health Organ 78(9):1078–1092

Carpenter R (1995) Risk assessment. Impact Assess 13(2):153–187. https://doi.org/10.1080/07349165.1995.9726088

Ceron-Breton J, Ceron-Breton R, Kahl J et al (2015) Diurnal and seasonal variation of BTEX in the air of Monterrey, Mexico: preliminary study of sources and photochemical ozone pollution. Air Qual Atmos Health 8:469–482

Chabukdhara M, Nema A (2013) Heavy metals assessment in urban soil around industrial clusters in Ghaziabad, India: probabilistic health risk approach. Ecotoxicol Environ Saf 87:57–64. https://doi.org/10.1016/j.ecoenv.2012.08.032

Chartres N, Bero L, Norris S (2019) A review of methods used for hazard identification and risk assessment of environmental hazards. Environ Int 123:231–239. https://doi.org/10.1016/j.envint.2018.11.060

Cheng Q, Zhou W, Zhang J et al (2019) Spatial variations of arsenic and heavy metal pollutants before and after the water-sediment regulation in the wetland sediments of the Yellow River Estuary, China. Mar Pollut Bull 145:138–147. https://doi.org/10.1016/j.marpolbul.2019.05.032

Choi A, Sun G, Zhang Y et al (2012) Developmental fluoride neurotoxicity: a systematic review and meta-analysis. Environ Health Perspect 120(10):1362–1368. https://doi.org/10.1289/ehp.1104912

Coster SD, Larebeke NV (2012) Endocrine-disrupting chemicals: associated disorders and mechanisms of action. J Environ Public Health:1–52. https://doi.org/10.1155/2012/713696

Cushen R, Brunt H, Jones S et al (2019) An unusual incident: carbon monoxide poisoning risk in 540 homes due to faulty wood burner installations. Public Health 173:17–20. https://doi.org/10.1016/j.puhe.2019.05.003

Damania R, Desbureaux S, Rodella A et al (2019) Quality unknown: the invisible water crisis. Washington, DC: World Bank. http://hdl.handle.net/10986/32245

Darbre PD, Aljarrah A, Miller WR et al (2004) Concentrations of parabens in human breast tumours. J Appl Toxicol 24(1):5–13. https://doi.org/10.1002/jat.958

Darko G, Akoto O (2008) Dietary intake of organophosphorus pesticide residues through vegetables from Kumasi, Ghana. Food Chem Toxicol 46(12):3703–3706. https://doi.org/10.1016/j.fct.2008.09.049

De Miguel E, Iribarren I, Chacón E et al (2007) Risk-based evaluation of the exposure of children to trace elements in playgrounds in Madrid (Spain). Chemosphere 66(3):505–513. https://doi.org/10.1016/j.chemosphere.2006.05.065

Dhananjayan V, Ravichandran B (2018) Occupational health risk of farmers exposed to pesticides in agricultural activities. Curr Opin Environ Sci Health 4:31–37. https://doi.org/10.1016/j.coesh.2018.07.005

Ding Y, Gao Y, Sun H et al (2011) The relationships between low levels of urine fluoride on children’s intelligence, dental fluorosis in endemic fluorosis areas in Hulunbuir, Inner Mongolia, China. J Hazard Mater 186(2–3):1942–1946. https://doi.org/10.1016/j.jhazmat.2010.12.097

Duan Q, Jiao J, Chen X et al (2018) Association between water fluoride and the level of children’s intelligence: a dose–response meta-analysis. Public Health 154:87–97. https://doi.org/10.1016/j.puhe.2017.08.013

Feng J, Hu P, Su X et al (2018) Impact of suspended sediment on the behaviour of polycyclic aromatic hydrocarbons in the Yellow River: spatial distribution, transport and fate. Appl Geochem 98:278–285. https://doi.org/10.1016/j.apgeochem.2018.10.008

Feng J, Zhao J, Xi N et al (2019) Parabens and their metabolite in surface water and sediment from the Yellow River and the Huai River in Henan Province: spatial distribution, seasonal variation and risk assessment. Ecotoxicol Environ Saf 172:480–487. https://doi.org/10.1016/j.ecoenv.2019.01.102

Foley J (2011) A five-step global plan could double food production by 2050 while greatly reducing environmental damage. Sci Am: November:60–66. https://web.mit.edu/12.000/www/m2019/pdfs/Foley_2011_ScientificAmerican.pdf

Garg A, Gupta NC (2019) A comprehensive study on spatio-temporal distribution, health risk assessment and ozone formation potential of BTEX emissions in ambient air of Delhi, India. Sci Total Environ 659:1090–1099. https://doi.org/10.1016/j.scitotenv.2018.12.426

Ge H, Yamazaki E, Yamashita N et al (2017) Particle size specific distribution of perfluoroalkyl substances in atmospheric particulate matter in Asian cities. Environ Sci Process Impacts 19: 549–560. https://doi.org/10.1039/C6EM00564K

Geng F, Tie X, Xu J et al (2008) Characterizations of ozone, NOx, and VOCs measured in Shanghai, China. Atmos Environ 42:6873–6688

Gianicolo EAL, Mangia C, Cervino M (2016) Investigating mortality heterogeneity among neighbourhoods of a highly industrialised Italian city: a meta-regression approach. Int J Public Health 61(7):777–785. https://doi.org/10.1007/s00038-016-0868-y

Goodman S (2009) Poisoned water haunts Bhopal 25 years after chemical accident. Available via Scientific American. https://www.scientificamerican.com/article/groundwater-contamination-india-pesticide-factory/. Accessed 9 Aug 2019

Guaita R, Pichiule M, Mate T et al (2011) Short-term impact of particulate matter ($PM_{2.5}$) on respiratory mortality in Madrid. Int J Environ Health Res 21:260–274. https://doi.org/10.1080/09603123.2010.544033

Gul M, Ak F (2018) A comparative outline for quantifying risk ratings in occupational health and safety risk assessment. J Clean Prod 196:653–664. https://doi.org/10.1016/j.jclepro.2018.06.106

Guo M, Lyu Y, Xu T et al (2018) Particle size distribution and respiratory deposition estimates of airborne perfluoroalkyl acids during the haze period in the megacity of Shanghai. Environ Pollut 234:9–19. https://doi.org/10.1016/j.envpol.2017.10.128

Gupta J (2002) The Bhopal gas tragedy: could it have happened in a developed country? J Loss Prev Process Ind 15(1):1–4. https://doi.org/10.1016/S0950-4230(01)00025-0

Hackney JD, Linn WS, Law DC et al (1975) Experimental studies on human health effects of air pollutants: ii. Two-hour exposure to ozone alone and in combination with other pollutant gases. Arch Environ Health 30(8):385–390. https://doi.org/10.1080/00039896.1975.10666730

Halonen JI, Lanki T, Tuomi TY et al (2009) Particulate air pollution and acute cardiorespiratory hospital admissions and mortality among the elderly. Epidemiology 20:143–153. https://doi.org/10.1097/EDE.0b013e31818c7237

HES (2019) Health, executive safety gas safe register. Carbon monoxide poisoning. What are the symptoms? Available via Health Executive Safety. https://www.gassaferegister.co.uk/help-and-advice/carbon-monoxide-poisoning/. Accessed 30 July 2019

Houben AJ, D'Onofrio R, Kokelj SV et al (2016) Factors affecting elevated arsenic and methyl mercury concentrations in small shield lakes surrounding gold mines near the Yellowknife, NT, (Canada) region. PLoS One 11(4):e0150960. https://doi.org/10.1371/journal.pone.0150960

ICMR (2010) Indian Council of Medical Research report on health effects of the toxic gas leak from the methyl isocyanate plant in Bhopal. Bhopal Gas Disaster Centre & Institute of Pathology, ICMR, New Delhi

Ielpo P, Mangia C, Marra GP et al (2019) Outdoor spatial distribution and indoor levels of NO_2 and SO_2 in a high environmental risk site of the South Italy. Sci Total Environ 648:787–797. https://doi.org/10.1016/j.scitotenv.2018.08.159

Jamieson H (2014) The legacy of arsenic contamination from mining and processing refractory gold ore at Giant Mine, Yellowknife, northwest territories, Canada. Rev Mineral Geochem 79(1): 533–551. https://doi.org/10.2138/rmg.2014.79.12

Kim H, Kim H, Park YH et al (2017) Assessment of temporal variation for the risk of particulate matters on asthma hospitalization. Environ Res 156:542–550. https://doi.org/10.1016/j.envres.2017.04.012

Koch I, Wang L, Ollson C et al (2000) The predominance of inorganic arsenic species in plants from Yellowknife, northwest territories, Canada. Environ Sci Technol 34:22–26. https://doi.org/10.1021/es9906756

Koplan JP, Falk H, Green G (1990) Public health lessons from the Bhopal chemical disaster. J Am Med Assoc 264(21):2795–2796

Krauskopf J, Veldhoven KV, Chadeau-Hyam M et al (2019) Short-term exposure to traffic-related air pollution reveals a compound-specific circulating miRNA profile indicating multiple disease risks. Environ Int 128:193–200. https://doi.org/10.1016/j.envint.2019.04.063

Laidlaw M, Filippelli G, Sadler R et al (2016) Children's blood lead seasonality in Flint, Michigan (USA), and soil-sourced lead hazard risks. Int J Environ Res Public Health 13:358. https://doi.org/10.3390/ijerph13040358

Liu Z, Guan Q, Luo H et al (2019a) Development of land use regression model and health risk assessment for NO_2 in different functional areas: a case study of Xi'an, China. Atmos Environ 213:515–525. https://doi.org/10.1016/j.atmosenv.2019.06.044

Liu H, Liao J, Jiang Y et al (2019b) Maternal exposure to fine particulate matter and the risk of fetal distress. Ecotoxicol Environ Saf 170:253–258. https://doi.org/10.1016/j.ecoenv.2018.11.068

Lohani B, Evans J, Ludwig H et al (1997) Environmental impact assessment for developing countries in Asia. Volume 1 Overview. Asian Development Bank, Manila, p 356

Masters GM, Ela W (2008) Introduction to environmental engineering and science (No. 60457). Prentice Hall, Englewood Cliffs

Meyer PA, McGeehin MA, Falk H (2003) A global approach to childhood lead poisoning prevention. Int J Hyg Environ Health 206(4–5):363–369. https://doi.org/10.1078/1438-4639-00232

Mishra PK, Samarth RM, Pathak N et al (2009) Bhopal gas tragedy: review of clinical and experimental findings after 25 years. Int J Occup Environ Health 22(3):193–202. https://doi.org/10.2478/v10001-009-0028-1

Mrugesh T, Dipa L, Manishika G (2011) Effect of lead on human erythrocytes: an in vitro study. Acta Pol Pharm 68(5):653–656

Müller CE, Gerecke AC, Bogdal C et al (2012) Atmospheric fate of poly-and perfluorinated alkyl substances (PFASs): I. Day–night patterns of air concentrations in summer in Zurich, Switzerland. Environ Pollut 169:196–203. https://doi.org/10.1016/j.envpol.2012.04.010

Neuberger M, Moshammer H, Kundi M (2002) Declining ambient air pollution and lung function improvement in Austrian children. Atmos Environ 36:1733–1736. https://doi.org/10.1016/S1352-2310(02)00179-6

Nevin R (1999) How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy. Environ Res 83(1):1–22. https://doi.org/10.1006/enrs.1999.4045

Ngajilo D, Jeebhay MF (2019) Occupational injuries and diseases in aquaculture – a review of literature. Aquaculture 507:40–55. https://doi.org/10.1016/j.aquaculture.2019.03.053

Oishi S (2001) Effects of butylparaben on the male reproductive system in rats. Toxicol Ind Health 17(1):31–39. https://doi.org/10.1191/0748233701th093oa

Paithankar A (2011) Hazard identification and risk analysis in mining industry. Department of Mining Engineering National Institute of Technology Rourkela

Perez L, Tobías A, Querol X et al (2012) Saharan dust, particulate matter and cause-specific mortality: a case-crossover study in Barcelona (Spain). Environ Int 48:150–155. https://doi.org/10.1016/j.envint.2012.07.001

Pope CA, Burnett RT, Thurston GD et al (2004) Cardiovascular mortality and long-term exposure to particulate air pollution epidemiological evidence of general zathophysiological pathways of disease. Circulation 109(1):71–77. https://doi.org/10.1161/01.CIR.0000108927.80044.7F

Posthuma L, Suter G (2011) Ecological risk assessment of diffuse and local soil contamination using species sensitivity distributions. In: Dealing with contaminated sites. Springer, Dordrecht

Pourhassan B, Meysamie A, Alizadeh S et al (2019) Risk of obstructive pulmonary diseases and occupational exposure to pesticides: a systematic review and meta-analysis. Public Health 174: 31–41. https://doi.org/10.1016/j.puhe.2019.05.024

Pruss-Ustun A, Wolf J, Corvalán C et al (2016) Preventing disease through healthy environments: a global assessment of the burden of disease from environmental risks. World Health Organisation, Geneva

Ramirez N, Cuadras A, Rovira E et al (2012) Chronic risk assessment of exposure to volatile organic compounds in the atmosphere near the largest Mediterranean industrial site. Environ Int 39(1):200–209

Razdan P, Patthi B, Kumar JK et al (2017) Effect of fluoride concentration in drinking water on intelligence quotient of 12–14-year-old children in Mathura district: a cross-sectional study. J Int Soc Prev Community Dent 7(5):252–258. https://doi.org/10.4103/jispcd.JISPCD_201_17

Reynolds M, Jennifer G (eds) (2015) Laboratory bio-risk management biosafety and biosecurity. CRC Press Inc, Boca Raton

Rich AL, Phipps LM, Tiwari S et al (2016) The increasing prevalence in intersex variation from toxicological dysregulation in fetal reproductive tissue differentiation and development by endocrine-disrupting chemicals. Environ Health Insights 8(10):163–171. https://doi.org/10.4137/EHI.S39825

Ruan Y, Lalwani D, Kwok K et al (2019) Assessing exposure to legacy and emerging per- and polyfluoroalkyl substances via hair – the first nationwide survey in India. Chemosphere 229: 366–373. https://doi.org/10.1016/j.chemosphere.2019.04.195

Samoli E, Aga E, Touloumi G (2007) Short-term effects of nitrogen dioxide on mortality: an analysis within the APHEA project. Environ Health Perspect 115:1578–1583. https://doi.org/10.1183/09031936.06.00143905

Samoli E, Peng R, Ramsay T et al (2008) Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. Environ Health Perspect 116(11): 1480–1486. https://doi.org/10.1289/ehp.11345

Sandlos J, Keeling A (2012) Giant mine: historical summary. Memorial University August 8, 2012. Available via Memorial University Research Repository. https://research.library.mun.ca/638/. Accessed 6 May 2021

Seraj B, Shahrabi M, Shadfar M et al (2012) Effect of high water fluoride concentration on the intellectual development of children in Makoo/Iran. Int J Dent 9(3):221–229

Shin I, Oh JB, Yi HK (2011) Workers' compensation insurance and occupational injuries. Saf Health Work 2(2):148–157. https://doi.org/10.5491/SHAW.2011.2.2.148

SoE (2007) Report on the state of the environment in China. Ministry of Environmental Protection, The People's Republic of China. Available via http://english.mee.gov.cn/Resources/Reports/soe/soe2007/201605/P020160531580295293022.pdf. Accessed 6 May 2021

Sun Z, Chen J (2018) Risk assessment of potentially toxic elements (PTEs) pollution at a rural industrial wasteland in an abandoned metallurgy factory in North China. Int J Environ Res Public Health 15(1):85. https://doi.org/10.3390/ijerph15010085

Tchounwou PB, Yedjou CG, Patlolla AK et al (2012) Heavy metal toxicity and the environment. In: Luch A (ed) Molecular, clinical and environmental toxicology. Experientia Supplementum, vol 101. Springer, Basel. https://doi.org/10.1007/978-3-7643-8340-4_6

Thorpe KL, Hutchinson TH, Hetheridge MJ et al (2001) Assessing the biological potency of binary mixtures of environmental estrogens using vitellogenin induction in juvenile rainbow trout (*Oncorhynchus mykiss*). Environ Sci Technol 35(12):2476–2481. https://doi.org/10.1021/es001767u

Trivedi M, Verma R, Chinoy N et al (2007) Effect of high fluoride water on intelligence of school children in India. Fluoride 40(3):178–183

Trivedi M, Sangai NP, Patel RS et al (2012) Assessment of groundwater quality with special reference to fluoride and its impact on IQ of schoolchildren in six villages of the Mundra region, Kachchh, Gujarat, India. Fluoride 45(4):377–383

Trivedi M, Vaidya D, Patel C et al (2020) In silico and in vitro studies to elucidate the role of 1HYN and 1QKI activity in BPA induced toxicity and its amelioration by Gallic acid. Chemosphere 241:125076. https://doi.org/10.1016/j.chemosphere.2019.125076

UNDP (2019a) Goal 3: good health and well-being. Facts and figures. https://www.undp.org/content/undp/en/home/sustainable-development-goals/goal-3-good-health-and-well-being.html. Accessed 30 July 2019

UNDP (2019b) Goal 6: clean water and sanitation. Facts and figures. https://www.undp.org/content/undp/en/home/sustainable-development-goals/goal-6-clean-water-and-sanitation.html. Accessed 1 Aug 2019

USEPA (2008) Child-specific exposure factors handbook. National Center for Environmental Assessment Office of Research and Development, Washington, DC; EPA/600/R-06/096F. 20460

USEPA (2011) Exposure factors handbook: 2011 edition. National Center for Environmental Assessment, Washington, DC. EPA/600/R-09/052F

USEPA (2014) Human health evaluation manual, supplemental guidance: update of standard default exposure factors. OSWER Directive 9200.1-120. Office of Solid Waste and Emergency Response. US Environmental Protection Agency, Washington, DC. Available via EPA. https://www.epa.gov/sites/production/files/2015-11/documents/oswer_directive_9200.1-120_exposurefactors_corrected2.pdf

USEPA (2019a) About risk assessment. Available via EPA. https://www.epa.gov/risk/about-risk-assessment#whatisrisk. Accessed 26 July 2019

USEPA (2019b) CCL 3 Universe list. Available via EPA. http://www.epa.gov/safewater/ccl/pdfs/report_ccl3_microbes_universe.pdf. Accessed 1 Aug 2019. Accessed 6 May 2021

USEPA (2019c) Conducting a human health risk assessment. Available via EPA. https://www.epa.gov/risk/conducting-human-health-risk-assessment. Accessed 26 July 2019

USEPA (2019d) Criteria air pollutants. Available via EPA. https://www.epa.gov/criteria-air-pollutants. Accessed 26 July 2019

WHO (2006) Air quality guidelines of WHO for particulate matter, ozone, nitrogen dioxide and sulfur dioxide, Global update 2005: WHO/SDE/PHE/OEH/06.02. World Health Organization. Available via WHO. http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf. Accessed 6 May 2021

WHO (2011) WHO guideline for drinking water quality, 4th edn. World Health Organization, Geneva

WRI (1998) World Resources Institute – world resources, 1998/1999. Oxford University Press, UK

Yu Y, Xu J, Wang P et al (2009) Sediment-porewater partition of polycyclic aromatic hydrocarbons (PAHs) from Lanzhou Reach of Yellow River, China. J Hazard Mater 165(1–3):494–500. https://doi.org/10.1016/j.jhazmat.2008.10.042

Zhang P, Qin C, Hong X et al (2018) Risk assessment and source analysis of soil heavy metal pollution from lower reaches of Yellow River irrigation in China. Sci Total Environ 633:1136–1147. https://doi.org/10.1016/j.scitotenv.2018.03.228